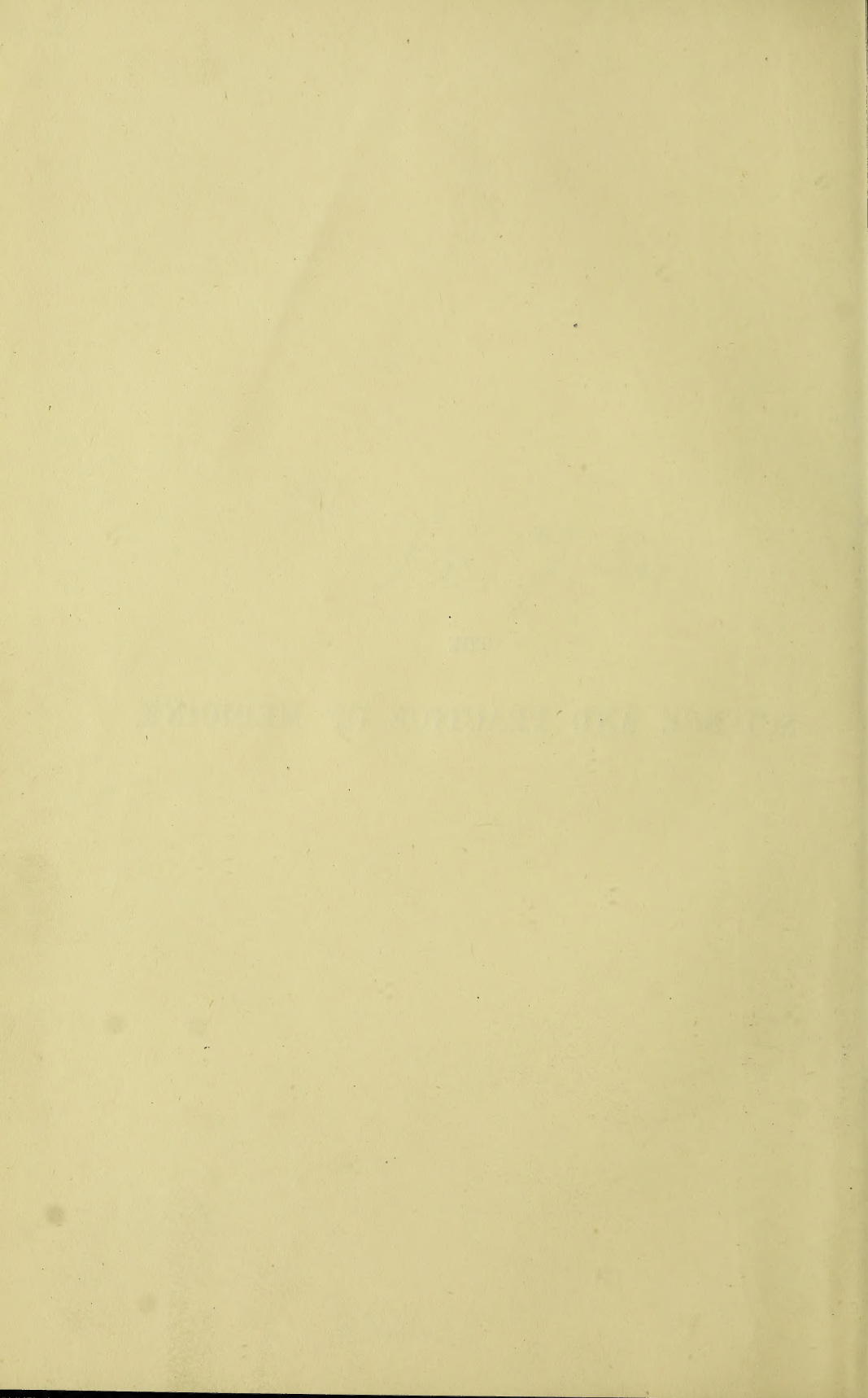


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THE
SCIENCE AND PRACTICE OF MEDICINE.



THE
SCIENCE AND PRACTICE
OF
MEDICINE.

BY

WILLIAM AITKEN, M.D. (EDIN.), F.R.S.,

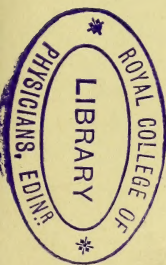
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PATHOLOGIST ATTACHED TO THE MILITARY HOSPITALS OF
THE BRITISH TROOPS AT SCUTARI IN TURKEY
DURING THE CRIMEAN WAR.

SEVENTH EDITION,

TO A LARGE EXTENT REWRITTEN; ENLARGED, REMODELLED, AND
CAREFULLY REVISED THROUGHOUT.

IN TWO VOLUMES,

*Illustrated by One Hundred and Ninety-six Engravings on Wood, and a Map of
the Geographical Distribution of Diseases.*



VOL. I.

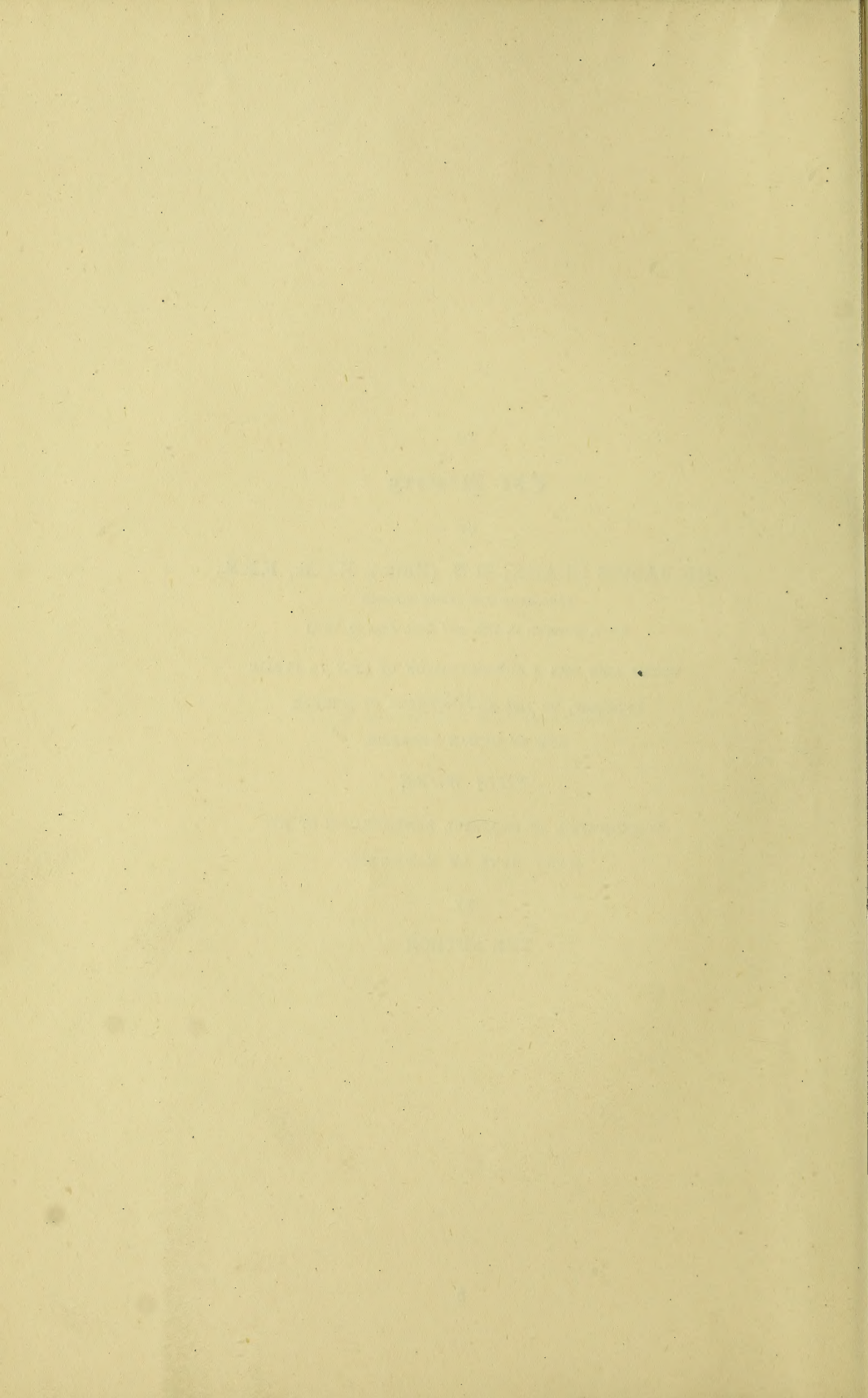
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TO
The Memory
OF
SIR JAMES CLARK, M.D. (EDIN.), K.C.B., F.R.S.,
BARONET OF THE UNITED KINGDOM
(Born, December 14, 1788, and Died, June 29, 1870.)
WHOSE LIFE WAS A SUBORDINATION OF SELF TO PUBLIC
INTEREST, TO THE ADVANCEMENT OF SCIENCE,
AND OF HUMAN PROGRESS,
THIS WORK
IS DEDICATED, IN GRATEFUL REMEMBRANCE OF HIS
MANY ACTS OF KINDNESS,
BY
THE AUTHOR.



PREFACE TO THE SEVENTH EDITION.

It is now (1880) twenty-two years since the First Edition of this work was published, and eight years since the Sixth Edition issued from the press. For two years the work has been out of print, and I gladly avail myself of the opportunity afforded by the call for a Seventh Edition, to express my grateful acknowledgments for the kind reception which each succeeding edition has met with.

The extensive sale of six large impressions in twenty years is, perhaps, the most satisfactory proof that the work has been—what it was originally designed to be—a faithful “Representative of the Medical Science and Practice of the day;” and I hope that this, the Seventh Edition, may deserve and continue to retain the confidence alike of Students of Medicine, of Teachers, and of the Profession.

The present Edition has not only undergone a very careful revision, but in many parts it has received large additions; in many other parts it has been entirely remodelled; and, to a large extent, it is altogether re-written; ever keeping in view Boileau’s advice to authors:—“*Ajoutez quelquefois, et souvent effacez.*” Attention has been given to the condensation of the text, to the removal of inaccuracies, and to the saving of space in the mechanical execution of the work; further, by increasing the width of the page (the number of lines remaining the same), the quantity of matter has been to an appreciable extent increased, without augmentation of the actual bulk of the volumes.

The additions and changes in this Edition are scattered throughout the two volumes; but perhaps the greatest alterations are to be found in the sections under Diseases of the Brain and Nervous System. The amount of scientific work in the topics comprehended in these sections during the past ten years has been simply overwhelming. This part has been entirely re-written. From amongst the large mass of available material, I have endeavoured, as heretofore, to select the leading and best determined facts representing the

present aspect of the Science and Practice of Medicine in these important sections of local diseases; and, in the treatment of these sections, I feel called upon to express particular obligations to the valuable writings of Ecker, Gowers, David Ferrier, Hughlings Jackson, Turner, Erb, Charcot, Crichton Browne, Bristowe, Clifford Allbutt, Althaus, Michael Foster, Alexander Robertson, M'Kendrick, and others, to whom acknowledgment is duly made in the text; as well as specially to Quain's *Anatomy*, and *The West Riding Asylum Reports*.

In the present Edition the distribution of the material between the two volumes has been arranged so as to equalise their bulk, and it is hoped the changes and alterations may render the work, in other respects, more useful.

The First Volume contains the elementary chapters on General Pathology, and on the Specific and Constitutional Diseases.

In the Second are comprised the Local Diseases, the part on Medical Geography, and a copious Index to both volumes.

I have endeavoured, as heretofore, to make due reference to the leading authorities on the different subjects, from whom I have derived special information. A few new woodcuts have been introduced where it was thought they would render the description in the text more intelligible, and the sources whence obtained—Ferrier, Ecker, Turner, and Allen Thomson, in the Eighth Edition of Quain's *Anatomy*—are duly specified in each instance.

In conclusion, I can but express again, as on former occasions, my hearty thanks to numerous kindly and considerate fellow-workers (many of whom are personally unknown to myself), for the valuable and painstaking communications with which they have favoured me from time to time, and which have contributed not a little to the completeness of the present Edition.

My acknowledgments are also due to Dr. Morell Mackenzie, Physician to the London Hospital, who has again kindly revised the sections bearing on the application of the Laryngoscope in the Diagnosis and Treatment of Diseases of the Throat and Larynx.

WILLIAM AITKEN.

ARMY MEDICAL SCHOOL,
ROYAL VICTORIA HOSPITAL,
NETLEY, HANTS, *September, 1880.*

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THE SCIENCE AND PRACTICE OF MEDICINE.

PART I.

TOPICS RELATIVE TO PATHOLOGY.

CHAPTER I.

OF MEDICINE AS A SCIENCE AND AS AN ART; ITS OBJECTS AND ITS EXTENT.

THE study of MEDICINE is prosecuted under two relations, namely, as a *Science* and as an *Art*.

Considered as a *Science*, MEDICINE takes cognisance of all that relates to our knowledge of diseases; and, especially, of the circumstances under which they arise or become developed, of the conditions of their existence, of their nature and of their causes, in the widest sense of these terms.

Considered as an *Art* (in so far as Medicine has that practical value), its object and aim are to distinguish, to prevent, and to cure diseases; to alleviate human suffering, and to lengthen out human existence, by warding off or by modifying disease "as the greatest of mortal evils;" and, by restoring health, and even at times reason itself, "as the greatest of mortal blessings."

In general terms, therefore, MEDICINE is prosecuted as a Science and as an Art, with the practical objects in view of so learning to understand the nature of diseases and to appreciate their causes, as to prevent their occurrence when possible, promote their cure when they do occur, or to relieve the sufferings of those whose diseases cannot be cured.

Many branches of human knowledge must therefore be combined in the study, the construction, and the elucidation of the *Science* of Medicine; and the *practice* of the ART ought to be founded on principles and facts of universal applicability. It now divides itself into two distinct branches, having two distinct and separate ends in view, namely:—*First*, THE PREVENTION OF DISEASE, AND MAINTAINING THE CONDITIONS FOR PRESERVING HEALTH (*Hygiène*); *Second*, THE CURE OF DISEASE, AND THE RELIEF OF THOSE WHO SUFFER (*Therapeutics*).

The Practice of each of these separate divisions of Medicine is now

undertaken by separate classes of men, each class being specially trained for the respective ends in view. Already we practically recognise—(1.) Sanitary Officers, or “Medical Officers of Health,” whose duties consist in maintaining the conditions for preserving health, and legally enforcing the means for the prevention of disease, in accordance with the several Sanitary Acts and Public Health Acts passed by Parliament; and, (2.) General Practitioners of Medicine, whose duties consist in the curing of diseases as they inevitably occur. Both these classes of professional men must make themselves acquainted with the elementary facts which are comprehended in the Science and Practice of Medicine; and which it is the object of this work to teach.

A consideration of the different topics of human knowledge which together make up the Science of Medicine suggests its division into the following departments, namely:—

(1.) **PHYSIOLOGY**, which embraces a study of the living phenomena of the human body, in their natural or healthy state;—of the peculiar physical structure and conformation of its solid parts;—of the determinate chemical composition of its several solids and fluids;—of its dynamical characters; and, lastly, of the occurrence of certain chemical, physical, and vital changes which go on during that active state which we call “Life;”—a state which results from the concurrent exercise of the several functions performed by various organs, which follows a determined evolutionary history from the commencement of life to its close, and so passes through different phases, which succeed each other regularly and in a certain order.

(2.) **PATHOLOGY** (subdivided into *Special Pathology* and *General Pathology*), which embraces a consideration of everything relative to the origin, the existence, and the nature of diseases.

(3.) **THERAPEUTICS**, expounding the actions of remedies upon the diseased economy, and the means by which nature may be aided in her return to health.

(4.) **HYGIENE**, which embraces a consideration of the means of preventing disease, or, in other words, of preserving health.

These departments of science imply an intimate acquaintance with Anatomy, Chemistry, and Physics,—all of which are preliminary subjects of study, and they constitute a necessary and appropriate introduction to the Practice of Medicine, in which Special Pathology and the treatment of special diseases are the leading topics of consideration.

Each of these departments has grown or expanded itself into a great branch of science; and any single section is sufficient of itself to occupy the lifetime of an individual in working out and studying it in detail. It is therefore not possible for the human mind to embrace all of these departments in their whole extent or relations to each other; and, setting aside the consideration of theories and systems, it has been truly observed, “that no man possesses all the pathological knowledge contained in the records of his art” (CHOMEL). Still less possible is it to embrace in any single treatise a complete and connected view of the whole Science of Medicine in all of these departments.

But for the purpose of teaching the Science in its application to Practice, its elementary principles, as developed in the departments of

Pathology, are the most useful guides to the student; and the aim of the following introductory chapters relative to Pathology, is to elucidate these principles.

CHAPTER II.

HOW THE PROVINCE OF PATHOLOGY IS MAPPED OUT.

AN inquiry into the Pathology or nature of diseases embraces a consideration of the following topics:—

(1.) *The accurate observation and correct registration of Facts.* On the efficiency of the machinery devised for these important ends will rest our power to curb the invasion of our science by the guess-work of theory; and eventually to root out the traditional errors which so largely pervade medical literature, especially as to matters of fact and observation.

(2.) *Descriptive Pathology, embracing GENERAL and SPECIAL Pathology.* *Special Pathology* is intended to comprehend a consideration of the essential nature and origin of particular diseases as they occur in man and animals, and *General Pathology* to include those more general facts or principles which result from a comparison of particular diseases with each other. Although *Special Pathology* comes first in the order of Nature, yet, wherever the arrangements for Medical education are complete, *General Pathology* is taught as an introduction to, or conjointly with, the special study of diseases; just as in other sciences—for example, in chemistry—it is found convenient to give a general view of the principles which have been established by experiment and observation, before entering upon the particular details of the science. All theory in Medicine, all *Descriptive Pathology*, all grounds for rational speculation regarding the nature of diseases, and for the framing of experiments, as well as all maxims of practice which aim at the prevention or cure of diseases, must rest ultimately on observed and recorded facts.

Accuracy of observation is therefore the first lesson the student has to learn in all methods of investigation; and the lesson is one of paramount importance. The best observations, however, will avail but little unless the observed facts are recorded in such a way as to secure their preservation; and *Descriptive Pathology* mainly concerns itself, in the first instance, with the *Registration of Facts*; (a.) In the *History of cases of disease* from their origin to their end, thus preserving, in an authentic and permanent form, the memory of Facts in Pathology as they occur; (b.) In the *Statistics of disease*. Materials are thus eventually obtained upon which future pathologists and statisticians will build a comprehensive and definite system of scientific Medicine; and they will furnish the means of teaching all that is necessarily involved in our notions regarding the nature of diseases.

The *Descriptive Pathology*, so arrived at, considers diseases as they exist, or have existed in man, in the lower animals, or in plants. It

considers the conditions under which diseases originate; how far certain conditions are fulfilled before disease establishes itself; and it aims at demonstrating how far such conditions are inconsistent or incompatible with the maintenance of health. Subsequently, with extended information, Descriptive Pathology may undertake to assign the conditions which give rise to certain diseases rather than to others. It will eventually define the elements necessary to establish, to originate, or to constitute particular diseases; and will show how the same disease, or class of diseases, may assume various forms, but in all of which definite typical elements are still recognisable. Descriptive Pathology thus aims at determining and describing the essential elements of a disease. The Pathological and Clinical Societies widely established throughout the country are doing good service in these directions.

(3.) *Speculative Pathology*, which assumes that we know what a disease is—that we know the effects it produces—that we know the conditions necessary for its existence—that we know its relations to other diseases. It seeks, therefore, to inquire how certain conditions or circumstances will operate in bringing about disease. It seeks to determine, from certain observed facts in the course of a disease, what is the tendency to the recurrence of similar results in the course of similar diseases. Statistical data are thus the main basis in the operations of Speculative Pathology.

(4.) *Pathology as dictating the maxims of rational practice*.—It is in the nature of the science of Pathology that it always ought to be in advance of our certain knowledge regarding the treatment of diseases. It is the basis of rational medicine. It is necessary to know the nature of a disease, in order—(1.) To prevent it; (2.) To understand the principles which ought to guide us in the management of it.

It is intended, in the first part of this work, to guide the student to appreciate,—

- (1.) The *relative* nature of the terms "*Life*," "*Health*," "*Disease*."
- (2.) How the nature and causes of diseases may be elucidated.
- (3.) The nature of morbid phenomena, symptoms, and signs of disease.
- (4.) The Methodical examination of patients and keeping of case-books.
- (5.) The means and instruments of investigation into the nature and causes of disease.
- (6.) Some of the more elementary constituents of disease.
- (7.) Some complex morbid states associated with individual diseases, or with conditions of ill-health (*cachexiæ*).
- (8.) The modes by which diseases terminate fatally; the types of disease, and their tendency to change.
- (9.) The management and general treatment of the more complicated morbid states of the system, comprehended under the name of GENERAL DISEASES.

In the subsequent parts of this work, it is intended to consider some of the details of the Science and Practice of Medicine; to furnish the student with,—

- (1.) A nosological system by which to arrange, classify, and name diseases.

(2.) A detailed description of characteristic diseases in the respective classes of that nosological system. In this part a definition (provisional) and a history of the nature of each disease will be given; the probable course and succession of events in the progress of each will be described, and the grounds on which an accurate diagnosis may be made, or a prognosis expected; and, lastly, a detailed account of those rational modes of treatment and general management of cases of disease which are consistent with the established principles of the *Institutes of Medicine*.

(3.) An account of what is known relative to the geographical distribution of diseases.

CHAPTER III.

RELATIVE NATURE OF THE TERMS *LIFE, HEALTH, DISEASE.*

THE word *Disease* is used in a general, and also in a specific sense; as when it is said that a person is diseased, without the nature of the affection being stated; or, that he suffers from a particular disease, such as small-pox. Attempts to give a precise definition of the term *Disease* have generally been unsuccessful. The relations of the morbid state to the condition of health, and of health to the performance of the vital functions, are of such a kind that they can only be described in connection, comparison, or contrast with each other, but not defined.

If *Life* is understood to imply an active state, resulting from the concurrent exercise of the functions of the body, then there are conditions of activity and of mutual adaptability of functions and of parts, both as regards body and mind, which are necessary to healthy existence. Our notions of the *conditions of health* have thus considerable latitude; and *Health* is merely a name we give to that state or condition in which a person exists fully able, without suffering, to perform all the functions and duties of life; "a state in which there is all the normal activity of all the component parts of the complex organism of man." Many degrees of *Health* are therefore at first sight obvious, from the possession of a feeble existence to the most robust and active condition of the body; and there are even many degrees of feebleness and delicacy of *Health* without any disorder of the system. Our notions of *normal life* are thus so extremely indefinite, that it is only by a forced abstraction the normal can be separated from the abnormal. Hence also our idea of *Disease* is very indefinite, implying "some deviation from the normal activity of one or more of the component parts of the organism;" it cannot be separated by any well-defined boundary from our idea of *normal life*; and thus the two conditions of *health* and *disease* are sometimes connected by a kind of debatable border-land.

When we regard, therefore, the phenomena of the living state and the conditions of health, we can readily observe when and how *Disease* is but a deviation from the state of health, consisting for the most part

in a change in the properties or structure of any tissue or organ, which renders such tissue or organ unfit for the performance of its actions or functions according to the laws of the healthy frame. It is now also an accepted doctrine that *Disease* does not consist in any single state, entity, or special existence, but is the natural expression of a combination of phenomena, arising out of impaired function or altered structure, and recognising some deleterious agency acting on the body as a cause. All attempts to define disease by the use of such terms as "*derangement*," "*modification*," "*alteration*," "*change*," from the pre-existent state of health, show, in the first instance, that various ideas are attachable to the terms or to the state to which they are meant to apply; and, secondly, that these terms point to a nosological division into structural and functional diseases, rather than to a state common to all forms of disease.

The cause, therefore, must be regarded as an essential part of the disease, so that the term "*Disease*" ought to convey to our minds some definite, special, or peculiar cause, born in the system (inbred, innate, constitutional, or inherited), or acting on the system from without; and in any case determining, according to its nature and operation, the character and grouping of the morbid phenomena which ensue. Thus the biography of every disease comprises some special cause and certain resultant phenomena—vital, chemical, or mechanical, or all combined—producing the symptoms and signs by which the presence of disease is recognised (BRISTOWE).

A definition of any disease ought, therefore, to include—(1.) The cause on which the morbid phenomena depend; (2.) All the circumstances, whether functional or organic—all the collective morbid phenomena or symptoms which indicate disease, and which constitute the deviation from health. For obvious reasons such a definition can only be approximately expressed, and thus it can but very incompletely circumscribe the subject by shadowy outlines.

If the deviation from normal action be accompanied by appreciable alteration of structure, *disease* is then said to be *structural* or *organic*; if there be no appreciable change of structure, *disease* is then said to be *functional*. But this is merely an arbitrary and relative distinction; for, if no lesion can be found, it does not follow that there is no alteration of structure. It may have escaped being found out; and some better method of examination may eventually teach us the nature of many diseases which hitherto are said to exist without any characteristic lesion to be seen after death—nothing that our senses can appreciate.

CHAPTER IV.

HOW THE NATURE AND CAUSES OF DISEASES MAY BE ELUCIDATED.

THE nature of the derangements to which the human body is liable may be studied under the three following aspects:—

(1.) As diseases presenting themselves in individual cases, which are thus the subjects of CLINICAL or BED-SIDE INVESTIGATION and INSTRUCTION—a method of teaching in which the *Natural History* of the disease becomes a special subject of study.

(2.) As diseases constituting particular genera or species, forming the topics of SPECIAL PATHOLOGY.

(3.) As diseases which may be studied in their primary elements, forming thereby the science of GENERAL PATHOLOGY.

But, in whatever aspect we may view disease, there is invariably presented to the student the five following subjects for investigation, namely,—

First, The morbid phenomena or symptoms by which the patient and the physician become aware that disease exists. Some of these can be appreciated only by the patient; such, for example, as *pain, dizziness, impairment* or *alteration* of the functions of the *special senses*. These are known as SUBJECTIVE SYMPTOMS. They indicate the commencement of many serious diseases, but from their nature they are apt to be deceptive alike to the patient and to the physician. The feeling of chilliness of which a patient complains during the cold stage of ague, or in the rigors of a commencing fever, is calculated to make not only him, but those around him, imagine that the temperature of the body is *below* the normal standard, whereas the objective evidence of the thermometer shows that it is actually increased. The intrinsic value of subjective symptoms will vary according to the truthfulness, the emotions, and the descriptive power of the patient, who may unconsciously exaggerate such subjective symptoms; and whenever there is reason to suspect that he may have a motive in deceiving, we must trust mainly, if not solely, to objective symptoms, and a careful investigation into the general surroundings of the case. At the same time, it is necessary to keep in mind that the commencement of many serious diseases is indicated by subjective phenomena alone. Of late years, the practical tendency has, perhaps, been too much to ignore these subjective symptoms, and to pay too little attention to complaints which cannot be seen or demonstrated by means of the stethoscope, microscope, or thermometer. (MURCHISON; *Brit. Med. Journ.*, Jan. 6, 1872.)

Certain symptoms, again, are appreciable by others, and may or may not be also obvious to the patient; such as, *jaundice, alteration of temperature, of pulse, or of breathing*. These are known as OBJECTIVE SYMPTOMS, and are the most important *physical signs* for the physician to recognise and appreciate at their true relative value.

Taken individually the same symptoms are common to many diseases; but when several symptoms constantly occur in groups together, or in a uniform succession, they then point to definite and specific diseases; and so become certain *signs* of such diseases. Certain symptoms or groups of symptoms are thus peculiar to, or distinctive of, certain diseases, so that whenever they appear they indicate with certainty a particular disease, and so clench the diagnosis, if any doubt existed before. Such symptoms are said to furnish *pathognomonic signs* of disease—*e.g.*, the peculiar eruptions of some specific diseases when super-added to the febrile phenomena and other symptoms. Again, symptoms

are still further distinguished as *general* or *constitutional*, because they affect or pervade the whole system, and are significant of general or constitutional disease.

Second, The agents by which diseases are produced, generated, or brought about, constituting the department of *Etiology*, or *causes of disease*. To know the cause, and to be able to remove it, is often the first step towards a cure, and, in regard to many diseases, the most certain means of preventing their development and propagation (MURCHISON). The causes of disease are usually classified into—(1.) *Predisposing*; (2.) *Exciting*; and (3.) *Proximate*.

The *Predisposing Causes* are those which favour or which render a person unable to resist the (morbific) influences or causes which immediately excite disease. They comprise all those conditions which so modify his health as to render him apt (or predispose him) to contract the disease to the specific influence of which he may be exposed; and thus they are generally for a long time in operation before the disease appears. The *predisposing causes* thus favour the action of the immediately exciting causes, by the influence which they exercise on the constitution during their prolonged action.

The predisposing causes of disease embrace a consideration of the following influences:—

1. *Age*.—From the earliest period of infancy to that of puberty there is a peculiar proneness to various maladies, especially, first, to derangements of the mucous membrane and glands of the alimentary canal and epileptiform convulsions. Childhood, during the first few years, is liable to rickets, which, when once developed, may continue during the whole period of growth of the osseous system; also to hypertrophic paralysis, and true spasmodic asthma. From eight to fifteen years of age is the period when attacks of chorea and of epilepsy are most apt to occur. Enteric fever is peculiarly a disease of youth and manhood; and as the decline of life approaches, decay and degenerations of tissues and organs bring about symptoms of various disease-processes going on in the nervous system, heart, lungs, great blood-vessels, or kidneys and liver.

2. *Sex*.—Besides the differences necessarily associated with the peculiar structures and functions of the sexual organs, there are some diseases to which women are more prone than men; such as, chlorosis and hysteria, erythema nodosum, exophthalmic goitre, and goitre itself.

3. *Hereditary transmission of personal peculiarities and proneness to certain ailments*.—The transmission of the tendency to certain diseases from parents to children is only part of the general fact of the influence of constitutional or anatomical peculiarities of parents on their offspring (*heredity*). A knowledge of the personal constitution of the patient is, in this respect, of great value to the practitioner, and not less so to the patient himself, as showing the importance of avoiding, or fortifying the constitution against, exciting causes of disease which very often co-operate with the hereditary tendency to produce it. The *predisposition to disease from hereditary constitution* is exemplified in the following affections, namely:—Gout, rheumatism, scrofula, tuberculosis, syphilis, cancer; certain forms of diseases of the nervous system,

such as epilepsy, mania, melancholia, hypochondriasis, neuralgia, paralysis, chorea ; also various other conditions, such as physical deformities, and impairments or absence of organs or parts of special sense, degenerations of blood-vessels, premature decay, baldness, loss of teeth, various skin diseases, especially psoriasis, acne, ichthyosis, emphysema, asthma, diabetes, gravel, and calculous disorders. In some instances a few of these inherited diseases are known to appear at birth (*congenital*), having developed themselves while the foetus was still *in utero* ; while the appearance of others is more frequently determined by the application and concurrence of other causes—"exciting causes." *Atavism* is the name given to the occurrence of hereditary disease when it passes over one generation in a family history without affecting any of its members, and then appears in the next generation ; or the inherited diseases may appear, as it were, sporadically in families, so that some in a family may become affected and others escape, the affected and the unaffected procreating indifferently healthy and unhealthy offspring. It is also to be observed that the inherited tendency to disease does not in all cases manifest itself in an exact reproduction of the morbid peculiarity of the parent. There are certain functional disorders which run in families, and which may be said "to alternate, so that a parent suffering from one of them may beget children in whom one or other of the remaining members of the group replace, as it were, the particular parental malady" (BRISTOWE). These alternating nervous disorders are, especially, insanity, epilepsy, hysteria, asthma, and neuralgia. The marriages of near kin, as investigated by Huth, ought to be a subject of still more extended study in their influence on health and disease. Many erroneous notions prevail regarding them as causes of disease ; and Mr. Huth's work shows that the statistics on which so much reliance has been placed as a proof of the harmfulness of consanguineous marriages are, when not absolutely false, miserably misleading and defective. (*The Marriage of Near Kin*, p. 353.) *Idiosyncrasies*, or special individual peculiarities, are also often inherited, such as the susceptibility to the influence of the emanations from fresh hay in the production of hay-asthma, of the smell of ipecacuanha and of many flowers in causing nausea, of the specially poisonous action of many drugs, such as opium and other drugs in the smallest doses, and even of such wholesome food as mutton ; and also the peculiarly unfortunate susceptibility which some persons seem to possess of contracting any of the contagious diseases, commonly called "catching," to which they may be exposed, "and even to take the same one over and over again, while other persons seem always to escape." (BRISTOWE.)

4. *Certain occupations, habits of life, quality of food or drink, over-indulgence, abstinence, privation.*—Miners, knife-grinders, and masons, for example, are prone to special forms of destructive lung disease ; painters and workers in lead to the specific phenomena of lead poisoning, such as wrist-drop and colic. Exposure to noxious gases or vapours, as carbonic acid, sulphuretted hydrogen, lead, copper, phosphorous fumes ; over-work, inordinate use of one limb or set of muscles, night-work, long hours, all show the influence of occupations as predisposing to disease ; while the want of occupation suggests privation. Habitual intemperance in the use

of alcohol deranges the digestive organs, and so leads to cirrhosis of the liver, and to other changes of structure and degeneration of it and of other organs; to impairment of function, and to the development of a peculiarly characteristic subcutaneous fat, especially in the abdominal region, and to similar omental fat, and especially to *delirium tremens* and chronic alcoholism.

Indigestion, constipation, and undue accumulation of fat are also the lot of those who lead a sedentary life, while insufficient muscular exertion predisposes to general plethora and constipation of the bowels, to hemorrhoids, to other symptoms of portal obstruction, and to gall-stones. With regard to over-indulgence in food, it is consistent with experience that many cases of hyperæmia, bilious congestion, and enlargement of the liver, with increase of cell growth and connective tissue (but without tendency to abscess), and enlargement and partial fatty degeneration of the liver cells, are caused simply by diet. Annesley, Macnamara, Norman Chevers, have fully recognised its influence in predisposing to disease in India. We eat much more than is necessary for healthy nutrition. Any excess in quantity and many alterations in quality (especially commencing decomposition in the albuminates, and perhaps rancidity of fatty substances), predispose to diarrhoea and to dysentery; and, generally, the diseases in which food in some way acts as a predisposing cause, form perhaps the most numerous order of diseases—diseases of nutrition—with which the physician has to deal (see PARKES' *Hygiène*, 5th Edition, p. 203). Dyspepsia, constipation, and irritation causing diarrhoea (which does not always empty the bowels) are produced—fæcal fermentation takes place, and putrid substances are absorbed, causing fever due to blood poisoning, torpor, and heaviness of mind, fœtor of breath, and sometimes jaundice. A predisposition to gout is no doubt engendered from the use of liquids which delay metamorphosis, and from indigestible articles of food.

The influence of deficiency of food in predisposing to diseases is well known in the long catalogue of ailments which follow famine, characterised especially by feebleness, inanition, and the occurrence of relapsing fever. How far deficiency in any one of the four great classes of aliments—namely, albuminous substances, fatty substances, carbohydrates, and salts—predisposes to disease has been the subject of definite experiments. Each of these classes seems to serve different purposes in nutrition, and all of them are in due proportion necessary for perfect health. Dr. Parkes found that complete deprivation of albuminates, without lessening the other classes, produced marked effects only after some days. In a strong man fed on fat and starch only, full vigour was maintained for five days. In a man on whom the amount of nitrogen was reduced to one-half, full vigour was retained for seven days; but if abstention from albuminates is prolonged beyond these periods, there is eventually great loss of muscular strength, mental debility, fever, and dyspeptic symptoms. Then follow anæmia and great prostration; elimination of nitrogen, as urea, greatly lessens, though it never ceases, while uric acid diminishes in a less degree. But if starch be largely supplied, the weight of the body does not lessen for seven or eight days (HAMMOND, PARKES, p. 204). If the deprivation of albuminates be less complete (70 to 100 grains of nitrogen being given daily), the body gradually lessens in activity of

function, and passes into an adynamic condition, which predisposes to the attacks of all the specific diseases (especially of malarious affections and typhus), and of pneumonia, and modifies the course of some of them, such as typhoid, which then runs its course with less elevation of temperature than usual, and with less or no excess of ureal excretion. The deprivation of starches can be borne for a long time if fat be given, but if both fat and starches are excluded, though albuminates be supplied, illness is produced in a few days. Men can be fed on meat for a long time, as a good deal of fat is then introduced, and if the meat be fresh scurvy is not readily induced. The deprivation of fat is not well borne, even if starches be given; but the results are not known. The great remedial effects produced by giving fat in many of the diseases of obscure malnutrition, prove that the partial deprivation of fat is both more common and more serious than is supposed; and in all diets ordered for soldiers and for prisoners the fat is greatly deficient in every country (PARKES). The deprivation of salts is also attended with marked results, which are worthy of more attention than they have yet received. Bad effects also result if the intervals between meals are too long, and by conditions of indigestibility of food, either dependent on physical or chemical conditions of the food itself, or of the digestive fluids. To some persons certain foods are indigestible at all times, or at particular times. The retention of indigestible food leads to chemical changes and putrefaction, which go on in the stomach and bowels under the influence of warmth, moisture, and air. Irritation of the mucous membrane throughout the alimentary canal is produced, and so predisposes to dyspepsia, diarrhoea, and dysentery. Indigestibility of food also tends to allow albuminates to pass into the circulation less properly prepared to undergo the action of the liver, and so produces irritation in the liver, and then passing into the blood in some unassimilable state produces irritation of the skin or kidneys. Sometimes albumen appears in the urine as if it had circulated like a foreign body in the blood. Such conditions point to some error in primary digestion, occasionally not obviously accompanied by any gastric derangement.

In any form of disease in which the functions of nutrition are impaired, it is therefore necessary for the physician to inquire regarding the food in the following respects:—

(1.) Is the food excessive or deficient in quantity as a whole or in any of the four primary classes of aliments? On these points the student is referred to PARKES' *Hygiène*, edited by my colleague, Professor DE CHAUMONT, for the best methods of calculating out diets, p. 191, 5th Ed.

(2.) Are the different articles digestible and assimilable; or, from some cause inherent in the food, or proper to the individual, is there difficulty in primary digestion or want of proper assimilation?

(3.) Is the quality of the food altered either before or after cooking?

5. *Debility, impairment of constitution, vulnerability of the system, and the cachexia* of previous diseases modify in a remarkable way the predisposition to subsequent attacks of disease. Many inflammations tend to repeat themselves, such as erysipelas, rheumatism, pneumonia, bronchitis, tonsillitis, catarrh. A liability to other diseases is also sometimes engendered by the previous occurrence of others. Scarlet fever is apt to engender

renal disease; scarlet fever and gonorrhœa are also apt to be followed by rheumatism of an acute kind, and by ophthalmias. Destructive lung disease is apt to follow or be engrafted on enteric fever, small-pox, dysentery, diabetes, and pyæmia; scrofulous enlargement of the cervical glands, on mumps and measles; and chorea as a sequela to acute rheumatism and scarlet fever; while sarcomatous and other kinds of malignant growths are apt to appear on places which have been the site of repeated or continuous attacks of inflammation (BRISTOWE).

Certain organs and tissues also seem to have a special predisposition to suffer from certain diseases; for example, the primary manifestations of carcinoma are more often in the uterus, mamma, and particular regions of the alimentary canal, than in other parts of the body. The inflammation of rheumatism and gout especially affects the tissue of the white ligaments and the soft parts of the joints, while these diseases have almost nothing else in common; and specific lesions of various parts are characteristic and peculiar to the several specific infectious fevers. Excessive or repeated evacuations, either of pus or blood, or of the serous portion of the blood, as by menorrhagia, leucorrhœa, the discharge from an issue connected with diseased bone, or from piles, hyperlactation, are common causes of general debility, predisposing to albuminoid degeneration of liver, spleen, kidneys, or intestines.

6. *The influences of climate*—that is, of heat and cold, of dryness and moisture, of atmospheric impurities—are well recognised agents in predisposing to disease. The lowering of temperature has an obvious effect in the production of bronchitis, pneumonia, and rheumatism; and especially during winter and early spring, the death column in the newspapers shows the effects of cold in the obviously increased mortality from those diseases, and especially from acute pneumonia amongst aged patients. Protracted hot weather, with little of the invigorating influences of occasional reductions of temperature, tends to induce general debility. The liver and digestive mucous membrane are prone to suffer from too long continued exposure to heat; hence diarrhœal affections prevail in summer. Dampness, or excessive moisture of the atmosphere, predisposes more especially to rheumatism, to destructive lung diseases, to croup and ague. The deficiency of the natural stimulus of pure air, as in those who sleep or work in overcrowded or badly ventilated dwellings, and who are thus constantly breathing vitiated air, tends to induce general debility, sickness, and mortality amongst children at very early periods of life; and after puberty, to a proneness to the many diseases of nutrition which, in the proper sense of the word, are yet undiagnosed. The conditions of the soil and water, as local agents in predisposing or actually exciting endemic disease, must not be lost sight of in such diseases as goitre, cretinism, guinea-worm, elephantiasis, pellagra, malaria, cholera, dysentery, and enteric fever.

The exciting causes of disease are those which especially act mechanically, or chemically, or which are specific and vital. The predisposing causes of disease (with the exception of those numbered 1 and 2) are also often the immediately exciting causes.

As examples of mechanical causes, the following may be named:—obstructions of orifices or tubes, either by thickening or contracting of

their walls, or by pressure on them from without, or by impacted concretions. Hence we have stricture, hernia, intussusception, lodgment of gall-stones, and similar obstructions in the liver, pancreas, kidney tubes, and calyces, ureters and bladder, larynx, trachea and bronchial tubes, at the cardiac orifices, and in the blood-vessels. They include also impediments, however originating, to the transmission of nerve currents along the nerves, dilatation of arteries and of other tubes and cavities, perforations or ruptures of their walls, extravasations or effusions of serum, and the like; also the occurrence of thrombosis and embolism.

As examples of chemical causes are the mineral, vegetable, and, perhaps, snake poisons, which undergo absorption, and exercise their specific effects on special organs, or on the system generally. Some are inhaled or absorbed by the skin, or otherwise continuously introduced in minute quantities; such as lead by painters and workers in lead, bringing on plumbism or chronic lead poisoning; the inhalation of mercurial vapours bringing on muscular tremors and mercurialism; the fumes of phosphorus leading to acute yellow atrophy of the liver, and to necrosis of the bones of the jaws; ergotised food leading to a peculiar form of gangrene of the extremities; the special poisonous action of some mussels, fungi, sausages and fish, of tobacco, alcohol and opium, must be considered as exciting causes of disease. Certain general diseases are also excited and gradually developed by the constitutional origin of some chemical products within the body as a result of the defective action of some of the excreting organs. The retained effete matters accumulating in the blood then act as poisons, inducing epileptiform convulsions, dropsy, anæmia, jaundice, and cancer.

The peculiar and specific disease-poisons are vital in their nature, giving rise to the well-known specific diseases whose symptoms and course are as constant, uniform, and specific now as of old; such as, small-pox, measles, scarlet fever, typhus fever, typhoid or enteric fever, relapsing fever, malarial fevers, specific yellow fever, syphilis, hydrophobia. The specific nature of the exciting causes of these diseases will be considered further on.

Proximate causes of disease are largely represented by those mechanical conditions which are established as local lesions, and which cause especially inflammatory thickenings, ulcerations, or to long antecedent attacks of previous disease, as when rheumatism is followed by cardiac lesions.

Observations as to the causes of disease are best made on large bodies of men—as in the experience of military practice, bodies of police, prisoners in gaols, and the like. But there are certain elementary facts now well established by ample experience, by occurrences within individual observation, and by special statistical returns, which may be here enumerated as suggesting sources and causes of disease, namely:—(1.) The greater amount of disease and mortality in large towns, and especially in the worst aired parts of towns, as compared with agricultural districts; (2.) The greater amount of disease in seasons of scarcity, as compared with seasons of plenty; (3.) The greater amount of disease amongst the poorer classes of society, and of hot or very cold climates, as compared with those in comfortable circumstances, and habitually protected from the extremes of temperature; (4.) The greater amount

of disease amongst convalescents from acute diseases, compared with persons previously healthy; (5.) The greater amount of diseases amongst the intemperate, as compared with the sober; (6.) The greater amount of disease in beaten armies, or among depressed and disheartened individuals, as compared with victorious armies, or more fortunate, flourishing, and well-to-do members of society; and, (7.) Generally, it may be stated that no circumstance in the condition of mankind so uniformly increases mortality and promotes disease as destitution, particularly in large towns; (8.) More or less of a contagious property is possessed by certain specific diseases, namely,—small-pox, chicken-pox, measles, scarlet fever, typhus fever, enteric fever, relapsing fever, dengue, dysentery, influenza, cholera, certain parasitic diseases, syphilis, gonorrhœa, soft chancres, purulent ophthalmia. But in ascribing this property of contagion to these and other diseases, it is not to be understood as determined that contagion is the only exciting cause of any one or all of them. These diseases also are not equally nor alike contagious one with another, nor does the contagious property exercise its influence similarly in all. Some of them may occasionally *originate* from causes still not definitely determined, and may extend themselves in an unknown way, besides being propagated by contagion; and with regard to some of the forms of fever originating in malaria, there is some evidence to show that they may occasionally, and under certain circumstances, spread by contagion, especially the malarious form of yellow fever.

Third, The seats or localities of disease, or of derangements, constituting *Pathogeny*. Here the peculiar nature, general forms, and types of disease must be studied, together with varieties in their course, duration, and termination.

Fourth, The morbid alterations discoverable in the structure of the body before, but more especially after death, constituting *Morbid Anatomy*. These alterations must be studied in connection with the symptoms, the causes, and the course of the disease.

Lastly, The elementary constituents of disease products, constituting *Morbid Histology*, must be recognised and contrasted with analogous constituents of the body in the healthy state.

CHAPTER V.

OF MORBID PHENOMENA, SYMPTOMS, AND SIGNS OF DISEASE.

It has been stated that only by a mental effort is the student or physician able to convert *symptoms* into *signs* of disease, because symptoms cannot become signs unless they are understood, and their significance interpreted. Therefore the idea associated with "*sign*" is of a much more comprehensive kind than that which is connected with the word "*symptom*:" the former implies the possession of more extensive knowledge—a knowledge such that comparisons may be instituted amongst the symptoms which

present themselves. Certain symptoms of disease, or of disordered function, are thus recognised to be peculiar, characteristic, or *significant* of a particular morbid state. A *symptom* is thus converted into a *sign*, and what is called a *diagnosis* of the disease is made.

Symptoms and signs of disease derive their importance from the fact that they are capable of being connected with lesions of structure or disorders of function; and both of these conditions mutually act and re-act upon each other. Thus it is that such mutual reaction greatly aggravates any general disease. In place of the concurrent exercise of function, and the mutual co-operation of parts in a state of health, both as regards mind and body, we have *symptoms* of disease expressed in various ways, characteristic of the function at fault, and incompatible with the normal existence of the part or organ affected, or of the body generally. Hence a diagnosis becomes necessary not only as to the nature of the disease, but also as to the nature of the lesion or disorder of function. From such phenomena the physician makes up his mind,—(1.) As to whether or not disease exists. (2.) How far the condition of the patient is removed from the state of health usual to him. (3.) As to the nature of the disease, and how it is distinguished from other ailments, or in what respects it may differ from the same ailment in other people in similar circumstances. Thus a *diagnosis* is made by the art of converting *symptoms* into *signs* of particular diseases, and so discriminating one disease from another. But the physician at the same time generally carries his mental exertion a little further. He tries to arrive at a just estimate of the probable result or *event* of the malady, and so makes up his mind,—(4.) As to whether the illness will terminate in the death of the patient, in permanent organic mischief of greater or less extent, in persistent impairment of the general health (*cachexiæ*), or in complete recovery. As in Politics, so in the Science of Medicine: the politician and the physician have each to deal with the future as well as with the present. Both endeavour to *forecast* events; and thus, in the Practice of Medicine, we are said to make or give a *prognosis*. (5.) The physician must be able also to appreciate with reasonable rapidity those *symptoms* which are peculiar or distinctive, and to recognise them when associated together as the *signs* of particular or definite morbid states. (6.) The physician must further discriminate and try to put a fair and just value or interpretation upon those symptoms which are only experienced by the sensations (subjective) of the patient himself, as contrasted with those which may be seen or appreciated by others, such as objective phenomena or physical signs.

The interpretation of symptoms can only be successful after a close observation of the patient—often prolonged, and repeated for more complete investigation—so as to connect the results arrived at with his previous history. The utmost logical acumen is required for the due interpretation of symptoms. The individual value of each ought to be duly weighed; one symptom must be compared with another, and each with all; while the liability to variation of a similar symptom in different cases of a like kind must not be forgotten; and the occasional absence of the usual pathognomonic signs may be sometimes calculated upon. Thus only can the nature of a disease be clearly determined—its severity and dangers fully appreciated—its treatment indicated, and the probability of recovery foretold.

CHAPTER VI.

"CASE-TAKING," OR THE METHODICAL EXAMINATION OF PATIENTS AND
THE RECORDING OF MEDICAL CASES.

A CLOSE study and observation of the general symptoms of diseases, in all their details, is absolutely necessary for their accurate diagnosis; and the investigation is aided practically by the improved instruments of the present day, and the better methods of examining patients. Above all things, *methodical examination* is essential, if the student would acquire the habit of carefully and accurately learning the nature of the cases of disease with which he will have to deal. Patients must be examined methodically, in order that the symptoms of disease may be correctly interpreted, and that nothing be overlooked or neglected.

The systematic examination of patients and recording the results of the inquiry in writing, commonly called "case-taking," should be practised from the earliest period of clinical study. Clinical case-books are of great assistance.* To elicit the facts of a case with facility and precision requires continual practice. Some well-prepared plan of examination is necessary to accomplish this; and such a plan should be adopted and followed out by every student as the best method of training to that accuracy of observation, which is at once a necessity and a leading feature of the practice of medicine of the present day. If the attempts of a beginner are watched, who sets about the examination of a patient without any definite method, it will be seen that he wanders in his inquiries from one part of the patient's body to another; that he is attracted by varied and different symptoms; that he asks pointless and fruitless questions; and that he finally jumps at hasty conclusions, which are generally erroneous; or that he fails altogether to make up his mind as to the nature of the case he has before him.

Directions have been given by many authorities for acquiring and habitually following a definite system of examining patients, especially as to what are the essential points to be brought out and recorded in "case-taking." A skilful practitioner may be able to get at the facts and learn the true nature of a case in any order, or in no specific order; yet it is very desirable that a regular order should be followed by learners. The

* These may be had suitable for reporting all kinds of cases, or for special purposes. (1.) Mrs. Garrett Anderson, M.D., has published (Lewis & Co.) a clinical note-book for reporting diseases peculiar to women, for the use of the lady medical students at the Hospital for Women. (2.) Mr. Spencer Wells has published (Churchill & Co.) a specially prepared note-book for Ovarian cases. (3.) Mr. Rowell, of the Leeds Hospital, has published (Smith, Elder & Co.) a valuable little book consisting of a large number of diagrams in outline of each part of the body, for rapidly illustrating note-books, by adding the required indications with the pen, in which are included outlines of the abdomen and pelvic organs. These outlines have adhesive backs, so that they can be stuck into any note-book. (4.) The author of this work has also published (Griffin & Co.) temperature charts and outlines of the body, which will be found similarly useful.

habit so acquired will eventually become a necessity, and will be found most useful in practice.

A combination of a *regional* or *physiological* method seems to be the simplest for beginners, inasmuch as it ensures a thorough investigation of the whole organism—a plan which has been so well expounded by Professor Sanders, in *Edinburgh Medical Journal* for November, 1873. The student being already familiar with the division of the body into anatomical Regions, the Systems of organs are to be examined in connection with the anatomical region to which they respectively belong. The method has also this advantage, that the existing symptoms of disease will come to be arranged in such scientific groups as will most readily and naturally lead to the detection of the causes which account for them, and nothing can be neglected or omitted which may throw light on the nature of the patient's ailments.

The following is the outline or skeleton plan for "*taking a case*" according to this method; and suggestive hints are added under the several appropriate headings for the kind of information to be sought for in the examination of each region and system of organs.

The *order or method of examining* a case should be the same as the *order of recording or reporting* the results in a case-book; and the written description ought to be a full and accurate statement of facts, as seen, heard, felt, or otherwise appreciated by the examiner. A most distinguished clinical teacher—the late Dr. Latham—truly observes that "each case is made up of its own facts; and the facts alone must teach the nature of the disease and suggest the remedy" (*Clinical Lectures*, p. 114, Lect. v.) No theory or conclusion ought to be embodied in the history of a patient's case—all conclusions or theories must be worked out at the termination of the inquiry, and embodied under the heading of "*Diagnosis*." For example, it is not admissible here to record the existence of *tubercle*, or *inflammation*, or *cavities* in the lungs. It is only admissible to state any symptoms or physical signs which have been discovered in the lungs.

But before subjecting any patient to such a prolonged systematic examination as is set forth in this chapter, it is necessary to observe and determine the following points:—

- (1.) Whether the case is obviously a *febrile* or a *non-febrile* one—as shown by the temperature of the skin and the number of pulse-beats.
- (2.) Whether the patient is sufficiently intelligent as to be able to answer questions.
- (3.) Whether the patient is in a condition as to bodily strength to undergo the fatigue of a protracted physical examination.

If it is found that the patient suffers from fever, or is delirious, or is in a weak or dangerous condition, the facts should be recorded, and the further course of examination must be modified accordingly.

Skeleton Form or Scheme for Examining and Recording a Case.

DATE,

DISEASE, RESULT,	{	Here the <i>name</i> of the disease and <i>result</i> of the case can only be filled in after the case is completed.
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I. Preliminary Information.

1. *Name*, (and of course) *sex*, *age*, *occupation* (see pp. 8, 9, *ante*), sedentary or active—in or out of doors—how many work-hours per day; *residence*, at home, number of persons in the same room—married or single.

2. *Complaints* (*a.*) as stated by the patient himself, or (*b.*) as reported by his friends; (*c.*) any very obvious morbid appearance, such as *jaundice*, *anæmia*.

3. *History* (*a.*) of present ailment; (*b.*) of previous healthiness or diseases; (*c.*) of social condition and habits, lodging, especially as to efficiency of shelter and clothing; condition of drainage, and water supply; food, lived well or badly—amount of meat and fresh vegetables; drink, temperance, or intemperance—if intemperate, the degree of, and whether occasional or habitual—the amount or kind of stimulant employed—the previous occurrence of *delirium tremens*—the use of opium, tobacco, chloral hydrate, or chlorodyne—personal cleanliness as to baths; irregularities as to living, or excesses of any kind. The patient ought to be asked to enumerate any previous diseases; and then special inquiry should be made as to the following:—namely, small-pox, cow-pox, pertussis, scarlatina, measles, rheumatism—acute or chronic—rheumatic or neuralgic pains, palpitation of the heart, winter-cough, hæmoptysis, epistaxis, hæmatemesis, indigestion, piles, gonorrhœa: the previous or *present* existence of syphilis and use of mercury, exposure to contagion or infection, with inquiry as to the existence of epidemic diseases; exposure to extremes of heat, cold, or wet in various climates; length of residence in extreme climates; (*d.*) family history, especially as regards parents, brothers, sisters, and children, with reference to any history of diseases amongst them known to be hereditary. (See p. 8, *ante*.) Hereditary predisposition is often difficult to trace; and hereditary disease may not be acknowledged, merely because the patient may be quite ignorant of the subject and meaning of the questions put. The leading points to be noticed are—(*a.*) Evidences of *scrofula* in fair or dark complexions, such as the thick upper lip and broad expanded nostrils, dilated sluggish pupil, and clubbed fingers and nails. (*b.*) Syphilitic cachexia, shown by eruptions with cicatrices at the angles of the mouth, also of nose and prominences of face; pegged teeth, state of the glands, sunken nose, loss of hair; thickening and cicatricial-like pale contractions of skin in patches. (*c.*) Evidences of gout in the enlarged joints, deposits in tragus, bursæ knuckles; deposits of red gravel in urine; attacks of faintness, palpitation, dyspnœa, acid indigestion, irritation of kidneys or of bladder. (*d.*) Rheumatism, insanity, asthma, cancer, and tuberculosis should also be inquired into. Previous health in general terms—good or bad. If a female, at what age catamenia appeared first, second appearance, when quite regular, regular or not since, and scanty or profuse; how many children or abortions, labours easy or the reverse, any flooding, suckling own children—when catamenia ceased.

The inquiries regarding the history of the patient should be instituted with special reference to the causes of disease as given at the end of a previous chapter. (See p. 12, *ante*.)

By some the medical history of the patient is taken last,—that is,

after finding out the disease—as practically less inconvenient than taking the history first. This was Dr. Latham's method after trying both. His reasons for selection are thus stated:—"If you first learn the existing complaint, you know how much of its previous history you will require to illustrate it; but if you first inquire the history, since you do not yet know what it is to illustrate, you cannot tell how much of it you shall want, and must allow the patient to tell what he thinks fit; and since every person's complaint is interesting to himself, he is apt to discourse about it rather too much at large, and too little to edification."

Patients ought to be allowed to begin the account of their ailments in their own way. They ought also to be requested to give a date, if possible, to the very first deviation from health which they experienced, and how this was manifested; the order of occurrences since that time, and the origin of any new symptoms. The story of the patient must then be subjected to such cross-examination as the nature of the case suggests, in order to obtain accurate data. The questions must be put with mildness, and the replies listened to with kindly interest; and all personal examinations must be conducted with prudence, delicacy, and gentleness of manipulation.

The first inquiry should be as to what direction present sensations point to as evidence of disease, always bearing in mind that sometimes symptoms seem to point to one organ when another is actually diseased; such, for example, as vomiting, significant of cerebral lesion, or of the presence of a renal calculus, or of pelvic or abdominal tumors, or of pregnancy; pain in the right shoulder, significant of hepatic disease; pain in the region of the sacrum, of disease of the uterus; pain in the thigh and testicle, as significant of nephritis; pain in the knee, from disease of the hip; or in the urethra, from stone in the bladder. Such pains and symptoms are sometimes called *sympathetic*. The patient is always apt to have some theory or belief as to his ailments; and hence two important questions ought always to be put to him in a particular form, so as to elicit two important facts. These are—(1.) "*How long have you been ill?*" and (2.) "*Where do you feel pain or discomfort or uneasiness?*" To obtain a reply, as definite as possible, to the latter question, the patient ought to *indicate the seat of pain by placing the hand over the region*. If pain thus indicated by the hand is alleged to be circumscribed to a part of the surface, the place referred to ought at once to be examined by sight and by pressure. With reference to the first question—how long the patient has been ill—the period of commencement is usually fixed as regards acute inflammation and febrile diseases by finding out when the first rigor or chill was noticed, remembering also that sometimes convulsions take the place of rigors. Sometimes sickness or pains, headaches or feverishness, sudden or gradual, may mark the onset; but in chronic ailments a good deal of inquiry is necessary to get at the truth regarding the first beginnings of disease. The patient ought to be called upon to state what he feels to be the matter with him. The answers should be taken down in his own words. No leading questions should be asked. Lastly, the patient should be questioned concerning the following subjective symptoms, unless they have been mentioned before:—Pains, aching and soreness, feelings of uneasiness or prostration, flushes, coldness or shivering, tingling or

burning, alterations of vision, hearing, touch, taste, smell, numbness of extremities, altered or impaired cutaneous sensibility, dyspnoea, cough or desire to cough, palpitation, choking, dysphagia, degree of appetite, thirst, nausea, desire to micturate, griping, tenesmus. The following inquiries must then be instituted as to :—

II.—Present Condition—Regional Examination.

This is to be determined by (A.) “EXTERNAL (REGIONAL) INDICATIONS;” and (B.) “INTERNAL (REGIONAL) EXAMINATION.”

(A.) “EXTERNAL (REGIONAL) INDICATIONS.”

These are to be inquired into under the following heads:—

(1.) *General Condition* as to conformation or development (tall, slight, short, or square); colour of hair and eyes; temperament (sanguine, nervous, phlegmatic, bilious); habitual state of mind (cheerful or melancholy). Height and weight ought to be accurately determined, and condition as to muscularity made a note of; also, bulkiness, nutrition of body, or emaciation; any loss of weight or of strength should be inquired into; posture or attitude of the body, as indicative of debility or of helplessness, is to be noted. A bulky aspect suggests either corpulence or general tumefaction. If bulkiness is most expressed in the abdominal region, it may be due to omental fat or to ascites. General bulkiness of the body, when not due to corpulence, may be tumefaction, due to the presence of fluid or air in the areolar tissue. If due to air, the tissue will crepitate under the fingers; if due to fluid, the skin will pit on pressure. A history of diminishing bulk suggests wasting, which, if continuous, generally points to some impairment of nutrition, the cause of which is to be sought for. Such wasting occurs during protracted febrile states, the most chronic abdominal diseases, and in slow but malignant or deadly diseases—cancer, diabetes, phthisis; and when discharges are continuous, as in chronic dysentery, diarrhoea, and suppurations. The loss of flesh (emaciation) is more marked and excessive in chronic dysentery than, perhaps, in any other disease. It becomes most obvious in the face by the subsidence in certain parts, as the tissue about the eyeballs and the zygomatic arch, from the absorption of fat; and the prominences of the long muscular bundles in the neck, and their attachments.

With regard to the position, posture, or attitude of the patient, it should be observed whether and how he lies (mode of *decubitus*); and whether and how he walks, as suggestive of impaired health or acute disease. A sudden and imperative obligation to lie down in bed, suggests an acute, a severe, and perhaps a dangerous illness. Lying for a long time on the back (*dorsal decubitus*) is suggestive of *exhaustion*, and in some fevers—of *paralysis*, or *pain increased on pressure or motion*, such as that of *peritonitis* or *rheumatism*. Lying on one and the same side, suggests impaired action of the lung on that side; respiration being carried on with the sound lung of the opposite side. But this rule is not absolute. Inability to lie down, and the obligation to sit up more or less erect, suggests a form of *dyspnoea*,

or distress in breathing, unless when sitting erect or leaning forward. To this condition the name of "*orthopnœa*" has been given; and it is met with especially in diseases of the heart, œdema of the lungs, or extensive effusion into the pleural cavities in laryngitis, congestive or asthenic pneumonia, and in severe cases of epidemic influenza. A restless mode of lying down accompanies severe thoracic inflammation and acute rheumatism. The position of the head, shoulders, trunk, and limbs ought each to be separately observed; the head, as to whether it lies low or hangs over; the body, as to whether the patient lies prostrate, motionless, flat on the back (*supine*), or on the face (*prone*); the limbs, as to whether they are outstretched, flaccid, and relaxed, or drawn up, bent, or fixed in any definite position. It ought to be noted also whether the patient has put himself into any particular posture to relieve pain or pressure upon a part which gives pain. The movements of a patient when still able to walk about are to be noted as to erectness, quickness, and firmness of gait, or the reverse. Weakness from illness is associated with a stooping, slow, and difficult gait. Stooping, irrespective of age, is suggestive of spinal disease, or some affection of the larger joints of the lower limbs. A staggering gait suggests *cerebral disease*; laborious or one-sided movements suggest *paralysis*; uncontrollable or excessive movements are suggestive of *chorea* or *mania*; trembling motions point to *extreme debility*, *blood poisoning of the typhoid state*, or to *delirium tremens*.

(2.) *Aspect of the Features, and Expression of the Countenance.*—The patient should be so placed that the light falls fully on his face. Pallor, as of anæmia, chlorosis, or shock, is to be distinguished one from the other; so also the localised flush of *hectic* over the malar bone in phthisis, specific fevers, and pneumonia; the duskiness, cyanosis, and lividity suggestive of cardiac and pulmonary mischief. A pallid, white, glazed, or pasty look of the skin of the face, is suggestive of certain forms of renal disease; and, combined with puffiness, or œdema of the skin, over the eyelids and underneath the eyes, points to the existence of Bright's disease, or chronic valvular disease of the heart.

The *expression of the features* may suggest *shock*, as of injury, when combined with pallor, anxiety, and a frightened look. The eyes may then be fixed or oscillating, with a contracted pupil. The *expression* also may be simply that of pain or anxiety, or unnatural indifference; or it may be suggestive of forms of mental diseases, such as *mania*, *melancholia*, or *dementia*; or of other nervous diseases, such as *epilepsy* or *hysteria*. The *expression of feature* may be different on the one side of the face compared with the other. A serious or dull expression may exist on *one side*, while the other side is perhaps playfully expressive, as in laughter. Such features indicate *facial hemiplegia* or *paralysis*, with or without muscular twitching, commonly called "*Tic*," and with or without convulsions, squinting, or alteration of the pupils. An expression of stupor or apathy, with a dull and listless eye, paleness on sitting up in bed, or flushed if lying down in bed, is common in febrile states of a typhoid type, with black accumulations, called *sordes*, on lips, gums, and teeth.

A bluish or cyanotic appearance of the lips denotes impaired circulation, so that the blood is imperfectly aerated. Fulness, or turgidity, with

congestion, especially venous, of the surface of the face, is most frequently found with cardiac hypertrophy and habitual ingestion of alcohol, in apoplexy, and in typhus fever. A dusky flush over the face, with increased rapidity of breathing, suggests pneumonia. A pinched, sunken, worn expression of face suggests pain or anxiety, acute peritoneal inflammation, constant suffering from chronic disease—the forerunner of approaching death, especially if there be also “a sharp nose, hollow eyes, collapsed temples, the ears cold and contracted, skin about the forehead rough, distended and dry, and colour of whole face greenish, black, livid, or lead-coloured” (*Facies hippocratica*). Retraction of the corners of the mouth to the extent of a “sardonic grin,” is suggestive of inflammation implicating the diaphragm, or of painful affections of the stomach or bowels, near the tendinous aponeurosis of that muscle.

A straw-coloured, anæmic appearance of the skin is suggestive of the ill health (cachexia) of malignant disease. A jaundiced face, with a melancholy air, is suggestive of hepatic disease. A downcast expression, elongated upper lip, and mobile features, are suggestive of hysteria. Thickened upper lip, delicate skin, and fair complexion, are suggestive of scrofula.

(3.) *Condition of the General Cutaneous Surface*, as to temperature, perspiration, eruptions, tumours. Increase of temperature is to be measured accurately by a thermometer; and, with quickened pulse, it is a sure sign of fever. Dryness of the skin is suggestive of arrested secretion generally, and of defective perspiration in particular. General coldness of the skin betokens a weakened capillary circulation—a condition common to the invasion of many acute diseases. A cold, moist skin in severe diseases is evidence of collapse; and a clammy skin generally indicates debility. Marked paleness of the skin is suggestive of a poor and watery blood, or of an internal excessive hæmorrhage. Eruptions, other than those of special skin diseases, suggest the existence of some exanthematous or contagious fever. Petechiæ, or extravasations of blood, suggest the existence of a scorbutic or purpuric condition, or greatly altered blood, by decomposition during life, as in some forms of putrid or malignant fever, with so-called typhoid symptoms. To determine the condition of the skin, the examination should first include the face and neck, next the shoulders and back, chest and abdomen, and, lastly, the extremities, hands and feet. If the skin is moist, the smell and reaction of perspiration (acid or alkaline) should be noted.

(4.) *Condition of the Limbs and Joints*.—Apart from paralysis, loss of power in the limbs is suggestive of *typhus fever*, or of *diabetes*, or some exanthematous fever; the prostration of muscular power being excessive in all kinds of fevers, and always attended with flaccidity of the muscles, indicating impaired rigidity and tone of muscular fibre, and loss of irritability. *Continued tremors*, or constant shaking of a limb, or frequent twitching of a muscular part, are suggestive of irritation at the origin of the nerves supplying the limb or part. *Cramps or spasms* of the limbs suggest irritation of the bowels, the existence of acidity or flatulence from impaired digestion, or of diabetes, or of gout, when they affect the lower limbs.

When they implicate more than the lower limbs, they are usually of serious import—as in *cholera*, *phrenitis*, or *apoplexy*, or in *fevers*, or the *puerperal* state and convulsive movements in *Bright's disease*. Tumid conditions of the joints, and relaxation of their ligaments, singly or in conjunction, suggest predisposition to scrofulous affections. Symptoms of the joints being affected are suggestive of *rheumatism* or *gout*; or of the digestive or assimilative functions being impaired, particularly when the functions of the liver, bowels, or kidneys betray disorder. Affections referred to the joints are also sometimes suggestive of *hysteria*.

Having thus taken a general survey of the patient and of his history, which cannot fail, in cross-examination, to point to prominent symptoms of disease or illness—if any disease or ailment exists—it also must furnish some clue to the region, organ, or system likely to be most affected, so that the PRESENT CONDITION, as determined by (B.) REGIONAL EXAMINATION OF SYSTEMS, will complete the inquiry.

This examination ought to commence with the region presumedly the most diseased; but whatever region is commenced with, it is necessary that the investigation and recording as to the condition of all the systems of one region be completed before going farther. “One step securely ascertained leads to another; and from what we see upon the exterior we obtain a clue for directing our inquiry to the seat and centre of the disease within. If locomotion be hindered, we look well to the brain and spinal marrow; if there be the livid lip and dusky skin, we scrutinise particularly the condition of the heart and lungs; if the whole body or some of its parts be attenuated, we examine well the organs of nutrition.” (LATHAM, *On Subjects connected with Clinical Medicine*, Lect. II., p. 48; a work which ought to be carefully studied by every student. It has been reprinted by the New Sydenham Society, having been long scarce and out of print.)

(B.) REGIONAL EXAMINATION OF SYSTEMS.

HEAD.—*Nervous System.*

(a.) *Peripheral Indications* embrace conditions as to motion, sensation, special senses—taste, smell, vision, state of pupil and influence of light on it, examination of eye by ophthalmoscope, movements of tongue, as suggestive of the state of the brain and organic nervous system. Impairment or alteration of *vision* or functions of the eye and eyeball are suggestive of cerebral mischief. The *sense of smell* is influenced, not only by the brain, but by the state of the respiratory passages as well. *Taste* is very closely connected with the state of the digestive functions, as well as with the brain. The impression that ought to be made through any one of the special senses may fail,—(1.) From the special organ not being itself in a normal condition to receive the impression; (2.) from the nerves of special sensation not being able to transmit the impression to the brain (atrophy, injury, wounds, tumours, morbid deposits pressing on them); (3.) although the impression may be received and transmitted, the brain itself may be diseased at the parts connected with the origin of these nerves of

special sense. The fact of any function of special sense being suspended, increased, diminished, impaired, or perverted, suggests the existence of cerebral disease, or of fever, or disorder of digestive functions.

(b.) *Central Indications* refer to the brain and spinal cord, as to feelings of exhaustion or the reverse; intelligence, dulness, or stupidity; lethargy, or sleepiness; wakefulness and activity; headache, vertigo, stupor, coma, nausea, pains in the back; general sensibility increased, diminished, absent, or impaired (using the anæsthesiometer); spinal irritation to be determined by percussion along the spinous processes of the vertebral column; and by the application of a hot sponge along that region; pain on movement, and the existence of special spinal symptoms. (See preliminary chapter to "Diseases of the Brain," Vol. II.)

THORAX.—(a.) *Respiratory System.*

1st. *Symptoms.*—Note the number of respirations per minute (16 to 18 per minute being the normal number in the adult, or one for every four beats of the pulse); if easy, calm, and full drawn, or difficult, painful, or laboured, short, forced, hurried, and incomplete; whether the breathing is attended with pain, or is checked or arrested at a certain limit of expansion by cough, with or without pain; whether respiration goes on through mouth, nose, or both; whether the movements are mostly expressed by the diaphragm and abdominal muscles (abdominal respiration); whether the alæ of the nose dilate and contract at each respiratory effort, with forcible and constrained movements; cough, sneezing, expectoration, quantity and character of the discharge—thin, inspissated, frothy, mucous, purulent, or both; rusty, bloody—(microscopic examination), odour of the breath, hæmoptysis.

2nd. *Physical Examination*, by inspection (external form of the chest and its symmetry), degree of prominence of clavicles—post and infra clavicular spaces—post sternal space—any general or partial bulging or retractions—width of intercostal spaces. Note pulsation in the neck, venous or arterial, under first bone of sternum—palpation, percussion, and auscultation of organs. (For an account of the regions of the chest and their contents, see preliminary chapters to investigation of "Diseases of the Thorax.")

(a.) *Peripheral Indications.*—Nose, action of *alæ nasi*; larynx, voice (use of laryngoscope), trachea. If speech is affected, the condition of the epiglottis, tonsils, and pharynx must be observed by means of an examination with the aid of a spatula, designed for that purpose, to depress the tongue.

(b.) *Central Indications.*—Lungs and pleuræ are to be carefully examined in every part by percussion and the stethoscope, noting :—(a.) Expansion, as ascertained by eye and hand—intercostal spaces marked or not—in full inspiration—full expiration—relative duration of inspiration and expiration. (b.) Expansion as determined by chest-measurer. (c.) Measurements of chest—circumference immediately below axilla, right and left—at complete expiration—at full inspiration. At point of ensiform cartilage—at complete expiration and at full inspiration. Sterno-clavicular articu-

lations to nipples—middle line to nipples. (*d.*) Vocal fremitus—infra-clavicular—mammary and lateral regions—friction or rauchal fremitus—passing tremors or thrills of any kind. (*e.*) Percussion—post clavicular—clavicular—infra-clavicular—*first on one then on the other side*—post sternal—sternal throughout—mammary—infra mammary—axillary—infra axillary. (*f.*) Respiration—in same regions as above, distinguishing between inspiration and expiration in easy, deep, and tussic breathing. (*g.*) The voice in the same parts.

Examination of the back (patient sitting up with the arms crossed)—General inspection:—Supra spinous fossæ—movements of ribs determined by eye and chest measurer—general or partial bulging—dorsal arch—curvature of spine—tenderness of spine.

Vocal fremitus. Percussion in supra spine fossæ—infra spinous—interseapular—angles of scapulæ—bases of lungs. Mark out the lower edges of lungs. Auscultation for respiration and voice is to be made in the same regions.

Examine loins, and mark out kidneys by percussion—grasp the kidneys so as to determine any pain on pressure?

(*b.*) *Circulatory System.*

1st. *Symptoms*, such as cardiac dyspnoea, palpitation, uneasiness or pain at precordia, syncope, angina pectoris, are to be carefully noted.

2nd. *Physical Examination* of the heart and blood-vessels by inspection, palpation, percussion, and auscultation. (See preliminary chapter to investigation of “Diseases of Heart and Circulatory System.”)

(*a.*) *Peripheral Indications* from arteries, especially radial and carotid; from pulse, as to its number, character, variations, and use of sphygmograph; also from veins, of neck especially.

(*b.*) *Central Indications.*—Heart and large vessels within the thorax, noting—(*a.*) Position of heart's apex, impulse whether perceptible, strong or weak, whether pulsation elsewhere, any passing tremor or undulation; (*b.*) percussion note over first, second, third, fourth, and fifth left cartilages and the first four interspaces as compared with the right side, percussion from inner or right line of cardiac dulness, then for the outer or left line; shape of dulness; (*c.*) sounds over second right cartilage, over second left cartilage, mid-sternum, apex, point of ensiform cartilage, outside left nipple, top of sternum, in the neck. Is there any friction?—the first and second sounds must be listened to separately. Then comes the question as to the existence and probable nature of intra-thoracic tumors (aneurisms, enlarged glands) or other growths. The two sounds of thoracic aneurism—as distinguished from the single sound of abdominal aneurism. Condition of lymphatics and lymphatic glands are also to be noted.

To feel the beat of the pulse at the wrist must never be neglected. It is popularly believed to be indispensable to the understanding of a case. The action of the heart is to be judged of by the pulse, and also the state of the artery as to pressure or expansion by the blood. In a healthy adult, the pulse beats at the rate of 65 to 75 times in the minute, and indicates a like number of contractions of the left ventricle. It becomes slower with

advancing years (60 to 65 in old age). In infancy, the rate is from 110 to 120; and in a child three years of age the rate of pulsation is from 90 to 95 or 100. Warmth and heat quicken the pulse; so does rapid breathing and exercise, or mere exertion, forced expiration, and the process of active digestion. In the recumbent position, and during sleep, the pulse is slower. Increased frequency suggests some cause exciting the action of the heart—such as exercise, restlessness, mental emotion, fevers, acute inflammation, or alcoholic fluids. The pulse has also more frequency and less force in fever than in inflammation, when it has greater force and less frequency. Besides its frequency per minute, its rhythm, its volume and strength, and its resistance must be noted. A slow pulse is suggestive of shock, depression, pressure on the brain; and is common also in cases of jaundice. The rhythm of the pulse is perverted when the beats do not follow each other in regular succession, but occur at unequal intervals, and when one or two beats intermit. Such irregularity in rhythm suggests digestive impairment, or mere debility; but may also frequently indicate cerebral or cardiac mischief. The volume of the pulse is measured by its fulness or distension with blood—the vessel being more or less completely and fully expanded with each beat of the heart. A hard or wiry pulse is one which combines smallness with force. A soft pulse is one which combines largeness with want of force. A full and strong pulse suggests plethora. It is the pulse of youth and robust health, as well as of inflammatory disease—the early stage of many fevers, and of general capillary obstruction. The sphygmograph attempts to obtain and indicate to sight that kind of information regarding the pulse which the physician acquires by “*tactus eruditus*,” and the student is referred to the preliminary section introductory to “Diseases of the Heart and Circulatory System,” in the second volume of this work, for details as to its use.

When symptoms connected with the heart are present, with acute rheumatism, rheumatic fever, or arthritis, they are suggestive of some one of the following affections:—Acute pericarditis, dry or with serous, seropurulent, or hæmorrhagic effusion; acute endocarditis, singly or combined with pericardial inflammation; acute myo-carditis, singly or combined with peri- or endo-cardial inflammation, or both; acute aortitis, singly or combined with the previously named lesions. In continued fever, like typhus, it suggests softening of the muscular texture of the heart itself.

3rd. A microscopic examination of the blood should never be omitted.

ABDOMEN.—(a.) *Digestive System.*

(a.) *Peripheral Indications.*—Examine as to symptoms and physical signs from alimentary passages, from the entrance by the mouth to the outlet by the anus. Symptoms are furnished by the teeth and gums, tongue and throat, deglutition (examination of throat with spatula), appetite (or the reverse) for food and drink, stomachal and bowel digestion, vomiting, character of matters vomited, flatulence, stomachal and intestinal eructations, regularity of bowel evacuations, number and nature of the stools; accompanied or not with pain or uncomfortable sensations during defecation. Perfect and enduring teeth suggest soundness of con-

stitution. Their early decay suggests impaired constitutional power, or prolonged disorder of digestive functions. *Grinding* of the teeth during sleep has a varied significance, according as the patient is a child or adult, or is aged. In children it is suggestive of intestinal worms or of cerebral disease, in which latter state it is generally associated with a bright eye, a contracted pupil, and a flushed cheek, startings in sleep, to be followed, perhaps, by convulsions and meningitis. In adults and aged it is suggestive of an apoplectic or paralytic attack. It is an unfavourable symptom during contagious fevers, as suggestive of cerebral or urinary complications. Accumulations of tartar around the teeth at the margins of the gums suggest a disposition to calculous, gravelly, or gouty affections. Inordinate sensitiveness of the teeth suggests nervous diseases, acidity of the stomach, and impaired digestion. The teeth become loose in scurvy, purpura, and during mercurial salivation; and the gums are often retracted from them, so that they appear elongated. This is common during chronic gastritis and chronic impairment of digestion. Note as to the cleanness of the teeth, or crusting over with blood or mucus (*sordes*).

Paleness of *the gums and lips* suggests anæmia, chlorosis, and excessive loss of blood. A blue margin surrounding the teeth suggests lead contamination; a red spongy swollen appearance suggests mercurial poisoning, chronic disorders of digestion, diabetes, incipient scurvy, or purpura; and if these morbid states are prolonged, the condition of the gums becomes worse, so that they bleed, or become ulcerated and even gangrenous. Note their condition as to redness and moisture, lividity and congestion, herpes or other eruptions, creamy or black exudations and crusts (*sordes*).

The state of the tongue varies—(1.) With the states of the several digestive functions; (2.) with the nature of the prevailing disease; (3.) with the existence and amount of general constitutional disturbance, as respects especially the organic nervous system, vascular action, and sanguineous contamination or blood-poisoning. In examining the tongue it must always be remembered that it may be coloured by food, or drink, or by medicines; and, that it may be abnormally dry by constant breathing through the mouth, but less so in health than in disease. The condition of the tongue, therefore, is taken to indicate the condition of the digestive canal, as to activity of function generally, the condition of the nervous system, the condition of the blood and state of the secretions.

Its movements, its volume, its colour, and its coating must be examined and recorded.

Impeded and tremulous movements of the tongue suggest exhaustion. It is protruded slowly and with difficulty in fever of a typhoid type, and in nervous disorders with marked debility, when it sometimes remains protruded, and in comatose, apoplectic, and paralytic conditions. Its movements are impaired in paralysis, and it turns to *one* of the corners of the mouth in hemiplegia, and when this is combined with imperfect articulation a serious deep-seated cerebral lesion is indicated. A swollen, broad, and flabby tongue, as if it were too large for the mouth, and on the edges of which the teeth leave their impression, as in œdema, is suggestive of chronic impairment of digestion, generally from chronic visceral disease, or of the action of mercury or of some poisons. It is also seen in affections of the brain, or as a consequence of disturbed circulation

attending disease of the heart, typhus fever, scarlet fever, small-pox, syphilis, and mercurial poisoning. Swelling of the tongue suggests danger in cerebral affections, exanthematic and continued fevers. Dryness denotes deficient secretion. It is seen in acute visceral inflammation, and is constant in continued fevers and the exanthemata. It is so dry in enteric fever sometimes as to cause the papillæ to appear prominent and the surface rough. A fissured or lobulated condition of the tongue, combined with enlargement, suggests chronic impairment of the digestive functions, with hepatic disease. A tumid and livid tongue, the surface being covered with a yellowish coating of milky or cream-like mucus, suggests disease of the lungs or heart, the urinary or uterine organs. A very moist and soft tongue, generally bearing evidence of indentation, betokens debility, and suggests tonic and not lowering remedies. When humidity appears after a dry tongue it is a favourable sign, the returning humidity appearing first at the edges of the tongue, whence it gradually extends over the surface to the middle line. The dryness of the tongue is generally accompanied with roughness, from the parched-up condition of the papillæ or fur, or it has a burnt appearance and is fissured, and sometimes it has a dark or deep red hue. The most extreme of this dry condition, when thirst ceases to prevail, is a most unfavourable sign, as in the dry brown crusted tongue—the mahogany-like tongue of *typhus* fever. Paleness of the tongue has a similar significance to paleness of the lips and gums—*i.e.*, suggestive of anæmia, chlorosis, loss of blood, or poorness of blood from excessive discharges or spleen-disease. A very red tongue occurs in inflammations of the throat and pharynx, and in the exanthemata. Redness limited to the point and edges suggests acute irritation or inflammation of the digestive mucous surface in continued and remittent fevers, the surface and base of the tongue being at the same time loaded, coated, or furred. After having been thus coated over, if it becomes very red and clean, glossy and varnished looking, and swollen at the tip, especially if fever does not abate, the condition is not favourable. A white creamy tongue suggests catarrhal febrile states, with functional disturbance of the digestive and respiratory organs—the premonitory stages of fevers and inflammations. A clammy sticky state of the tongue, with redness at tip and edges, and with a white, yellowish-white, or milky-like fur on the surface and base, with more or less humidity, suggests visceral disease, inflammations of mucous surfaces, the early stages of fevers. Regular, prominent, bright red points uniformly appearing above the white and creamy surface (strawberry-like tongue) suggests the fever of *scarlatina*, *measles*, or *small-pox*. A loaded condition of the tongue in the morning suggests impairment of the stomachal or bowel digestion. Late suppers, smoking, and drinking malt or alcoholic liquors before going to bed will induce this condition. A furred tongue resembles the pile on the surface of cotton velvet, from the erectness and development of filiform and fungiform papillæ. It varies in colour and thickness, and is generally covered by a clammy, dirty mucus—the fur and mucus together forming a coat on the base and dorsum of the tongue—the colour varying from a greyish white or yellow to a brown or even black. Such a state of the tongue suggests parenchymatous inflammation of solid organs, severe irritation of mucous membranes, an adynamic condition in continued or exanthematous fever.

The throat and fauces, especially the *uvulæ* and *tonsils*, ought to be examined (with the aid of a spatula to depress the tongue)—as to vascularity, relaxation, aphthous spots or ulcerations. Relaxation of the uvula, velum, or fauces, suggests general debility, with disordered stomach or catarrh. The tonsils are often enlarged from chronic congestion, or as an expression of general chronic derangement of health, or as a sign of a scrofulous constitution. They are generally enlarged, sometimes ulcerated or aphthous in cases of exanthematous and continued fevers. Pressure ought to be applied upon them to determine if secretion (mucous or purulent) is pent up in their crypts. With regard to *appetite for drink and food*, as suggestive symptoms, it is to be observed that the *desire for drink* is diminished in the advanced stages of fevers and inflammations; and, in such cases, when the tongue and mouth are dry, and fever does not subside, the absence of thirst, or of a desire for fluids or drinks of any kind, is of unfavourable import. Increased desire for drink is present during the earlier stages of all febrile maladies, also in diabetes and in cholera. A diminution or entire loss of appetite (*anorexia*) for food is common at the commencement of most acute diseases, especially at the commencement of early stages of fever.

Nausea and vomiting suggest the digestive system, or the brain, as the source of the vomiting. It is suggestive of pregnancy, or hysteria, in the female; and, in either sex, of some irritation or tumors in or amongst abdominal or pelvic viscera. It is also present on the invasion or at an early stage of continued fevers and the exanthemata; also in epilepsy and diseases of the brain.

Eruclations, heartburn, gastralgia, retchings and vomiting, rumination, flatulence (gastric or intestinal), pyrosis or water-brash, are all and each symptoms suggestive of impaired digestion. The period after taking food at which these symptoms come on is of importance to be noted; also the nature of the matters vomited, as to acidity, presence of blood, or *sarcina ventriculi*.

The *intestinal evacuations*, as to their regularity, quantity, and appearance, should receive the most attentive examination; also, as to their frequency or abnormal retention. Retained or delayed evacuation is often a symptom attending—(1.) Severe disease of the digestive canal, or of the liver, spleen, kidneys, or sexual organs; (2.) Mechanical obstruction to the passage, or in it; (3.) Cerebral disease, or disease of spinal cord or membranes, and different forms of insanity. Frequency of intestinal evacuations varies with age and habits. Infants evacuate the bowels twice or thrice a-day; adults, as a rule, once only, and sometimes twice; elderly persons less frequently; and sedentary persons, especially females, who eat little, only once in two, three, or even more days.

Frequency of evacuations, or purging, suggests irritation and hyperæmia of the digestive villous surface—*intestinal catarrh*—which, when continued, becomes *diarrhea*. Its presence in children may be associated with teething; and in adults suggests the taking of too much or improper food, or excesses in the use of alcoholic drinks; the irritation of fecal accumulations; an excessive flow of bile into the bowels; a complication of continued or exanthematous fevers; or lardaceous disease of the intestines. Note the presence of localised symptoms, such as pains, tormina, gurgling noises (*borborygmi*), especially as localised in the right iliac fossa. The fæces are altered by disease in form, consistence, colour, odour, and

quantity; in the nature of their constituents, and in the substances which may pass with them, or which they may contain.

(b.) *Walls and solid viscera of abdomen.*—Here the shape of the belly and measurement of its size are to be recorded, circumference round the navel (also length of trunk from upper edge of sternum to pubis). Pain, tenderness, general tumidity, hardness, or resistance of walls—tympanitic or flatulent; presence of swellings or tumors; distension or collapse; condition of peritoneum as to presence of fluid (*ascites*); of the ovaries; of the liver—its size and limits—noting position of upper and lower edge in full expiration and inspiration—height of liver. Can the edge of the liver be felt? Is it rough, smooth, round, or sharp? Then of the spleen—its size and limits by percussion, and auscultation for splenic murmurs (microscopic examination of the blood); the condition of the pancreas and lymphatic glands are to be inquired into. Abdominal tumors require most careful examination; but, from the predominance of aneurism and malignant disease, the question is generally between these two; and between fibrous tumors, cysts, fæcal accumulations, extra uterine foetation, abscesses (psoas, pericecal, perinephritic, pelvic), and enlargements of glands. There must be careful percussion of the abdomen, marking out any dull parts, and a special examination for tumors and fluctuation. No opinion should ever be given without examination; more mistakes arise “from not examining than from not knowing” (JENNER). (See introductory chapter on “Diagnosis of Abdominal Diseases,” Vol. II.)

(b.) *Genito-Urinary System.*

Micturition is to be noted as to its frequency, painfulness, or difficulty. The urine is to be examined in *every case*, and the following points noted:—1. The *total quantity* passed in the twenty-four hours must be collected, and its amount measured—the normal amount being 30, 40, or 50 ounces. 2. The general appearance and colour; clearness or turbidity; presence or absence of deposits, and of extraneous impurities. 3. Odour. 4. Re-action. 5. Specific gravity. 6. Presence or absence of albumen, and, if present, an approximate estimate of its quantity. 7. Presence or absence of sugar; if present, an estimate of its quantity. If there be a deposit, note—8. Its aggregation and colour; whether amorphous or crystalline, light or heavy; the manner of subsidence or precipitation. 9. Its solubility or insolubility by heat; solubility in nitric acid, in acetic acid, in liquor potassæ; insolubility in both acids and alkalies. 10. By the microscope—absence or presence of crystals, their appearance and form; of epithelial cells—renal or extra-renal; of blood discs; of pus globules; spermatozoa; tube casts; confervoid vegetations. Difficult micturition is termed *dysuria*; when it amounts to extreme difficulty, it is known as *stranguria*; while *ischuria* means the suppression or retention of urine. All these symptoms are suggestive—(1.) Of diseases of urinary

* All the necessary apparatus for such urine examination, arranged in a suitable stand by Mr. Payne for Dr. William Roberts, of Manchester, may be got of the firm of Mottershead & Co., Market Place, Manchester. I have found this a most convenient set of apparatus.

organs and passages; (2.) Abnormal conditions of the urine itself, or the presence of calculus or gravel in the bladder or passages; (3.) Diseases of adjoining, or even distant viscera, as in cases of dysentery, liver, spleen, uterine or ovarian lesions or tumors in the pelvis or abdominal cavity. The most careful examination is often required to ascertain the nature and source of the lesion giving rise to these symptoms. *Nephritic colic*, or paroxysms of pain in the loins, shooting along the ureters to the testicles and inside the thighs, with faintness, nausea, or vomiting, suggests concretions in the kidney, when the urine is voided with undue frequency, often with pain at the end of the penis, mixed with blood.

Suppression of urine suggests—(1.) Inflammation, hyperæmia, or structural alteration of the kidneys; (2.) Lesions in the course of continued or exanthematous fevers and cholera; (3.) Organic lesions of the brain, spinal marrow, or membranes. It is a most dangerous symptom—to be followed in a short time (if the secretion of urine does not return) by such alterations in the blood as denote extreme excrementitious plethora, effusion of fluid into shut cavities and areolar tissue, coma, and death. *Enuresis*, or inability to retain the urine, suggests over-distension of a paralysed bladder. Frequent micturition suggests inflammatory diseases of the urinary organs, and especially the bladder; also Bright's disease, or diabetes, and also mental excitement and hysteria. An abundant flow of pale, limpid urine negatives the idea of acute or febrile disease. (See preliminary section, Vol. II., on "Diagnosis to Kidney Diseases and Examination of Urine.")

Examination of the kidneys and bladder is to be made by percussion, to determine enlargement of the former and distension of the latter. Increased area of dulness on percussion, and fulness in the region between lower ribs and margin of ileum, especially when the parts are pressed forward anteriorly, indicate tumor. A dull sound on percussion immediately over the pubis indicates fulness of the bladder, and, combined with pain, over-distension. Pain behind or below the pubis suggests inflammation or ulceration of the bladder or its cervix, or the existence of stone. The *prostate* requires to be examined *per rectum*.

In the female, diseases must invariably be studied by the light of *menstruation*. Its monthly regularity must be inquired into; also, as to the usual amount of the discharge and its quality, and if there be much pain during the flow. If pain has been long continued, and if there is at the same time an increased flow from the vagina, with a purulent, white, or bloody character of the discharge, it will be necessary to make an examination by the finger as to the condition of the *os* and *cervix uteri*, and, if necessary, with the speculum. The condition of the uterus, ovaries, and vagina must be determined, and generally an accurate internal examination is necessary for diagnosis. It is an examination, however, which ought not to be had recourse to in the case of unmarried women, unless they cannot be cured without such an examination.

The question of pregnancy, and of uterine, pelvic, or ovarian tumors and diseases, require a very careful and painstaking investigation for efficient diagnosis. Pain in the back, difficulties in walking, or in defecation, are to be inquired into; and, lastly, the functions, condition, and appearance of the mammary glands.

In cases in which it may not be possible to go fully into the examination, an inquiry into the special previous history should be followed by an examination into the part to which the symptoms point as the seat of the disease; then in addition to the usual subjective and objective symptoms, the following points should be more or less closely examined :—

1. Apices of the lungs in front and behind.
2. Sounds of the heart.
3. Condition of abdomen.
4. State of urine and fæces.

III.—Diagnosis.

From the whole evidence and history of the patient, thus carefully committed to writing, and *all* the circumstances being fully known, the physician makes up his mind as to the nature of the case before him. He must convert the symptoms into signs of disease, or explain them as consistent with health. In other words, he must make a *diagnosis*—i.e., he must know the disease throughout, and discriminate it from all others. His primary object is to aim at a *correct* diagnosis. This is absolutely necessary, in order that a rational mode of treatment may be devised, and a satisfactory prognosis arrived at, as to the probable result of the disease—in recovery, in permanent organic mischief to some organ, in chronic ill-health, or in death.

By such a careful examination of the patient as has been described, which elicits the facts as to his condition, the physician can at once determine—

(1.) Whether or not disease or ailment exists at all; whether the patient is, consciously or unconsciously, deceiving himself, or trying to deceive others; whether, in fact, he is a “malingerer,” or is the subject of delusions, hysteria, hypochondriasis. “*Malingering*,” as a type of deception, is markedly disappearing from communities where it has hitherto been supposed to prevail (the army, navy, poorhouses, and gaols), since the greater and extended use of delicate instruments, and methods of explaining and determining the existence of symptoms with greater precision.

(2.) From the examination conducted as described, the symptoms of any ailment the patient may have are sure to be discovered, and the next step is to appreciate the significance of the symptoms, and their relations to each other. Are the symptoms the signs of acute disease; of general disease affecting the whole system; or of disease localised in one or more special organs; or is the case one of chronic disease? The preliminary survey of the case ought to determine these questions almost at a glance; and when the examination has been fully and carefully made, the kind of acute disease may be indicated also. Thus, increase of temperature, a quickened pulse, and respiration are certain indications of fever—either of specific disease or inflammatory; if combined with pains in the chest, cough and expectoration exist, with the signs also of pyrexia, acute thoracic disease is at once diagnosed; a *decubitus* on the back (supine), with symptoms of nervous prostration, muscular debility, and flaccid outstretched limbs, with dusky-brown tint of the skin, a stupid, lethargic, dull condition as to intelligence, indicates an acute specific fever

—typhus—if its characteristic eruption is present. A warm, soft, and moist skin, with an acid reaction and sour smell, and a temperature above 100° Fahr., denote an acute rheumatic fever. Complexity of lesion and functional impairment are generally associated with chronic disease; and in such cases it is necessary to determine how far the patient is removed from the state of health usual to him, or how much more impaired his health has become.

(3.) The diagnosis is also to be made as between structural or organic disease and impairment of function, as well as the nature of the structural change and extent of the damage to the organ and its functions. In every individual case (either acute or chronic) it is necessary to consider the respective value of each morbid sign in connection with one another. The previous history of the patient, and the whole series of symptoms as to the duration, cause, and mode of invasion of the existing illness, must be carefully analysed; and even indirect manifestations of disease furnish valuable circumstantial evidence when subjected to careful reasoning and reflection. A summary of the objective symptoms of functional and structural lesions should be stated in the diagnosis of every case, arranged in the order of probable succession and importance—the main facts, which are obviously the most direct signs of disease, being first laid hold of. Each part of the testimony as to the progress and duration of symptoms—the termination of the case, and the results of treatment—should have its proper position assigned to it, as elements in determining the diagnosis; and to do this, *all* the facts must be carefully sifted and weighed, and not indiscriminately collected or heaped together. It is in the endeavour to effect such a summary that a minute acquaintance with anatomy, physiology, pathology, and the natural history or biography of disease is of so much value to the student; and if to such knowledge he also possesses the powers of a good reasoner, with a varied extent of collateral knowledge and acquirements, these will greatly contribute towards that completeness and accuracy of diagnosis which distinguish the accomplished physician who thoroughly knows and practises his art. It is by such processes of analysis that a *direct diagnosis* is made, and this with greater or less difficulty in different cases. Taken individually, very many symptoms are common to many diseases; but when several symptoms constantly occur, singly, or in groups together, or in uniform succession, they point to definite diseases, and become certain signs of such diseases. These symptoms, which are peculiar and distinctive, ought to be well known, so as to be appreciated with rapidity. They are the “*pathognomonic symptoms*” of disease—so called because of the certainty with which they indicate the particular disease, so as to clench the diagnosis—*e.g.*, the appearance of the peculiar eruptions in small-pox, scarlet fever, typhus, and enteric fevers respectively, superadded to the febrile phenomena common to them all, but with characteristic differences in each.

A *differential diagnosis* is made when cognisance is taken of the essential signs by which one disease can be discriminated or differentiated from another which it resembles; as between *enteric* and *typhus fever*, between *gout* and *rheumatism*. The co-existence of several diseases in the same person is not to be overlooked in diagnosis, inasmuch as the direct symptoms of each separate lesion may be modified or neutralised by one another.

Time for the development of symptoms is an important element in diagnosis. The early manifestations of many disorders are so similar (as in fevers), that it is impossible to tell with which of several maladies we may have to deal, till after a few days some other element develops itself, which solves the doubt. *Varieties* of disease require to be identified and considered in differential diagnosis. A complete diagnosis does not limit itself to the determination of such primary ailments as may have led the patient to seek for medical advice. The state of all his organs and functions must be so completely determined as to enable the physician to say whether or not any organic disease exists in any part or function of the body, and so make an *entire* and complete diagnosis of the patient's case.

A diagnosis may be arrived at by the process of *exclusion*—that is, by finding out what the disease is not; but if it is necessary to use this method of diagnosis, it shows that the case must be unusually difficult and obscure.

A speedy and rapid diagnosis is often demanded, as at the bedside in private practice. In such circumstances the main facts of the case are first elicited, especially those which are the most direct signs of disease. The mind, working almost unconsciously, decides as to what part or organ is thus indicated as the seat of the malady; and this having been carefully examined, with knowledge and experience, a direct and differential diagnosis is thus almost intuitively arrived at. If no signs of local disease exist—only those of general or constitutional disturbance, as in fevers—clinical experience and knowledge of such diseases are then required to decide the nature of the case. It is the co-existence of many lesions which makes diagnosis difficult, and the pathological relations subsisting amongst them, as is the case with the numerous local lesions and intercurrent morbid phenomena arising during a case of Bright's disease, or specific fevers; and diagnosis is also especially difficult in cases of acute disease supervening in chronic affections. The absence of any particular symptom which usually exists in a particular disease ought to be specially noted, to show that its omission is not due to negligence or forgetfulness on the part of the observer.

IV.—Prognosis.

A correct and complete diagnosis implies also the forecasting of events, and the formation of a just estimate of the probable result of the illness or disease. To do this requires experience and knowledge of the natural history of diseases, combined with caution and tact.

When the physician has to make or to give a *prognosis*, he has to make up his mind upon the following points:—(1.) As to whether the illness will terminate in the death or recovery of the patient? (2.) If the patient lives, whether recovery will be complete, or whether permanent organic mischief of greater or less extent will remain? (3.) Whether persistent impairment of the general health will result (*cachexia*)? (4.) What will be the probable duration of illness? (5.) If death is the probable result, how will it likely come about? Is it likely to be slow or sudden; and attended with pain or not—with convulsions or not, or

with consciousness or unconsciousness? (6.) Whether certain symptoms, apparently trivial in themselves, are or are not indications of danger, or of serious disease, such as slight convulsive movements, numbness—“*pins and needles*”—suggestive of cerebral disease? (7.) Whether any complications may come on likely to increase the danger, or whether sequelæ of the disease may remain?

Patients or their friends are ever ready to put any one or all of such questions to the physician. With certain knowledge and experience, positive opinions may be expressed without hesitation. If doubt exists, let the patient and his friends have the benefit of the doubt, by explaining to them the exact nature of the disease and of the doubt. The stimulus of hope is of great value in favouring the return to health; and with all the growing resources of our art, prognosis should be as cheerful and favourable as possible. An unduly or prematurely hopeless prognosis is a violation of the first duty of the physician. (*Lancet*, March 13, 1869.) If the prognosis is generally unfavourable, it is better to communicate this in the first instance to one judicious friend solely, rather than to the patient; and the dangers of the case ought always to be clearly explained. If an opinion is asked by the sufferer himself in cases where there is *no* hope, it becomes our duty to tell him so.

The elements on which a prognosis must be based are as follow:—A complete and intimate acquaintance with the natural history of each disease, especially as to—(a.) its known rate of mortality. For example, the prognosis in *relapsing fever* is more favourable than in *typhus fever*, because the rate of mortality in the former is only 2 or 3 per cent., whereas in typhus fever it is as high as 20 per cent. (b.) The special circumstances known to influence the rate of mortality—namely, age, constitution, habits of the patient, and stage of the disease. For example, in typhus fever the mortality in persons between 10 and 15 years of age is not much over 2 per cent., but in persons over 50 it exceeds 50 per cent. Accordingly, the prognosis even at the commencement of an attack of typhus would be at least twenty times more unfavourable in the latter period of life than in the former. Again, the natural duration of uncomplicated typhus being fourteen days, and the symptoms and other conditions being alike in two cases on the tenth and fourteenth days respectively, the prognosis would be much more favourable in the case where the disease had reached its natural limit, than in that in which there were four days still to run. (c.) The appearance of certain symptoms of hyperpyrexia or complications, which add to the severity or danger of the original disease. In many diseases there are certain symptoms which, from their severity or very occurrence, are of grave prognostic import. Of this nature are convulsions in typhus, hæmorrhages in small-pox, and a gangrenous odour of the breath in pneumonia. As a rule, complications add to the severity and danger of the primary disease.

Much knowledge has still to be acquired on the subject of Prognosis. As applied to determine the eligibility of lives for insurance, the question of prognosis is a very important one, especially as affecting the calculations of the individual chances of longevity. For life insurance purposes a due consideration requires to be given to every circumstance of family

and personal medical history; and such a careful examination of every organ and system, as that which has been described, is necessary to furnish data on which to decide the eligibility of individuals for life assurance, combined with an extensive knowledge of the natural history of diseases. Considering the great importance of the subject, it is remarkable how little special attention has been devoted to it. While there are numberless works on pathology, diagnosis, and therapeutics, no special work on the general subject has appeared since the "Prognostics" of Hippocrates. At present, skill in prognosis is confined to a few members of our profession, who have been close observers of nature and had great experience, but this knowledge dies with the individual. A rigid analysis of ascertained facts, with the view of arriving at something like definite conclusions as to prognosis in different diseases, is still a desideratum in medical literature. (MURCHISON, *Brit. Med. Journ.*, Jan. 20, 1872.)

V.—Treatment.

The principles according to which the management of a case is to be conducted, and the particular indications for Treatment, must be given in detail, embracing also special directions as to diet and regimen. Copies of the prescriptions ordered are to be recorded. The treatment of a case implies the prevention or further extension of disease (prophylaxis), as well as the alleviation or cure of the existing ailment. The food, drink, and administration of alcohol; the temperature of the sick-room and its ventilation; the dryness or moisture of its atmosphere; the use of baths, hot or cold; the use of electric or galvanic appliances; the nursing and general management of the patient,—all require to be prescribed for in detail, and personally attended to by the physician, with the same care as the medicinal agents of the pharmacopœia; and each detail of management ought to be in accordance with the best and most approved theories respecting diseases and their remedies, as well as with physiological and pathological knowledge. In prescribing remedies, the individuality of the patient must be carefully considered, not less than the nature of the disease. The points essential to the individual, which must influence the prescription of remedies, are—the age of the patient, the habits of life as to alcohol especially, the general strength of the patient's constitution, the stability of the nerve centres, the muscular power of the heart, the contractile resistance of the arteries, and the specific nature of the disease. Every phase in the natural course and progress of the disease must be foreseen and provided for, implying a full knowledge of its nature, seat, and stage. The known tendencies of the disease to a fatal termination ought to be guarded against, especially as regards failure of the heart's action, systemic death by decline of aortic pressure—rapid as in syncope, or gradual and prolonged as in disease of days', weeks', or months' duration; the state of the chief organs and functions of the body, especially the heart, lungs, and kidneys, must be specially investigated, with reference to medicines and the management of the case, or impairment of the nervous centres or of the lungs.

VI.—Progress of the Case.

The patient must be visited and re-examined at intervals, varying as regards time, according to the gravity of the disease, and the rapidity with which it progresses from one stage to another in its natural course. In thus noting the progress of the case from day to day, the same plan of examination in regions and systems is to be followed out as recommended for the first examination of the patient. The continuous history naturally divides itself into two parts—(1.) The accounts given by the patient of his own condition from day to day, and those given by his friends, attendant, or nurse; (2.) the phenomena actually observed at the bedside by the physician. Any changes in the patient's state, or new phenomena, are to be specially looked out for and inquired into at the time. In fever cases, daily records of temperature ought to be made as often as may be considered necessary—twice or thrice daily, at least—and all observations ought to be accurately recorded at the time, while the facts are fresh in the mind. The morbid phenomena, of which a summary is given under *diagnosis*, must be especially noted. The state of the tongue, as to depth of colour, dryness, contraction, and diminution of the fur or coating, ought to be closely observed and recorded from day to day. The pulse and number of respirations are also of daily importance. The successive daily evacuations from the bowels and the kidneys ought always to be preserved in suitable vessels by the nurse or attendants for inspection by the physician. The urine for the whole twenty-four hours is to be preserved separate from the faecal evacuations, and its amount measured and recorded daily. The effect of remedies must be especially inquired into—*after* having ascertained whether the medicine, diet, and instructions prescribed have been actually administered and followed out. Details as to the amount of food and drink taken are to be carefully noted for the information of the medical attendant.

VII.—Termination of the Case.

The date and nature of the result is to be carefully noted; having special reference to the questions summarised under the head of *Prognosis*, giving an account of the patient's condition when he ceases to require medical care. The influence the disease may have had on the state of the different functions should be indicated. If the case prove fatal, and a *post-mortem* examination is made, a record of the lesions or stamps of disease seen after death ought to be preserved—every organ in every cavity of the body being carefully examined, after the best methods of conducting investigations in morbid anatomy. When this is complete, an abridged summary of the whole case should then be made in the most concise language, stating as near as possible the phenomena in the order of sequence, with the combined light of the history of the case during life and appearances after death.

CHAPTER VII.

MORBID ANATOMY AND PATHOLOGICAL HISTOLOGY: THE SPECIAL MEANS AND INSTRUMENTS BY WHICH THE NATURE OF DISEASES MAY BE INVESTIGATED.

THE vestiges left by the prolonged existence of disease, whether in the body of man or of the lower animals, have always claimed from the physician a large share of attention. In proportion also as the knowledge of healthy anatomy and physiology has become extended, so has pathology and morbid anatomy gradually but steadily acquired an important and prominent position among those branches of study on which Medicine rests its claims as a science.

MORBID, or, as it is also sometimes called, PATHOLOGICAL ANATOMY, is that department of medical science which treats of the changes in structure appreciable by the naked eye, or by microscopic examination, or by chemical investigation, which are produced by disease in the solids and fluids of the body. Such changes of structure are known as *lesions*, which are sometimes constant, well marked, and characteristic "*stamps*," "*anatomical signs*," or evidence of certain diseases, the symptoms and fatal termination of which they at the same time explain.

MORBID or PATHOLOGICAL HISTOLOGY treats of the origin, development, growth, and decay of the new products or new formations which are the elementary constituents of structural or organic lesions. Thus the anatomy of diseased parts stands in the same relation to the development of morbid phenomena and conditions of disease that the anatomy of healthy structures and the histology of the textures do to the natural functions and processes of development, growth, and nutrition in the healthy body.

MORBID ANATOMY is a department of medical science which has gradually grown out of the accumulated experience and observation of ages; but PATHOLOGICAL HISTOLOGY, as a science, is of modern origin. It is but yet in process of development, although its foundations may be traced in the works of the earliest medical writers of antiquity. All of them refer to changes which they *merely supposed* had taken place in the internal organs; and they were doubtless led to this assumption by observing the connection that existed between structural lesions of the external parts and their accompanying symptoms. Hippocrates describes the deposit of tubercles in the lungs, the symptoms occasioned by them in a crude state, and those which attend their softening and discharge. The science of MORBID ANATOMY is a record of facts. In its relation to the progress of Medicine it is a living record—a history whose pages must be ever open to receive the observations which are constantly being made by those engaged in pathological pursuits—a record from which one may ascertain at any time the conditions under which morbid changes or new formations in the body have taken place. The pages of this history show

that at the present day the department of pathology is in a transition and ever progressing state; and the position of Medicine, as a science, must eventually result from a re-arrangement of the innumerable details which the sciences of morbid anatomy and histology may disclose and unfold. It is necessary, therefore, and often advantageous, to look back upon the past, and see what has already been done, so that its venerable facts may not be lost sight of, but may be grouped in series with the extensively verified experiments and observations of the present day. Such a retrospect will at the same time have the effect of placing in a prominent aspect the influence which the study of Morbid Anatomy has had on the Science of Medicine, the conditions under which it has flourished, and the legitimate objects of its investigations.

The art of printing had not been long invented when books on morbid anatomy began to issue from the press; and although the early period of the fifteenth century has left little enduring literature of any kind (but has been mainly distinguished by the number of colleges then founded), yet about this time pathological anatomy in the medical school of Florence shows the earliest evidence of an existence. The facilities for study which the art of printing introduced soon stirred up ardent students; and the sixteenth and seventeenth centuries produced much that will ever remain famous in the annals of medical science. Eustachius, Tulpus, Ruysch, Harvey, Malpighii, and Leuwenhoeck are names familiar as household words to the student of Medicine. The earlier attempts of this period to form a system of pathological anatomy are characterised by abortive endeavours to explain all results upon some exclusive and general principle. A spirit of speculation marks the character of the age. The men of that time had observed but few facts; and on these facts they preferred to speculate and dogmatise, rather than prosecute the further interpretation of nature, or record more observations. Accordingly, theories in abundance successively led captive the minds of the medical world, and, disappearing one after the other, demonstrated the unstable foundations on which the science of Medicine had been placed. The leader of each sect founded his so-called school or system, all of them distinguished by a due amount of arrogance and contempt for predecessors and contemporaries—a feeling unhappily not yet quite extinct. The “*vital agency*,” the “*influence of the humors*,” and of the “*solid organs*,” have each been considered by turns as the only orthodox belief; and each has had their school and sect, respectively designated as the *Vitalists*, the *Humoralists*, and the *Solidists*. The theories of Galen, of Paracelsus, and others, have all been famous in their time, but are now unheard of, and almost unknown. The same fate awaits the false theories and absurd conceits of more recent times, although, as in the case of Stahl, Cullen, Brown, and Broussais, they had a wide prevalence in the schools of Europe, and made impressions on the sentiments of the profession which yet influence their modes of practice and the reasons for their belief. Broussaisism, Hahnemannism, Mesmerism, and some other systems, “the fruits of a luxuriant fancy and of few facts,” must all descend, as others have done, the same inevitable slope to oblivion; but the vast collection of facts which the founders and followers of such systems eventually accumulate and bring to notice, remain unchangeable, and will continue to recur in

the daily experience of our profession, just as they appeared to the venerable fathers of medicine centuries before the Christian era. The practice of medicine, as based upon rational principles and a knowledge of the nature of diseases, has thus oscillated through these varied systems and innumerable theories; and the science of Morbid Anatomy has been marked throughout by unmistakable periods of *progress*, of *stationary existence*, or even of *retrogression*, according as one or other exclusive system had the ascendancy, or as each principle or theory of practice challenged for itself a supreme importance.

The modern doctrines relative to the nature of diseases and the practice of Medicine are guided solely by the dictates of *Physiology*. The ordinary *post-mortem* examinations of the body have long since ceased to furnish us with facts before unknown; and new modes of extending observation and research, by taking advantage of every physical aid to the senses, are diligently looked for by the modern anatomist, physiologist, and physician. Thus the means and instruments which advance the science of physiology are well able to advance our knowledge regarding the nature of disease-processes. For, however widely these may seem to depart from those of health, they are merely modifications of them; and the types of disease-processes are to be sought for in the normal physiological processes by which the body is developed, grows, maintains itself, and finally dies. (BRISTOWE.)

To physiology, therefore, in its most comprehensive sense, and to a knowledge of the natural and normal development of animal and vegetable beings, we must look for future progress in pathology; while the means and instruments which advance physiology will simultaneously advance our knowledge regarding the *nature of diseases*,—a sound knowledge of which can alone enable us to “*appreciate their causes*,” and so arrange measures for the *prevention of many of them*, based on the great truths of science. *Organic chemistry, the microscope, the ophthalmoscope, the sphygmograph, the laryngoscope*, and such like instruments, have opened up new fields of labour, which are being diligently cultivated; and while alterations in the ultimate tissues and organs are more especially attended to, the first beginnings of disease, the development of new formations, and the examination of excretions and of specific products, claim a large share of attention. *Histology*, or the study of the development and arrangement of the tissues in the formation of normal and healthy organs, is characteristic of the anatomical investigations of the present day; while the histology of morbid products and chemico-physiological investigation into the nature of morbid changes, are characteristic of the pursuits of the science of modern PATHOLOGICAL ANATOMY.

It is also a significant fact that now, in the nineteenth century, some of the leading doctrines of the *humoral* pathology which prevailed in the seventeenth have been again revived. The experience and learning of that erudite period are now being made available for modern uses. By the improved means, instruments, and methods of research of modern times, important truths may be sifted from the errors and theories with which they are mixed up in the ancient chronicles of medical science; and when we get analogous conditions of disease with which the phenomena described by the ancients may be compared, “not a few of the apparently

modern beliefs are daily found to have a time-honoured reputation unappreciated before." The chemist and the histologist now combine their researches, and, working hand in hand, we regard them as the most inquisitive anatomists of the time. They lend assistance of the most important kind in laying the foundation of our knowledge regarding the nature of diseases, the details of which can only be made more certain and perfect by taking advantage of every kind of scientific knowledge which can be brought to bear upon medical research, and more especially, —(1.) By physical aids to the senses, extending our means for the actual inspection and appreciation of phenomena, such as by the use of the thermometer, the sphygmograph, microscope, ophthalmoscope, laryngoscope, the stethoscope, and specula of various kinds, aided by a careful study of the writings and labours of the men who have more particularly devoted their attention to observations by such means. (2.) By the knowledge (gradually being made more extensive) of the textures, organs, and functions of the body, whose normal exercise constitutes a healthy existence. (3.) By an intimate knowledge of the normal development of the human textures, as well as those of plants and animals from the fecundated ovum. (4.) Besides these kinds of investigations, the science of practical medicine has been, and is being, advanced by operations and experiments upon the internal organs of living animals. At some of our great schools of medicine such investigations are now being actively, but judiciously, prosecuted and taught under Government regulation and supervision, empowered by an Act of Parliament passed in 1876.

A belief is now rapidly gaining ground, and acquiring a hold on the popular mind, that advances in the science of Medicine in future years will be mainly due to a *better appreciation of the causes of disease*; and just in proportion as our knowledge of physiology and pathology becomes more exact and extended, so will the *causes of disease* be appreciated, and the *occurrence of disease* on a large scale prevented. An amiable and large-minded physician, the late Sir John Forbes, emphatically stated, more than thirty years ago, that "here the surest and most glorious triumphs of medical science are achieving, and are to be achieved." He himself lived to see great and good results; to see improvements in social and sanitary works which continue to be realised, and whose rapid progress is characteristic of the present time. Land-draining and town-sewering have now ripened into well recognised methods. From rude beginnings, insignificant in extent, and often injurious in the first instance, the systematic sewerage of towns and draining of land have become of the first importance. Land has thus, in not a few instances, doubled its value. Town-sewering, with other social regulations, have contributed to prolong human life from 5 to 50 per cent. as compared with previous rates in the same district. Agues and typhoid fevers are reduced in the frequency of their occurrence. Since 1840 an annual mortality in English towns of 44 in 1,000 has been reduced to 27; an annual mortality of 30 has been reduced to 20, and even as low as 15. Not less remarkable reductions have taken place in the mortality and loss of strength in the army and navy (See end of Second Volume on "The Sickness and Mortality at Different Stations"); so that generally it may be said that human life has now more value in England than in any other country in the world—a result entirely

due to better sanitary arrangements (RAWLINSON, "On Sewering of Towns," *Soc. of Arts Journal*, Vol. X., p. 276). The political economist, therefore, cannot now regard MEDICINE in any other light than as a productive art; and the labours of the physician, whether in civil or in military life, cannot be regarded as unproductive labour.

But the science of Physiology (on which much of our sanitary improvements are based) has immeasurably outstripped the science of Pathology in the comprehensiveness of its views, and in the value of its results; while Pathology, in its turn again, has always been, and ought to be, in advance of Therapeutics. The best physiologists have distinctly recognised that the basis of their science must include not only a knowledge of animals below man, but a knowledge of the entire vegetable kingdom. Without such an extensive survey of the whole realm of organic nature, we cannot possibly understand human physiology, and far less comparative physiology. The science of Pathology, therefore (whose aim is to expound the *nature of all diseases*), must be, *à fortiori*, very far behind Physiology. The diseases of the lower animals, for instance, rarely form any part of the study of the student of Medicine. The diseases of plants are almost entirely neglected by him. Yet it is clear that until all these have been studied, and some steps taken to generalise the results, every conclusion in pathology regarding the nature of diseases must be the result of a limited experience from a limited field of observation.

Successful inquiries into Pathology, or the nature of diseases, cannot be said to have commenced till the middle of the eighteenth century, when the great work of Morgagni issued from the press. It was the work of his lifetime. In the eightieth year of his age, and not till then, did he consider himself warranted to publish his observations, *De Sedibus et Causis Morborum* (1761)—a work whose material and circumstances of publication read us the practical lesson, that the more frequently a disease occurs, the more necessary it is that its phenomena should be carefully investigated. And when we think of the prudent reserve, the anxious and the conscientious delay exhibited by Harvey, Morgagni, and Jenner, in the publication of their respective researches, we cannot but contrast the circumstances with those under which the exuberance of medical publications are now given to the world. Morgagni modified and corrected many of the views entertained and promulgated by his predecessors; and the study of the nature of diseases was carried into the commencement of the present century by CULLEN, DE HAEN, WILLIAM and JOHN HUNTER, PORTALL, and BICHAT.

Our knowledge regarding the nature of disease-processes has advanced simultaneously with that of *general anatomy*; and when the component parts of an organ, and of the human body, came to be distinguished, it was observed that membranes and tissues might be individually diseased, while neighbouring membranes and tissues remained untouched. Bichat's idea, therefore, of decomposing the animal body into its elementary parts must be regarded as the foundation of modern Special Pathology and Morbid Anatomy. He pointed out the necessity of studying diseases with reference to the different tissues as separately and specially affected; and it has been since shown, in a remarkable manner, how general anatomy, deduced from the physical properties of parts and crude observation, may coincide with

more minute investigations of a chemical and microscopical kind. The membranes and tissues of the human body, roughly torn asunder by Bichat, are now themselves being daily subjected to a more inquisitive analysis of an anatomical and chemical nature, which unravels them into still more minute histological elements; and what we now want is the systematic study of general pathological conditions conducted in the manner in which Bichat studied general anatomy. We now want a more complete knowledge of the coincident pathological tissue changes. The clinical aspect of chronic changes in the tissues and organs are apt to be overlooked, or altogether neglected. Disease is mostly a chronic process ending in acute changes; and such changes do not soon or often occur in really healthy people. Fibroid, fatty, and vascular changes precede the development of extensive local organic degenerations. But although Bichat entertained the view that each tissue had its own *diathesis*, it is to Cullen and the Hunters, in this country more especially, that the application of the distinctions of tissues was made to illustrate the nature of disease-processes. Cullen's descriptions of diseases are descriptions of groups of phenomena which comprise morbid states. The written labours of the Hunters form but a small part of the memorials of what they did to elucidate the nature of diseases, and only those who have had the opportunity of carefully examining their museums, preserved in London and in Glasgow, are able to form any conception of the comprehensive nature of their labours, or to assign to them a proper place among those who have successfully advanced the science of Medicine. They hold a position in science at least one hundred years in advance of the age in which they lived.

Bichat, Cullen, and the Hunters, in their respective countries, have thus reciprocally influenced and advanced the progress of our knowledge regarding the nature of diseases. And although it was reserved for Bichat to complete a more perfect system of general anatomy, it must not be forgotten that Dr. Carmichael Smith, in 1790, applied his knowledge of textural anatomy to elucidate the nature of disease-processes; and that Pinel, after him, in his *Nosographie Philosophique*, made the distinction between the membranous and other animal structures the foundation of his pathology. The classic work of Baillie (his *Morbid Anatomy*), published in 1793, closed the labours of the past century. If we look to the tendency of the researches of those men just named, including Bichat, the truth gradually asserts itself, that it was necessary to study alterations of structure so as to connect morbid changes with symptoms of diseases and with the operations of ascertained causes of morbid action. The nature of the morbid changes was observed to be more apparent in the progress of external diseases; and therefore surgical experience was brought to bear upon the elucidation of internal disease-processes. One especially marked result of this is to be seen in the modern application of the ophthalmoscope, which reveals information the most important for the diagnosis of many general diseases, by an examination of the interior of the eye.

Thus the progress of Morbid Anatomy is, in a great measure, a record of the history of Medicine; and we can trace the science of special morbid anatomy, giving a character to the various systems of the healing art

which have prevailed from time to time. All the writers up to the time of Bichat, Laennec, and Abercrombie were pure morbid anatomists, who did not connect the effects of disease with their causes, and who recognised the changes of disease as important only in proportion to their magnitude as apparent to the senses. They are therefore regarded as pure solidists, whose researches doubtless contributed much towards a correct knowledge of the changes in the organs of the body, while the condition of the fluids was neglected, as well as the relations of the textures, organs, and fluids, in the combined exercise of their functions. Simple functional disturbances were thus wholly overlooked, and the constitutional connection of local affections entirely lost sight of.

The contemporaneous surgery of the period previous to Bichat was marked by its unwillingness to recognise anything but material facts, mechanical processes and contrivances. The surgeons of those days desired to know nothing but anatomy and mechanics; and, accordingly, it may be recognised as the period of pure anatomical and mechanical surgery, distinguished by the writings of men whose works bear ample testimony that the surgery of the period was founded on exact and even minute anatomical knowledge. No allusion is made, however, by them to medicine—they make no application of physiological truths, and they encourage no therapeutic tendency apart from mechanical or instrumental interference.

Tested by extensive clinical observations, the character of the present period in the history of Practical Medicine is one of *probation* as well as of *progress*, marked by a close inductive examination of past generalisation and classification of facts, however remotely connected, which illustrate the nature of diseases and their treatment. Side by side, since 1816 and 1819, the microscope and the stethoscope have advanced our knowledge of the nature of diseases with a regular and accelerated velocity; but they have only done so as assistants and in subordination to laws and facts whose knowledge we have acquired, by a close observation of general symptoms, of which such instruments have never been intended to take precedence. They have never accomplished, nor can they ever accomplish, useful practical results, to the exclusion of such other methods of observation as have just been noticed. We are not to confound *relative* smallness with *absolute* simplicity, and believe that because a simple organic cell is a small object—because we can see around it, through it, and on every side of it—the functions and conditions of its existence are less *complex* or less obscure on that account than are those of a more large and complex organ, or the functions of a larger living animal. Moreover, we are not to suppose that because the stethoscope enables us to detect a mitral murmur, or a crepitation in a lung, we are justified at once in adopting one, and only one, method of treatment. It is this exclusive use of instruments, to the disregard of general symptoms and signs of disease, derived from close observation and knowledge of the living functions, which sometimes leads to the repudiation of the use of such instruments by the sagacious and experienced physician, who sees the numerous errors not unfrequently committed by his younger brethren, trusting too exclusively to instruments in the diagnosis of disease. Like the stethoscope the microscope has also been unjustly and unnecessarily burdened

with labour, and has been equally unjustly blamed, and brought into unmerited discredit, when it has failed to elucidate the nature or even presence of a morbid state the existence of which could not be doubted, but which the sense of sight could not appreciate, even when presented in small quantities greatly magnified. In such instances the microscope has been applied to uses which it was not the nature or province of the instrument to detect. The gravimeter or hydrostatic balance, the microscope, the stethoscope, the ophthalmoscope, the laryngoscope, the pleximeter, the sphygmograph, and the thermometer, are merely instruments of pathological inquiry, each one adapted for the determination of particular classes of facts. They can only elucidate disease when they are brought to bear upon physical properties, the nature of which they are able to appreciate; and it is only from their *combined and appropriate* use, in connection with a history of the general signs and symptoms of disease in each particular case, that our knowledge of the nature of diseases will be advanced.

The industrious employment of these aids to diagnosis, and an intimate acquaintance with the results, are attended with this further advantage, that such practice and knowledge enable their possessor to appreciate the general symptoms of disease with infinitely greater certainty than heretofore. This is the usual consequence of training in all exact methods of observation. The thorough study of these aids to the senses in appreciating disease leads directly to the possibility of dispensing with them in many instances, after an extensive experience of their use. By means of auscultation and percussion, for example, our attention has been drawn to numerous conditions of the thorax, which enable us to make the diagnosis at the first glance, which had not been possible before, because the conditions for diagnosis could never have been recognised without such physical aids to the senses as that derived from auscultation and percussion. In many cases, from the mere inspection of a patient, a well-instructed clinical student may decide upon the existence of *pleurisy*, *pneumothorax*, *emphysema*, or *pulmonary tubercle*. The initiated are thus frequently enabled to dispense with percussion and auscultation; but if they had never acquired the practical knowledge of the subject—if they had never examined numerous patients by means of these instruments as physical aids to diagnosis—and so learned to determine with great exactness the significance of the various forms and movements exhibited by the thorax, they would never have been able to appreciate their significance. So, also, the physician well instructed in the use of the thermometer may, in hundreds of cases, without its aid, draw conclusions, with great certainty, incomprehensible to others not so instructed; but if, led away by this skilfulness, he is induced to dispense with *exact* thermometrical control, he may soon fall into errors. So it is with the ophthalmoscope, sphygmograph, specula, and all other more or less exact physical aids to diagnosis. Let them be in constant and appropriate use, so that the results may be always taken and compared in connection with other general symptoms of disease. No exclusive doctrine will now stand the test of well-directed pathological inquiry, the main object of which is to connect all organic changes (lesions) and functional derangements with their symptoms and causes, with the view of applying rational remedies and

prophylactics. The too exclusive study of pure organic pathology and morbid anatomy leads to no distinction between the signs and causes of disease; and the obvious tendency of such exclusive study is to exaggerate the importance of the principles it may establish, to hold out no hopes of cure, and to undervalue the power of remedies and remedial measures. To obviate this tendency, it is necessary to have recourse to inductive reasoning, so as to connect all the morbid changes seen and appreciated after death with the signs, symptoms, and causes of disease observed during life. Thus it is that links in the chain of disease-processes which, from a one-sided or exclusive view, appear isolated and localised, are really found to be connected with each other. It may be, also, that they are connected with a long but intelligible series of processes developed during life through the metamorphosis of tissue, and going on in apparent health, or in an obviously morbid exercise of function, under conditions and surroundings which are continually fostering and disposing to disease. The constitutional origin of many local diseases, otherwise inexplicable, then becomes apparent.

Among the more eminent exponents of this rational school of pathology, who at an early period in this country discerned and appreciated such doctrines, we find the names of ALLEN, GOLDING BIRD, SIR ROBERT CARSWELL, GREGORY, HOPE, HODGKIN, MARSHALL HALL, PROUT, WILLIAM STARK, JOHN THOMSON, TWEEDY TODD, HUGHES BENNETT, SIBSON, LATHAM, PARKES, and many others, who, although now no more, have left behind them imperishable evidence of their labours. The younger pathologists of the present day, whose name is *Legion*, follow in the footsteps of these men, extending the fields of observation and the boundaries of the science of Medicine. By them the importance of morbid anatomy is fully appreciated, and its province distinctly defined and limited as follows, namely:—(1.) To detect the “stamps of disease,” or the changes which have taken place during the course of disease in the structure of tissues and organs of the body, commonly called “lesions;” (2.) to demonstrate the exact seat of these “lesions,” or local alterations, which have thus become established.

The investigation and elucidation of the *nature, course, and causes* of those changes, constitute the prominent objects of the science of Pathology. By the aid of clinical observation during life, and *post-mortem* examination of the body, Pathology seeks to establish the relations of the changes which have led to the lesions, and so to connect the general progress of disease with its symptoms, signs, and origin. MORBID ANATOMY goes beyond its province when it attempts to point out the nature of the proximate cause of disease. It is only by the application of inductive reasoning that the connection of causes and morbid effects can be shown; and such constitutes the main object, and is the highest aim, of the science of PATHOLOGY. The *morbid anatomist* finds a lesion or change for what ought to be the natural structure, appearance, or condition of a part. The *pathologist* seeks to connect such lesions with signs, symptoms, and causes of disease during life, that the *practical physician* may suggest a remedy for the disease, that the *sanitary officer* may adopt means for its prevention, and that the *nosologist* may give it a name and a place in his classification.

CHAPTER VIII.

THE ELEMENTARY CONSTITUENTS OF LESIONS AS SHOWN BY MORBID ANATOMY AND OTHER MEANS OF RESEARCH.

WHERE the material effects, "stamps" of disease, or lesions, can be rendered obvious, they are found to consist, for the most part, of—(1.) Morphological changes in the elementary textures of the body generally, and altered conditions of the fluids. (2.) The presence of new formations foreign to the normal condition of an organ or system of organs. (3.) Change in the position or form of some of the organs or parts of organs. (4.) Deposits within or surrounding the elements of tissues, or changes of a degenerative or retrograde kind in them.

The object of prosecuting the anatomy of disease is, therefore, in the first instance, to institute a comparison between the known appearances or standard of health, and such altered states of the parts as evidence of disease.

Means and Instruments of Research.—To institute investigations such as those indicated at p. 7, advantage must be taken of almost every branch of human knowledge. The methods of carrying on pathological research are therefore extremely varied, but may be shortly enumerated under the following heads:—(1.) The opening of dead bodies, to ascertain the condition of their organs and tissues in all that relates to their structural, chemical, and physical properties. (2.) Application of various instruments, such as the microscope, and of means to ascertain the absolute and specific weight of organs or parts, the relations, size, form, and colours of structures, and the like. (3.) Application of chemical investigations to the diseased products. (4.) Application of statistics to determine various points of interest in reference to the nature, course, and complications of diseases. (5.) Means to preserve objects for further study by the microscope, or any other mode of examination. (6.) Experiments instituted on living animals, and, in certain cases, on man, with the view of artificially producing a morbid condition. A careful study of such experiments by the previously-mentioned means affords valuable information, for the causes in action are more under control than those which are spontaneously brought about by disease in the living body.

The immediate object of such investigations is to obtain information regarding the material changes in the different parts of the body which accompany or produce morbid symptoms, and to connect these changes with symptoms and signs of disease during life. We thus learn how morbid products are formed at first and gradually developed; and by combining these two kinds of knowledge we learn the relative connection of two orders of phenomena—namely, (1.) How the perverted properties, disordered actions, or altered structures give rise to perverted or impaired secretions; disordered and irregular motions; deranged, impeded, or interrupted functions—in other words, the "*order of invasion of disease*—

processes” is learned from such investigations. And, (2.) We are thereby taught how parts, once the seat of morbid change, return by various processes of nutrition, growth, repair, or reproduction, to their normal condition. The questions arising out of such investigations are, or ought to be, the first object of thought to the conscientious medical practitioner. It is his duty, from an attentive consideration of the signs and symptoms of disease, to form an idea, as accurate as possible, of the nature and extent of the morbid action or change which is going on, or which may be set up, in the tissues, organs, and fluids of the living body. If, therefore, he does not avail himself of every means and instrument by which he can ascertain the existence of change in the dead body, and its alteration from some standard of health—if he does not embrace every opportunity of making *post-mortem* examinations—if he contents himself merely with observing signs or symptoms of disease without witnessing the changes of structure, if any, which may give rise to them—he can have little conscious satisfaction in the study of Medicine as a science, or in the practice of the healing art. In the words of Cruveilhier, he will, during his lifetime, “see many patients, but few diseases.”

Forms of the Constituent Elements of Lesions.—The histologist has now ascertained the various simple organic forms which compose the textures in their normal state, and the mode in which these textures are arranged and combined so as to form the organs and systems which carry on the healthy functions of the body. The pathologist has also made out (although with less completeness), by the methods of observation and experiment already indicated, the various simple organic forms which constitute the elements of those material changes whose phenomena of growth, decay, and varied change are associated with the manifestations of disease. By classifying and arranging these forms, we obtain more or less clear ideas of lesions; and we ascertain that the morbid processes follow, in their development, a very definite order of change, but not yet determined with absolute certainty in each disease. An anatomical investigation of morbid parts, conducted with the aid of the microscope and other instruments of research, shows that the material of which their substance is made up is of various structure, sometimes combined in forms of one kind throughout, and sometimes varied by the development and combination of many elementary forms, more or less solid, soft, or fluid.

An analysis of the morbid material, carried as far as scientific means at present enable us, shows that the elementary conditions in which morbid products are found may be described as follows:—

(1.) Fluid matter and hyaline substance, more or less soft, soluble, and probably nutrient to surrounding or imbedded morbid elements. (2.) Simple elementary forms of the nature of deposits, sometimes of a mineral or inorganic character:—*e.g.*, (a.) amorphous granules; (b.) crystalline structures in a granular state. 3. Simple, but organised products (minute rounded particles, nuclei, or germinal matter) capable of growth—*i.e.*, which live, change, convert, or germinate:—*e.g.*, (a.) amorphous masses (*plasm germs, bioplasm, protoplasm*); (b.) nuclei or granules; (c.) compound corpuscles; (d.) simple cells; (e.) fibres.

The various appearances and conditions which these simple forms may assume in disease, as well as the functional states with which they are

frequently associated, lead to a further enumeration and classification of morbid elementary products, as well as of more complex disease-processes, as below :—

A.—MORBID ELEMENTARY PRODUCTS.

I. EXUDATIONS MORE OR LESS SOFT, SEMI-FLUID, OR FLUID, AND FORMED OF—(a.) Germinal, plastic, and formed material, sometimes called *blastema*, *plasma*, *bioplasm*, *protoplasm*, *coagulable lymph*, *false membrane*, or *fibrine*, as seen adhering to free surfaces. (b.) Aqueous matter, as seen in the morbid state termed “*dropsy*,” and “*œdema*” of parts. (c.) Gaseous exudations, as seen in the various forms of *pneumosis*—e.g., *emphysema*, *flatulency*, *tympanites*, *pneumo-thorax*.

II. EXUDATIONS MORE OR LESS CONSOLIDATED, AND CONSISTING OF—(a.) Molecular or granular material, from the 800th of a line to an immeasurably small size, and consisting chiefly of the following simple elements,—(1.) Forms of an organic kind capable of growth, which live, grow, multiply, or germinate, and invariably take origin from a pre-existing structure, (bioplasm, protoplasm, &c.) (2.) Fatty molecules or granules. (3.) Deposits of an inorganic kind, generally calcareous salts. (4.) Pigment granules.

(b.) Coagulable compounds, resisting the action of most re-agents, such as are seen in the elements of *tubercle*, *scrofula*, *oleo-albuminous formations*, *lardaceous degenerations*.

(c.) Exudations of a transitional nature, organised, which are capable of growth, which may become vascular, which grow from pre-existing structures, and which are composed of—(1.) Consolidated homogeneous material, passing to—(2.) A fibrilloid arrangement of the molecular or granular particles composing connective substance, and a subsequent formation of fibres in it or from it. (3.) The formation of pyoid cells, the fibro-plastic or connective tissue cells, passing into fusiform cells and fibres as the material becomes consolidated. (4.) The formation or exudation of fluid matter holding pus, or other more compound cells.

III. GROWTHS AND EXUDATIONS OF A SPECIFIC KIND.

(a.) Lymph of small-pox and cow-pox, just removed from the vesicle, contains a great number of extremely minute particles. To these the active properties of the lymph are entirely and solely due (BEALE). (b.) Matter of glanders, of malignant pustule, and of the plague, contain similar particles. (c.) Fluid of infecting chancre, and of some forms of secondary syphilitic lesions, containing multitudes of living particles. (d.) Material of tubercle and scrofula. (?) (e.) Material of cancer. (f.) The growth in Peyer's glands during typhoid fever. (g.) The growth in Peyer's glands in cases of cholera. (h.) Melanotic or pigmentary germs.

The minute elements of all of these resemble each other so much in their microscopic appearances, that they cannot be distinguished from each other, or from pus; and pus containing specific contagious properties cannot be distinguished from ordinary pus, except in the matter of vital

power or virulent specific properties, as evinced by its effects. The experiments and observations of Dr. Burdon Sanderson tend to show that the specific material of each of these diseases consists of living germs; and many there are who now believe that the day is not far distant when we shall be able to demonstrate the material poison of each specific disease, just as the chemist is able to show the active principle of substances like opium, cinchona, and the like.

IV. MATERIAL OF A COMPLEX KIND. (a.) Media of repair and production of injured or lost parts—substance of granulations and cicatrices; (b.) Hypertrophy of parts; (c.) Tumors, either innocent or malignant; (d.) Concretions.

V. PARASITIC FORMATIONS, AND DISEASES INDUCED BY PARASITES.

B.—COMPLEX VITAL MORBID PROCESSES WHOSE PHENOMENA, MORE OR LESS COMBINED, CONSTITUTE DISEASE.

(1.) *Catarrh*. (2.) *Inflammation*, comprehending the following forms: (a.) *Ulcerative*; (b.) *Suppurative*; (c.) *Plastic*; (d.) *Rheumatic*; (e.) *Gouty*; (f.) *Pyæmic*; (g.) *Syphilitic*; (h.) *Scrofulous*; (i.) *Gonorrhæal*. (3.) *Gangrene*. (4.) *Passive congestion*. (5.) *Extravasation of blood—Hæmorrhage*. (6.) *Dropsy*. (7.) *Fibrinous deposit*. (8.) *Alteration of dimensions*, such as (a.) *Dilatation*; (b.) *Contraction*; (c.) *Hypertrophy*; (d.) *Atrophy*. (9.) *Degenerations*, comprehending (a.) *Fatty degeneration*; (b.) *Mineral degeneration*, or *Petrifaction*, these two (a.) and (b.) being the components of *atheroma* and so-called *ossification*; (c.) *Pigment degeneration—Pigmentation*; (d.) *Fibroid degeneration*; (e.) *Lardaceous or albuminoid degeneration—lardaceous, waxy or amyloid disease*. (10.) *Cyst*. (11.) *Calculus and concretions*. (12.) *Malformation*. (13.) *Functional diseases*. (14.) *Fever*.

The above classification is that given in the Nomenclature of the College of Physicians, and is merely intended to bring before the student at a glance the variety of morbid material which is concerned in the expression of many phenomena seen in the course of diseases, the distinctions made being mainly based on structural analyses. While it is more properly the province of the anatomist to describe the MORBID ELEMENTARY PRODUCTS which compose a lesion, it must ever be remembered that there are many important diseases which have no appreciable morbid anatomy. On the other hand, it is the COMPLEX VITAL MORBID PROCESSES, WHOSE PHENOMENA, MORE OR LESS COMBINED, CONSTITUTE DISEASES, with which the Physician has more immediately to deal; and these complex states obtain special notice in the following chapters.

CHAPTER IX.

COMPLEX MORBID STATES.

CATARRH.

LATIN EQ., *Catarrhus*; FRENCH EQ., *Catarrhe*; GERMAN EQ., *Catarrh*; ITALIAN EQ., *Catarro*.

Definition.—*Hyperæmia tending to engorgement of the blood-vessels of any region of the mucous membrane. An abnormal secretion of fluid oozes out, and flows from the surface. The tissue is succulent and swollen; and there is a copious generation or proliferation of young cells, pushing their way to the surface, and so contributing to the flux.*

Pathology and Anatomical Characters.—The regions of the mucous tract most prone to catarrh are the nose, mouth, pharynx, larynx, bronchi, stomach, intestines, gall ducts, urinary bladder, urethra (male and female), uterus, and vagina. These several regions give names to the catarrhal disease:—*e.g., acute or chronic bronchial catarrh; virulent or non-virulent catarrh of the urethra; uterine catarrh; vaginal catarrh, and so on; and some of these several forms of catarrh will be again noticed under the respective local diseases under which they are classified.*

A gradual flow of fluid secretion, known as “*mucus*,” from the mucous surface, is characteristic of catarrh, and hence the name. Under the microscope numerous transparent cells may be seen, called mucus-corpuscles, which are detached young epithelial cells from the deeper layers, or from the mucous glands, their formation being limited at first to a mere increase of the epithelial cells, the multiplication of which is at first normal, but attended with some hyperæmia of the mucous membrane. Very soon, however, the substance of the mucous tissue is swollen, moist, and flabby; and the submucous tissue may become the seat of considerable serous infiltration (oedema),—a condition of great danger when the mucous membrane of the larynx is involved. The increased secretion of mucus becomes rich in the young corpuscular elements, and is increased in tenacity, adhering firmly to the surface. The fluid contains an increase of “*mucine*,” to which this tenacity is due. The redness of the membrane is sometimes diffuse, due to uniform injection of the blood-vessels; sometimes mottled, when it is due to ecchymosis. The texture is softened, so as to be easily torn.

The first effect of catarrh is marked by an increase of the secretion of the mucus proper and normal to the part—a peculiar viscid, ropy, pellucid substance, without any visible corpuscles or organised particles. With an increase of this material there comes the mixture of epithelial particles from the mucous membrane, or corpuscles from the follicles and follicular glands imbedded in its substance. The follicular glands thus become enlarged from distension, by the rapid multiplication of their cell elements. After a time their contents soften, and at last

rupture takes place, leaving a follicular ulcer, as is often seen in the site of the solitary follicles of the pharynx and of the intestines. The particles of increased growth vary in shape and properties according to the part of the mucous tract whence the catarrhal secretion flows; and they increase in number from premature desquamation, while their characteristic local shapes are less and less perfectly preserved. With such increase of flow, there is afflux of blood to the part, and infiltration of its substance with fluid material, making the membrane thick and spongy. It is swollen also by the increased production of the corpuscles which may arise from nuclear multiplication, both within the epithelial cells on the surface of the membrane, or within its follicles, and in the corpuscles of the subepithelial connective tissue, from a greater or less depth, according to the degree of the irritation or inflammation in the part (Fig. 1). These corpuscles are commonly called

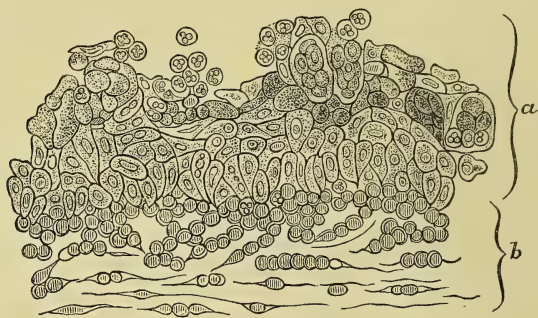


Fig. 1.*

mucus-corpuscles, and they differ only from the corpuscles formed from a serous membrane in a state of inflammation in the greater viscosity of the fluid in which they lie (PAGET, TURNER, REMAK).

The results of catarrh are—(1.) *Atrophy*, most frequent in stomach and intestines, after the proper gland structures have become so much involved that they become choked up, and finally disappear with the softening of the mass of superabundant epithelium; (2.) *Suppuration*, the increased flow of mucus becoming purulent, the mucous membrane becoming permanently thickened, the submucous connective tissue increased in amount; lymph structures enlarged; adhesion of fæces and discoloration; (3.) *Abrasion or ulceration*; (4.) *Thickening and induration of the submucous tissues*; nodular growth of polypi and warts from long continued irritation of the surface; (5.) *Pigmentation*. When the catarrh has become chronic, the evidence of this chronicity is seen in the extensive pigmentation of the mucous surface which has been so frequently implicated in the morbid process. It has a brownish colour, from the pigment derived from oft-repeated ecchymosis in its substance. The submucous tissue becomes thick, firm, and hypertrophied; the blood-vessels, varicose and gorged with blood. The mucous secretion is now made up of an opaque, yellow-coloured, tenacious substance, com-

* Catarrhal proliferation of epithelium of the conjunctiva, showing the origin of young elements within parent cells. (a.) Epithelial elements; (b.) connective tissue beneath the epithelium. (After RINDFLEISH.)

posed of cells indistinctly granulated, and containing divided nuclei—a “muco-purulent” secretion—where the mucus cannot be distinguished from the pus cell.

There seems to be a certain predisposition, or structural development, of the individual prone to catarrhal attacks. They are prone to occur in conditions characterised by poverty of blood and nutrition, in which the walls of the capillary vessels have a feeble power of resistance; while the tissues through which such vessels pass are at the same time soft and yielding. The predisposition to catarrh is strongly marked in scrofula and rickets; and, as bronchial catarrh, the morbid condition is a frequent attendant on typhoid fever, measles, and small-pox. Effeminate habits also augment the liability to catarrhal affections.

Catarrh is often regarded as an inflammation, and is so described under the name of *Catarrhal inflammation*; the other forms of inflammation of mucous membrane being the “*Croupous*” or “*Pseudo-membranous*,” and the “*Diphtheritic*,” to be considered under the next topic, namely:—

INFLAMMATION.

LATIN EQ., *Inflammatio*; FRENCH EQ., *Inflammation*; GERMAN EQ., *Entzündung*; ITALIAN EQ., *Inflamazione*.

Definition.—*A complex morbid process characterised by,—(1.) A suspension of the concurrent exercise of function among the minute elements of the tissue involved; (2.) Changes in the blood-vessels and circulation, consisting of—(a.) Stagnation of the blood and abnormal adhesiveness of the blood-discs in the capillary vessels contiguous to the tissue-elements whose functions are suspended; (b.) Contraction of the minute arteries leading to the capillaries of the affected part, with subsequent dilatation and paralysis of the contractile tissue of the affected blood-vessels. 3. Exudation of liquor sanguinis, and permeation of blood corpuscles through the walls of the blood-vessels. (4.) Altered nutrition of inflamed tissues, so that the nutritive changes between the blood and the minute component elements of the affected tissue become visibly altered; and if the inflammation persists, an appreciably excessive interstitial exudation infiltrates the parts, with a constant tendency to a profusion of growth, to which this interstitial exudation ministers abundant nutrition. This excessive exudation, which follows as a result of the inflammatory state, is apt to be associated with an unhealthy condition of the blood and of the blood-plasma, giving rise to varied types of inflammation associated, with varied forms of new growth, according to,—(1.) The elementary structure in which it occurs; (2.) The specific, constitutional, or local disease with which the inflammation may coexist; and, (3.) According to the progress of the inflammation, the amount and suddenness of the effusion, the extent of tissue involved, the diminished vascularity, and the powers of absorption of the surrounding parts.*

Pathology.—As it is not possible clearly to define the limits of natural processes, it is not possible to give a correct definition of inflammation. It is a process the most important of all morbid states; and a knowledge of its phenomena (at this very time—1879—undergoing an extensive reconsideration), the laws which regulate its course, and the relations which

its several events bear to each other, have been always considered as "the keystone to medical and surgical science," and the "pivot upon which the medical philosophy of the time has revolved." It is not wonderful, therefore, that much has been written on the subject of inflammation, more especially since microscopic research has been brought to aid in the investigation of its phenomena. Among the many who have investigated this morbid process with success, and by whose original observations its study may be said to have begun, the names of Wilson Philips, John Thomson, Gendrin, Kaltenbrunner, Gerber, and Müller; and more recently, those of Alison, Lebert, Gulliver, Addison, C. J. B. Williams, Bennett, Wharton Jones, Henle; and still more recently, Virchow, Paget, John Simon, Joseph Lister, Stricker, Sanderson, Klein, Cohnheim, Prussak, Dr. George Thin, and Recklinghausen, are well known; and no account of inflammation can be complete which does not embrace the results of the labours of these men.

The early experiments which illustrate the nature and phenomena of inflammation were made chiefly on the web of the frog's foot, the folds of the frog's mesentery, and the cornea of the frog. The phenomena in this cold-blooded animal are found to correspond, in all essential points, with the results of experiments performed on the more or less transparent parts of warm-blooded animals; such, for example, as the wings and ears of bats, the ears of rabbits, the mesenteries of these animals, the brains of rabbits and of pigeons, and the legs of dogs, upon the bones of which compound fractures had been inflicted.

As a general result of such experiments and observations, it may be stated that the chief constituents of the inflammation-process are to be found, in (1.) Altered conditions of the healthy nutritive changes—the phenomena of the abnormal state becoming more or less obvious by—(2.) Changes in the blood-vessels, and in the circulation. (3.) Exudation of *liquor sanguinis*, and transudation or permeation of the walls of the blood-vessels of the inflamed part by the white blood-corpuscles (*leucocytes*) without rupture into the surrounding texture.

Thus the process of inflammation is one in which many stages of morbid action are passed through; and the series of complex changes through which the inflammatory process is seen to proceed, as observed in the transparent parts of animals under the microscope, are found to occur nearly in the following order:—1st. The beautiful experiments and observations of Mr. Joseph Lister, Professor of Clinical Surgery in King's College, London, clearly prove that a suspension of the concurrent exercise of function among the minute elements of the tissue involved is the primary lesion in the hyperæmia of inflammation, which immediately leads to—2nd. Inflammatory derangement of the blood, which, in the vicinity of the impaired tissue-elements, tends to assume the same characters as blood always assumes when it is in contact with ordinary solid matter, and which renders it unfit for transmission along the course of the blood-vessels. But a return of the tissue-elements to their usually active state will be associated with a restoration of the blood to the healthy characters which adapt it for circulation (*Royal Society*, June 18, 1857). Thus the normal essential requirements of blood and tissue mutually counterbalance each other. If blood is depraved, tissue must suffer; and if tissue is morbid, blood becomes

morbid too, and may remain so if depuration is not completed. 3rd. The arteries of the affected part are at first narrowed, and the blood flows through them with greater rapidity. 4th. The same vessels subsequently, and sometimes suddenly, become enlarged, and the current of blood is slower. 5th. The flow of blood becomes irregular. 6th. All motion of the blood ultimately ceases, and complete stagnation ensues. 7th, and lastly, The *liquor sanguinis* is exuded through the walls of the blood-vessels, accompanied by the permeation through them of the white blood-corpuscles.

These different phenomena are associated with the production of the more obvious symptoms (and to some extent they physically account for them)—namely, redness, pain, heat, and swelling. But although these changes are here mentioned consecutively, it is not to be understood that in every instance of inflammation such changes can be traced in distinct succession. They are to be studied and looked upon as *nearly concurrent*, rather than as a distinct *series of events*, each of which stands in the relation of a consequent to one or more of its antecedents, and which, for the purposes of study, are here enumerated in sequence. An analysis of these concurrent phenomena has shown that the conditions for the healthy nutrition of the part are materially changed, somewhat as follows :—

I. The supply of blood to the part is altered,—(a.) By the changes in the blood-vessels, especially the narrowing of the arteries and subsequent enlargement of the capillaries; (b.) By the mode in which the blood moves through them; (c.) By the escape through the walls of the blood-vessels (veins and capillaries) of the white and red blood-corpuscles. This primary narrowing is denied by some, or rather, not having been observed, it has been stated that dilatation of the arteries commences at once, “and is not preceded by any contraction.” But the narrowing of the arteries, in the first instance, may be demonstrated under the microscope by the application of warm water simply to the web of the frog’s foot; and the same phenomena are presumed to occur in man, for the following reasons :—Sudden operations of the mind, and the application of cold, produce paleness of the skin—an effect which can only arise from contraction of the minute arteries, and the diminution of the quantity of blood thereby conveyed by them. The subsequent enlargement of the capillaries is presumed to be a constant event in the inflammation of a part. It gradually increases for about twelve hours, and it usually extends to some distance around what may be considered as the chief seat, centre, or focus of diseased action; but in some textures the enlargement and reddening are peculiar to the vascular parts in the vicinity. To this condition of the blood and blood-vessels the changes in the blood and blood-vessels, resulting in increased vascularity, or hyperæmia, is to be ascribed the usually first observable symptoms of inflammation in a part—namely, the redness, and also the heat and swelling.

But there are many circumstances under which inflammation has existed, and yet no redness is apparent in the part itself. We often find the cartilages of the joints ulcerated, and yet not a trace of a red vessel. Although in certain parts—extravascular tissues, such as the

cornea and the articular cartilages, and the arterial tissues—the previous existence of inflammatory action is obvious from the effects produced, and where no blood-vessels existed obvious to the eye, assisted or not by the microscope, yet it is, for the most part, found that enlargement of the blood-vessels of the adjacent parts, and especially of those from which the diseased part derives its nutrient supply, is a constant phenomenon, purely functional, which appears to be developed indirectly through the medium of the nervous system. In inflammation of the cornea, for instance, the blood-vessels of the sclerotic and conjunctivæ are enlarged. In ulceration of the articular cartilages, the surrounding synovial membrane and the articular extremities of the bones are more fully pervaded with enlarged blood-vessels. The *vasa vasorum* of the aorta round the morbidly thickened part are also the subject of enlargement, and the channel of increased supply of blood,—a series of vascular changes ministering to the act of inflammation in each of these extra-vascular parts. There is, therefore, no doubt that the conditions favourable to the existence of redness are always present to a greater or less degree at the early period of inflammation; and whether the redness be always present or only slightly perceptible, the same impairment of function among the minute elements of the tissue, and increased adhesiveness of the blood-discs, not only to each other, but to the tissue of the capillaries, take part in the development of the inflammatory process. The redness is of various intensity and shade, according to the degree of the inflammation, its stage, and the structural arrangement (especially as to blood-vessels) of the part affected. Its shades pass from a light rose-colour to a deep crimson, or even purple. It assumes the form of points where congeries of minute blood-vessels are concerned; or streaks, as where the vessels of fibrous structures are inflamed, as in tendon; or a series of minute and fine ramifications, as in synovial structures; and generally, it may be stated that the form of the redness derives its character from the normal arrangement of the capillaries of the part. The redness is most intense towards the centre of diseased action, gradually softening down towards the circumference, where the conditions of health exist. This gradual shading off serves to distinguish the redness of inflammation from the redness of extravasation. The margin of an extravasation is defined, its redness cannot be removed by pressure; while the disappearance of inflammatory redness under pressure is, to a certain extent, a measure of the activity of the circulation in the part. The brighter hues generally attend ordinary active inflammation; the darker hues of inflammatory action are generally associated with some specific cause of disease, a feeble action of the parts, or a tendency to gangrene. The increased depth of colour is mainly due, in the first instance, to the hyperæmia and stagnation of blood in the existing vessels, and not in any measure to the formation of new ones. The redness, however, always appears more than proportionate to the enlargement of the blood-vessels; and the red corpuscles are intensely adherent in the enlarged capillaries. The enlargement of the blood-vessels varies. It may be hardly perceptible, or it may increase their diameter to two or three times their natural size. John Hunter established this stage of the inflammatory process in the ear of a rabbit, by thawing it after it had been frozen: the rabbit was killed during the process, and

the head being injected, the two ears were removed and dried. Wood-cuts representing the comparative conditions of the two ears may be seen in the first volume of PAGET'S *Surgical Pathology*, p. 295, or at p. 221 of the edition edited by Professor Turner. The blood-vessels of the inflamed ear became greatly larger than those of the healthy one, and it was found that arteries before invisible in the healthy state of the rabbit's ear, were brought clearly into view during the stage of the inflammatory process.

The dilated vessels of an inflamed part appear crammed with red corpuscles, which lie or move as if no fluid intervened between them, or as if they were imbedded in a hyaline substance due to the solidification of the fibrine of the *liquor sanguinis*. An increase of redness is sometimes seen to depend upon extravasation of blood, the distended and softened capillaries breaking from the pressure of the blood, or the effusion of the colouring matter of the blood-corpuscles as well into the spaces between the blood-corpuscles as into the adjacent tissue through the walls of the blood-vessels. Lastly, the redness is sometimes intensified (as Hunter first suggested, and microscopic examination subsequently proved) by the passage of the blood unchanged from the arteries into the veins. No new formation of blood-vessels is necessarily concerned in the redness of inflamed parts; but as inflammation continues and advances, new blood-vessels gradually develop themselves, which, like the old ones, become filled with blood. When inflammation has subsided, these new vessels pass into any new growth of tissue which may have arisen, as if for its nutrition, development, and continued growth, or to effect its subsequent removal, degeneration, decay, or absorption.

Peculiar changes of shape of the blood-vessels are associated with their



Fig. 2.*

enlargement and irregular movements of the blood, consisting chiefly of tortuosity of distribution and aneurismal or varicose dilatations. The aneurismal or varicose state is seen to take place most frequently in the soft textures, as in the brain, where it is a frequent condition of the inflammatory red softening (KÖLLIKER and HASSE); and in subcutaneous

* Dilated blood-vessels in inflammation (after PAGET).

tissue the points of what appears to be extravasated blood are aneurismal dilatations of capillary vessels filled with the red corpuscles (LEBERT), as shown in the wood-cut (Fig. 2).

These varied conditions of the blood-vessels affect the motion of the fluid in the part; and, consequently, the supply of blood for the purposes of nutrition. Generally, it may be stated that there is stagnation of the blood in the focus or centre of severe inflammation. This stagnation is surrounded by a state of fulness of vessels, and *slow movement* of the blood, so that there is considerable retardation of the circulation, the vessels remaining dilated, more or less regularly, while farther around, and more distant still, there is fulness of the vessels, with a *rapid movement* of the blood. From the discrepancy existing among observers regarding the statement as to whether the motion of the blood is slower or quicker when the vessels are contracted or dilated, there is evidence that the contraction alone of a vessel, or its dilatation alone, is not always sufficient to cause the current of blood to be either slow or quick.

Other conditions are at work which contribute in no small degree either to accelerate or to slow the rate of movement in the vessels. Besides the force of the heart's action, there is a mutual relation which subsists between (*a.*) the blood, (*b.*) blood-vessels, and (*c.*) surrounding tissue, which materially influences the active motion of the blood. In the healthy body this mutual functional relation between the minute elements of tissue and the blood is necessary to maintain it in a state fit for transmission through the vessels. The mere contraction of the arteries leading to a part does not tend to stagnation of the blood in the capillaries of the inflamed part; on the contrary, the movement onwards of the blood in the vessels is influenced or modified by the vital functional processes going on between the capillary vessels and the surrounding elements of tissue; and which has been variously named the "capillary force," the "vital force," the "nutritive force." It is also mainly influenced by the action of the heart itself, and by the physical condition of the vascular tubes through which it has to pass. Accordingly, at first, with contraction of vessels, the current has been described as being quickened. It also sometimes slackens, or even retrogrades for a time, and not unfrequently oscillatory movements may be noticed. But when dilatation is complete, the blood flows with rapidity, and a greater quantity passes during a given time than in the unexcited state of the parts. This is known as the state of "*determination of blood to a part,*" or "*active congestion.*" The natural function of the part thus becomes simply exalted at the commencement of inflammation, and it may be said that a step beyond this will pass the confines of that neutral ground which exists between health and disease. With an increased circulation, and such "*determination of blood to a part,*" functional activity is not only maintained, but is promoted and increased; and unusual transudation of the nutrient material may take place, chiefly of the serum of the blood. Hence the œdema which surrounds an inflamed part.

After a time the motion of the blood becomes slower, usually suddenly slower, at first in the veins, while the volume propelled is increased, and the retardation gradually increases till the blood-corpuscles are no longer propelled floating in their *liquor sanguinis*; but, accumulating in masses,

they advance by a jerking intermittent motion, till at last complete stagnation takes place. The rapidity of the current varies, however, both in arteries and veins. In some it may be more rapid, in others more slow, the crowd of blood-corpuscles being seen to oscillate to and fro, or even to be for a time completely stagnant. The blood-corpuscles now detained exhibit a marked tendency to adhere alike to the walls of the vessels and to each other, accumulating together and sticking in the capillaries, while the *liquor sanguinis* flows onwards, passing by them with diminished velocity. To this condition the term "stasis" has been applied. In the immediate neighbourhood, and surrounding the part which is in the condition of *stasis*, the circulation of the blood goes on with increased rapidity; it may even pulsate in the arteries and oscillate in the veins, while it moves with a uniform but rapid flow through highly distended but less turgid vessels. When these conditions exist simultaneously, and the true morbid process is completely established, the capillary vessels may burst, causing hæmorrhage or extravasation into the surrounding tissue, or the serum and *liquor sanguinis* may transude through their walls, without rupture, into the surrounding texture. But not only does the serum and *liquor sanguinis* make way without rupture through the capillary walls (whose texture is peculiarly permeable), but the observations of Professor Stricker conclusively show that capillary canals undergo changes of calibre, while the blood-corpuscles, both white and red, sink into and through the substance of the unbroken capillary wall. This migration of the corpuscles, or permeation of the blood-vessels by them, was demonstrated by Cohnheim in the mesentery of the frog, after it had been

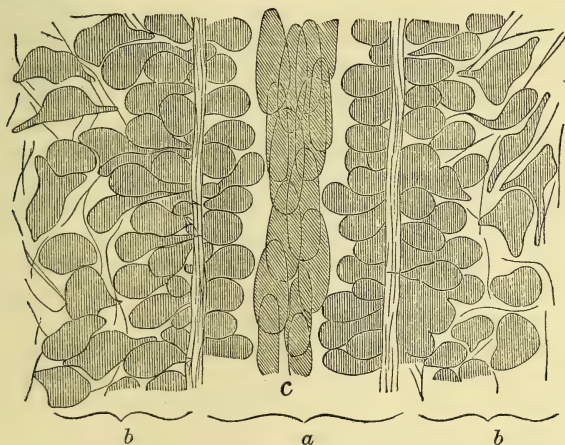


Fig. 3.*

paralysed by the subcutaneous injection of *curare* poison. To see this the mesentery must be stretched over a ring of cork and directly observed. The white blood-corpuscles (*leucocytes*) will be seen to accumulate in large numbers, in the veins especially which dilate, and the colourless blood-corpuscles first remain clinging to the inner surface of the wall of the blood-vessel, then gradually sink into it, and a process of the cells passing

* Cohnheim's experiment. *a*, vein; *bb*, contiguous connective tissue permeated by migrating colourless blood-corpuscles; *c*, column of red blood-corpuscles, 1-500.

through it, swells up outside, and eventually the cells escape through altogether into the surrounding tissue. They have been seen in various stages of progress passing through the blood-vessels (Fig. 3). Small button-shaped elevations are first seen to spring from the outer wall of the vessel, and gradually increase in size till they assume the form of pear-shaped bodies, adherent by their small ends to the vascular walls externally. Finally, this pedicle of attachment by the protoplasm of the capillary structure to the blood-corpuscle gives way, and the white blood-corpuscle remains free outside the vessel. Here the cells continue to exhibit abnormal activity of amoeboid movements, and so wander farther; and if a portion of tissue is irritated, this generally decides the direction of their course. Then they accumulate more and more, and form by such accumulation a quantity of embryonal formative tissue, which becomes the point of departure for all future changes by growth and secondary multiplication by division. Thus they rapidly increase in numbers. Sometimes these blood-corpuscles will remain with half their volume within and half their volume without the blood-vessels, having an hour-glass, constricted shape, the wall of the blood-vessel in this case having an amoeba-like capacity for being protruded or impressed upon, permitting the corpuscles to permeate or pass, as a swimmer passes through fluid without leaving a trace of his way. Dr. William Addison, in 1842, published, in *Transactions of Provincial Medical Association*, and in subsequent writings, that white corpuscles not only accumulate in the vessels of inflamed parts, and adhere to their walls, but that they seem to become incorporated with the substance of the vessel, and to pass through it (without permanent solution of continuity) into the surrounding tissue, where they constitute the corpuscles of inflammatory lymph, pus, or mucus. The red corpuscles act similarly. Dr. Augustus Waller recorded similar observations in 1846 (*Philosophical Magazine*, Vol. XXIX.) In 1867 and 1868, Cohnheim of Berlin came to the conclusion, from experiment and actual observation, that not only the white but the red corpuscles pass through the walls of capillaries and veins of irritated and inflamed parts; and that they may even migrate to some distance from the vessel through which they have permeated,—their movements being like those of an amoeba; that is, having a power of spontaneous movement by various changes of form, as by sending out processes, blunt or thread-like, to be again withdrawn and the spheroidal form resumed. Thus, by repetitions of these changes of form, the blood-corpuscles change from place to place. He believes (as did also Addison and Waller) that the white corpuscles (*leucocytes*) become pus-cells. Two novel features have now therefore been slowly demonstrated since Addison's observations on the phenomena of inflammation in 1832—namely, *first*, the yielding permeability of the soft, nucleated protoplasm which forms the walls of the living capillaries; and, *second*, the spontaneous amoeboid movements possessed by the corpuscles of the blood. Any visible pores, or stomata, are not found to exist in the walls of the capillaries. Professor Recklinghausen, in Berlin, Professors Burdon Sanderson and Bastian, of the London University, have each confirmed these observations.

The "determination of blood to a part," characterised by dilatation of the arteries with increased flow of blood through the capillaries, must be

distinguished from the "*passive* congestion," characterised by the accumulation and stagnation of red and white corpuscles in the vessels, tending to be abnormally adherent to each other and to the vessels, associated with low vital activity. Both of these phenomena, namely "determination" and "congestion," may result from irritation. The dilatation of the arteries seems to be immediately developed through the medium of the nervous system, while the accumulation of the blood-discs and stagnation of the blood is the immediate and direct result of impaired or suspended function of the minute tissue-elements contiguous to the capillary vessels. This "determination of blood" and dilatation of the arteries lead to no change in the quality of the blood itself; on the other hand, accumulation and stagnation of blood, in the congestion of an inflamed part, are associated with increased adhesiveness of the red and white discs. Mere determination of blood becomes obliterated after death by the *post-mortem* contraction of the arteries, whereas passive congestion is persistent. It is an evidence of organic lesion declaring itself as distinctly in the dead as in the living; and thus the most important, if not the only sign, of the early stage of inflammation having occurred during life, is recognisable, on dissection, by the intense redness due to the accumulation of red discs adherent to each other in the minutest ramifications of the vessels, and not due to dilatation of the vessels merely.

Such is a statement of the facts ascertained regarding the early phenomena of the inflammatory process. They are of such a kind that, with the facilities of study which ought now to be within the reach of every student of medicine, he ought to make such experiments as have been already noticed, or see them made by others, and thus really appreciate the steps of that morbid process which he requires to treat so extensively in practice, and of which he can form but a faint conception from the most lucid description.

II. The constitution of the blood in inflammation is altered as regards its adaptability to nourish the part.

The nature of this alteration cannot be chemically expressed; but microscopical observation has established one fundamental fact—namely, that the tissues through which the blood flows have such special relations to the living fluid that, in the healthy state, the functional activity of the minute tissue-elements maintains the blood in a state fit for transmission through the blood-vessels; and the first change observed in the blood subsequent to impaired function of tissue-elements, is an increase of adhesiveness of the red as well as of the white corpuscles. These, however, are now known to be susceptible of much greater adhesiveness than the red; so that slight irritation, leading to impairment of function, causes stagnation of the white sooner than of the red discs. The blood is not thus altered in the first instance throughout its whole mass; but the change is a local one, confined to the seat of the inflammatory process. At one time it was believed that the blood was altered in its constitution, chiefly by an increase of the fibrine and the white corpuscles; but it is now found that the white or rudimental corpuscles of the blood cannot be separated from the fibrine by any known process, consequently the relative amount of fibrine cannot be correctly stated in

relation to the blood. And as in many inflammations these corpuscles are increased, as well as in many conditions—such as pregnancy, in which no inflammatory process exists—the blood is similarly altered, it is not known how much of change is due to fibrine or how much to the white corpuscles.

A remarkable phenomenon presented by the red blood-corpuscles in inflammation was first observed in 1827 by Mr. Lister, Sen., and by Dr. Hodgkin, and afterwards accurately described by Mr. Wharton Jones. They observed that when healthy blood is received on a glass plate, or the clean surface of a polished lancet, and immediately examined, the corpuscles lie diffused in the *liquor sanguinis*, but in about half a minute they run together into piles or rouleaux, which arrange themselves in small-meshed networks. But if a drop of blood from a patient with acute rheumatism, or with an inflammation, be similarly examined, piles of red corpuscles instantly form, and are clustered into masses, leaving a network with wide interspaces. This appearance of itself, however, is not a sure sign of inflammation. It may be observed in the blood of the chlorotic female as well as in the pregnant one; in those also in whom a plethoric condition as regards the blood exists; in persons in health whose circulation has been much accelerated, as by violent exercise; and it appears to be the natural state of the blood of horses. It is a phenomenon resulting from an increased tendency to aggregation of the blood-corpuscles, and gives a granular appearance to a thin layer of blood when viewed with the naked eye. When blood is drawn off in quantity, the phenomenon is associated with the formation of what is termed the “buffy coat,” as the clustered blood-corpuscles, rapidly sinking, subside to some distance below the surface before the fibrine and the white corpuscles begin to coagulate. The connection is now well established between the yield of fibrine, as expressed by a buffy coat, and the existence of acute inflammation. This buffy coat has been found greatly increased in rheumatic fever, pneumonia, pleurisy, bronchitis, peritonitis, quinsy, erysipelas; and the buffy coat over the contents of the bleeding-basin was wont to be an ordinary element in diagnosis, and a justification for the blood-letting (SIMON). However indefinite and uncertain the changes may be, as observed upon a small portion of the blood, it cannot be doubted that the blood stagnant or retarded in an inflamed part undergoes important alterations; and by a constant succession of such changes the whole fluid may come at length to be materially altered, as indicated by the general effects and constitutional disturbance, extending throughout the nervous and the vascular system, and which may ensue in the train of an inflammation of purely local origin.

There are many special points or questions deserving of attention regarding the theory of the inflammatory process; but it is also obvious that, in a text-book such as this, any mere analysis of speculative doctrines ought not to take up much space. The following statement will therefore merely embrace as much as possible of those topics of special interest which a more extended and accurate physiological knowledge of the process of inflammation have shown to be the proper objects of more extended inquiry, and which have been especially the subject of

elucidation since 1866, by the experimental observations of Cohnheim, Stricker, Burdon Sanderson, and others. In the first place, as to the primary seat of the inflammatory process, there can be little doubt, from the phenomena already described, as well as from the results of dissection, which show the progress and effects of the process, and from recent experimental researches, that the vital morbid process known as "inflammation" is connected with the minute capillaries, and the most minute elements of tissues which they nourish. Questions relative to the theory of the process are therefore found to be intimately connected with the histological and physiological relations of these parts; especially as to the structure of the capillaries, the movements through their walls of blood-corpuscles, and the alterations in the nutrition of the inflamed tissue, all differing according to the structure of the part. During the earliest period of the process—the period of increment, or of incubation, as it has been termed—it appears to be the inherent properties of the minute component elements of tissues which first undergo a change; and, combined with the reflex actions of the nervous system, seem to maintain, to promote, or to increase the activity of the subsequent stages. The simplest effects upon the minute elements of tissue, and upon the blood-vessels, are seen to follow the application of the mildest or slightest physical or chemical agents, but which, operating powerfully, are also capable of extinguishing altogether the life of these elements of tissue. When the action induced is mild and gentle, the tissues become incapable of performing their wonted functions; and, provided the mechanical or chemical agency has not been too severe, the impairment of function may subside, and the tissues will return to their normal state of functional activity. This is "Resolution" of the inflammation. The minute arteries have been shown by the histologist to possess in abundance the structural elements of the non-striated contractile tissue, and in this respect they closely resemble the constitution of the muscular fibre of the intestine. Accordingly, the contractions they undergo have been considered as analogous to *spasms* (as Cullen first suggested); while the succeeding dilatation may be of the nature of *relaxation*, and ultimately of *paralysis*. This paralysed state is shown from the fact that the same vessels now dilated will not contract upon a re-application of the same stimulus which before made them contract. If the stimulus is made with a needle upon the vessels in the transparent parts of an animal, the needle may be repeatedly drawn over such dilated vessels, and no contraction will follow; but with a stronger stimulus, such as that of heat, they may be made to contract again, and even close; and this state of contraction may persist for a whole day, before the vessels again open up and permit the blood to flow (PAGET). On the other hand, the true capillaries seem totally destitute of any special structure known to be contractile. They merely consist of a delicate, yielding, homogeneous, protoplasm, beset with occasional nuclei, and, like protoplasm, having the power of developing processes or outgrowths. A film of collodion is not more homogeneous nor more continuous than the membrane of a capillary (VIRCHOW). Whereas the minute arteries possess distinct coats, one of them consisting of a single layer of muscular (or contractile) fibre cells, wound spirally round the internal membrane of the blood-vessel, so as to encircle it from one and

a half to two and a half times. The arteries, to their smallest branches, are sometimes contracted to absolute closure, and at other times are widely dilated; whereas the capillaries are never entirely closed, nor do they present any variations in diameter which are not due to the elasticity or yielding permeability of their parietes, (LISTER, 1. c.)

But the most interesting point in the whole process of inflammation is, perhaps, that which embraces an inquiry into the cause of the "stasis," or stoppage of the blood, the exudation of the *liquor sanguinis*, and the permeation of blood-corpuscles through the yielding protoplasmic walls of the capillaries. This is a point which the observations of Professor Lister have so very beautifully illustrated; while the explanations of other eminent pathologists and experimentalists serve to present the subject in a variety of aspects to the mind, which cannot fail to be both interesting and practically instructive. Henle, Simon, Bennett, Williams, Addison, Rokitsky, Paget, Stricker, Cohnheim, Thin, and Bastian, have all thus helped to elucidate the process by various theories. One of the most recent exponents (by experiments) of inflammation—Dr. Thin—rejects the theory by which a cell in inflamed tissue is supposed to undergo changes which are believed to be peculiar to young cells; and considers the conclusions unwarranted that have been drawn from the division of the nucleus in support of such views. This swelling of the cell, and the division of the nucleus, he believes to be invariably the forerunners of cell-death, and never of cell-growth; and that a pus-cell is invariably a colourless blood-corpuscle which has lost its vitality. (*Ed. Med. Journ.*, Ap., 1876.) On the other hand, the most conspicuous opponent to Cohnheim's theory, that the origin of pus-cells is to be sought for in colourless blood-corpuscles, is Professor Stricker. He and his pupils in the Vienna school show that in an inflamed tissue there is a rapid formation of cells from those pre-existing in the part, in circumstances and under forms that exclude the possibility of the new cells being white blood-corpuscles.

Whatever explanation may be given or accepted as to how the phenomena of inflammation in a part are brought about, our views regarding the essential nature of the process have been hitherto modified according as this complex morbid state has been studied by its effects as seen in the living body rather than on the dead.

While the late Dr. Hughes Bennett regarded an exudation from the blood-vessels as the necessary constituent of inflammation, Alison and Virchow, on the other hand, recognise the morphological changes of the living tissues, such as have been described in inflammation, as betraying merely a *tendency* in a part to such a local change as exudation amongst its structure. That *local tendency* may be so slight that hardly any difference can be appreciated between the healthy changes attendant on normal nutrition, and those changes between the blood and the minute tissues which are of such a kind that a morbid change (inflammation) is established in the elementary components of the tissues themselves, without any appreciable exudation having taken place either amongst the interstices or upon the free surfaces of membranes. To such a condition Virchow gives the name of *parenchymatous inflammation*, meaning thereby that it is a process established locally between the capillaries, the blood, and the component elements of tissue, and expressed by a tendency merely to the

effusion from the blood-vessels of such plastic material as may eventually take place.

Inflammation may thus exist as a local morbid process, characterised by an abnormal condition of the nutritive changes between the capillaries, the blood, and the component elements of a texture, without any appreciable interstitial exudation. Such an abnormal condition will, under proper regimen and proper remedies, in a case of simple inflammation, seen from the first, completely subside, no interstitial exudation ever taking place. Examples of this simple form of inflammation have been fully illustrated by Goodsir and Redfern in this country, by their demonstrations of what takes place within the large cells of cartilage. The cells become larger, the number of nuclei increases, and some or all of them may undergo fatty metamorphosis under the influence of this, the simplest form of inflammation; which is only manifested by this abnormal nutritive process between the blood and the cells. These changes *within* the cell-elements of tissue are described by Virchow as a "*cloudy swelling*" (*trübe Schwellung*, Fig. 4) of the parts, and are seen, for example, in the cells of the uriniferous tubes, and those of the mucous membrane in the state of catarrh previously described. In this abnormal nutritive process, however, there is a constant tendency to the interstitial exudation of a hyaline material, which may become fibrous or filamentous, and ultimately soft and gelatinous. Virchow, Weber of Bonn, His, Cornil, and Ranvier have demonstrated similar changes in the cells of the cornea, cartilage, and mesentery.

Thus the minute and penetrating observations of Virchow have given a more comprehensive meaning to the process of inflammatory exudation than it has hitherto, in this country, been understood to signify; and such alterations as he and others have described in the elements of the tissues of an inflamed part have been in a great measure overlooked, except by Dr. Alison and Mr. Simon. The latter especially states that the irritation of the inflammatory process is independent of the nervous influence, *but is a direct change operated by the living molecular structure of the part on the blood which traverses it, or on the vessels which convey that blood.* Dr. Alison, also, long ago recognised the *tendency* to interstitial exudation as attending such vital changes in the constituent elements of a part, which entitled it to be considered inflamed. The accurate observations of Virchow, Goodsir, and Redfern have shown that such primitive changes do take place before those more palpable phenomena occur which constitute the excessive exudation as described by Bennett—namely, the exudation of decolorised lymph into the interstices between the constituent elements of a texture. Both sets of phenomena alike show that *inflammation is only one of the various shades of deviation from the normal process of nutrition—a diseased*

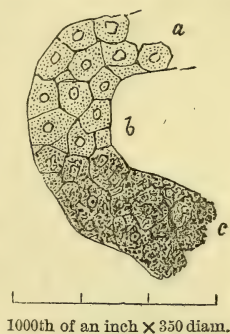


Fig. 4.*

* Convoluted urinary tubule from the cortex of a kidney in Bright's disease (after VIRCHOW). *a.* Tolerably normal epithelium; *b.* state of "*cloudy swelling*;" *c.* commencing fatty degeneration and disintegration.

action tending to a local lesion (*British and Foreign Medico-Chirurgical Review*, January, 1854). That the irritation of inflammation is in some measure independent of the nerves, the following interesting experiment, related and performed by Mr. Simon, may be quoted in proof:—

“A patient had complete anæsthesia of the fifth nerve, dependent (as a *post-mortem* examination subsequently showed) on its organic disease; the conjunctiva, as well as the integument of the face, was utterly insensible; not only was the function of the nerve destroyed, but those reflective nutritive changes, of which I have already spoken, had taken place, and had exhausted themselves; showing that the nerve was spoiled for participation in the acts of nutrition (whatever they may be), no less than for its more obvious uses as a medium of conscious sensation; the cornea had undergone ulceration, and had healed again. The following experiment was carefully made:—The lids being held open, a single granule of cayenne pepper was laid upon the insensible conjunctiva; in a few moments it had become the centre of a very distinct circle of increased vascularity, the redness of which slowly became more and more distinct as long as the stimulus was suffered to remain, so that, on its removal, there was a very evident circumscribed erythema on the surface of the membrane. I consider myself justified in believing that this change occurred without any nervous excitement; not only because the history of the case would lead me to consider the fifth as annihilated; not only because the experiment was totally unattended with sensation; but likewise because there was the very remarkable absence of that sympathetic phenomenon which the faintest remnant of nervous excitability would have produced—namely, there was not the slightest trace of lachrymation” (*Lectures on General Pathology*, p. 76).

Stricker also concluded that structural changes in inflammation of the cornea of the frog are owing to the stimulation of its elements by the liquid exuded from the blood-vessels, and are quite independent of nervous influence. He excised the cornea of a frog, and inserted it beneath the *membrana nictitans* of the opposite eye, in which inflammation had been previously induced. The transplanted cornea, when removed after twenty-four hours, exhibited all the inflammatory changes observed in the unexcised cornea of the opposite eye. Further evidence might be submitted from the papers of Mr. Joseph Lister to the Royal Society, already referred to.

The alteration of nutrition in an inflamed part is thus characterised by an increase or exaltation of the nutritive functions of the cell elements of the tissues involved. Those elements which normally exhibit the movements of living growth (as distinguished from the growth of a crystal), increase in activity as the amœboid cells of connective tissue and the cornea. The increased activity is also shown by variation in the form of cells, which usually remain of uniform shape, sending out abnormal processes of growth, followed by enlargement, multiplication of nuclei, and the formation of new cells, by the division of protoplasm and rupture of the old. Thus the first change is one of cell proliferation. This increased activity of nutrition and growth in inflammation varies greatly in the different tissues, and in the various elements of the same texture. The epithelial elements of the skin and mucous membrane become excessively

active in the abnormal nutrition of inflammation, the slightest irritation being sufficient to produce rapid cell proliferation to the extent of a catarrh. Common connective tissue, cartilage, and bone, whose elements do not exhibit any tendency to multiplication, are slow to cell proliferation. Their cell elements are more stable, and multiply with difficulty; but under the abnormal nutrition of inflammation they become enlarged, and undergo some alteration in form; while new cells are formed in their interior, by enlargement and subdivision of their nuclei and protoplasm (Figs. 5 and 6); while the matrix softens and liquefies, as was shown by Dr. Redfern in his valuable experiments on animal cartilage, published in 1850. In all cases during the abnormal nutrition of inflammation, the rapidity and extent of proliferation are in direct proportion to the intensity of the inflammation.

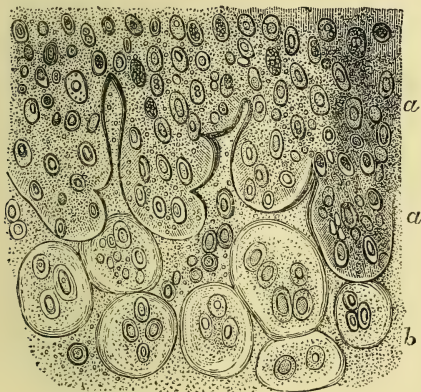


Fig. 5.*

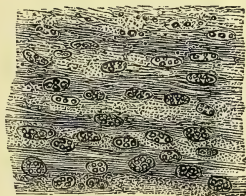


Fig. 6.†

Such being the essential nature of inflammation, it is easy to understand how reasonable is that doctrine which teaches "that the process of inflammation is susceptible, at all times and in all countries, of very great variety as to extent or intensity, and especially as to the constitutional affection associated with it or consequent upon it."

Products, Effects, or Events of Inflammation.—Care must be taken not to put the *products* of inflammation in place of the *symptoms* of inflammation. When the local impairment of function of the minute elements of tissue in process of inflammation is *confined to a small space*, or *is carried on upon a minute scale*, or *rapidly abates*, the inflammation is said to *terminate by resolution* as a general principle; that is, the abnormal action ceases, hyperæmia subsides, interstitial exudation ceases, the tendency to further impairment of function is subdued and passes off, and the part is left apparently as it was before by the absorption of the fattily degenerate products. If, however, interstitial exudation has taken place, and *resolution* is to be effected, the return of the part to health may be

* Section of the costal cartilage of the dog, showing, *a*, the discharge of the corpuscles of the cells, resulting from the passage of a seton thirty-four days before death; *b*, cells becoming spherical, and undergoing endogenous proliferation. (After REDFERN.)

† Section showing the first appearance of fibres in the hyaline substance between the changed and granular cells of the perichondrial layers of the costal cartilage of the dog, after the passage of a seton forty days before death. (After REDFERN.)

followed for some time by some impairment of its structure and function. *After the process has thus gone a certain length*, an increased local growth of cells, and their liquefaction or reduction to a state capable of absorption (what Dr. Addison calls *cell-therapeutics*), are essential to the restoration of the part. Before the process has attained such a length, however, *resolution* may be simply effected by a gradual return of all the parts to a natural state. A mere retracing of the steps by which the natural actions had been departed from sufficiently describes this process of *resolution*, which has been closely watched by Sir James Paget. He has seen, in those cases where impairment of function and actual lesion had taken place, that fragments of fibrine, washed from the blood in the vessels of the injured parts, were borne along and floated into distant vessels. The observations of the late Dr. Kirkes, also, leave no doubt that similar changes may occur in warm-blooded animals, and may be the source of great evils,—may be, indeed, productive of some of those constitutional effects yet to be noticed, by carrying the materials of diseased or degenerate blood from a diseased organ to one that was previously healthy. When the disappearance of the inflammation is unusually sudden and rapid, the event is technically called “*delitescence*,” and if at the same time the symptoms of inflammation appear at another part not anatomically connected with the part first diseased, the event is called a “*metastasis*.” When the process does not confine itself to the simple expression of altered nutritive changes between the constituent tissues of a part and the blood; but when the exudation amongst the interstices of texture continues, and does not subside, as already explained,—namely, by *resolution*,—then it is that, (1.) Such a material is separated from the blood as will become a medium, or *nidus-substance*, in which many changes connected with the growth of new particles, granules, or cell-forms will take place, the phenomena of which have been so well described by Bennett, Gluge, Paget, Virchow, Beale, John Simon, Cohnheim and Stricker, Pagenstecher, Reindfleisch, and others; and, (2.) Coincident with this exudation, and the changes which it undergoes, the tissue of the part itself sustains serious alterations. For in all such inflammations, especially of the more vascular parts, when there is increased exudation from the blood-vessels, there is a great deterioration of the surrounding elements of tissue. The texture is rendered soft and easily torn, and by such changes of cohesion the elasticity of parts (a circumstance often of very primary importance) becomes greatly altered and impaired. These changes, therefore, Sir James Paget happily described as consisting of,—(1.) *Productive* effects—that is, effects resulting from the growth of new particles, granules, or cell-forms, from pre-existing germinal elements of tissue, and which are susceptible of further development into a permanent tissue, and also of such impairment of nutrition as will bring about their *degeneration*; (2.) *Destructive* effects, such as *softening, degeneration, absorption, ulceration, and death* of tissue.

Productive Effects of Inflammation—Inflammatory Effusions or Exudations.—These consist of,—1. Serum, or *liquor sanguinis*; 2. Blood; 3. Fibrine; and, 4. Mucine.

1. *Serous Effusions*, or effusion of *liquor sanguinis*.—The effusion of pure serum is said to be very rare. In inflammation of a serous membrane, as

the pleura, the fluid effused is not only greater in quantity than natural, but is also greatly altered in quality. In health the serous secretions are little more than pure aqueous vapour, with a trifling addition of saline matters; but when exudation of *liquor sanguinis* occurs in an inflamed part, associated with the passage of the blood-corpuscles through the walls of the vessels, it contains a considerable quantity of albumen and of fibrine, the amount of both of which increases with the intensity of the inflammation, and with an excess of phosphates, carbonates, and chlorides. The quantity of serum effused varies, according to the part affected, from perhaps a portion of an ounce to a few pints, or even more than a gallon.

This product of inflammation, largely mixed with fibrine, may be seen in the fluid contained in blisters raised by counter-irritants in a healthy person; also in the fluid of *peritonitis*, of *pleurisy*, and of *pericarditis*. Such also is the fluid that fills the early vesicles of *herpes* and *eczema*, and other cutaneous eruptions; as well as in the fluid which surrounds an acute, deep inflammation beneath the skin. The fluid of a common *hydrocele* is another example of serous effusion. The phenomena associated with the production of such a secretion may be often seen surrounding a phlegmon or boil of large size. While the centre or *core* of the boil is hard, it is surrounded by textures into which the *effusion* that has taken place is *serous*. Such serum is seen to a great extent in *pelvic cellulitis* in its first stage (SIMPSON, in the *Medical Times and Gazette* for 1859, p. 27, July 9). In such cases the fluid fills the areolar tissue which immediately surrounds the inflamed parts; and when the finger is pressed firmly on the part, the fluid is displaced into the adjoining areolar spaces, which yield to receive it. When the finger is withdrawn, the fluid does not immediately return, but an impression is left in the shape of a pit. The part which is the seat of *serous* effusion is then said to *pit on pressure*, or to be *cedematous*.

The fibrine of inflammatory serous effusions remains in solution for weeks or months within the body during life, but will coagulate readily when withdrawn. This delay of the fluid to coagulate within the body is a propitious event. So long as it is liquid, absorption may still ensue without its undergoing any ulterior change when the inflammation subsides. This subsidence of the inflammation, however, is necessary; for, it is known, that so long as inflammation continues, there is impairment of function, and absorption, especially, is in abeyance.

One constant characteristic of the *productive* effects of inflammation is, that growing material is always developed from pre-existing germinal elements of or amongst the tissue involved, and in no case generated spontaneously in the effused liquid. The effusion of *liquor sanguinis* that results from mere mechanical obstruction to the flow of blood is very different from the albuminous and fibrinous effusion of an inflammation. In the former case the fluid effused from the blood is merely the serous part, as the fluids of *anasarca* and *ascites*, and will not coagulate. Such fluids, as a rule, neither present fibrine, nor are any granules, particles, or cell-forms developed in them from the elements of surrounding tissue. Fibrine can only be made to exude upon a surface or part in a state of irritation or inflammation. Such a local change is sufficient to cause the exudation of fibrine, independently of obstruction to the circulation; and

the cause of the greatest differences in the nature of exudations is to be found in the special constitution of the irritated parts (VIRCHOW). In some effusions there is a great yield of fibrine—*e.g.*, the exudation of diphtheria, so also in the effusions of arthritis and of muscular rheumatism.

It has been clearly shown (SIMON, LEHMANN, BEALE) that there are two essential characteristics of inflammatory effusion:—(1.) It tends to contain certain ingredients in larger proportion than that in which they exist in the blood—excess of chloride of sodium and of phosphates and albumen; (2.) Organic forms find in it a suitable place for growth. The large number of cell structures it contains consist, for the most part, of migrated blood-corpuscles, the remainder being derived from the superabundant growth (proliferation) of the elements of tissue involved. The cause of this increased nutritive activity of the cell-elements of the part is probably to be found in the stimulation of the cells by the *liquor sanguinis*, abnormally abounding in albumen, fibrine, phosphates, and carbonates, which has exuded amongst those tissue elements from the blood-vessels.

The site of effusion resulting from inflammation is important as sometimes constituting the chief element of danger—a danger sometimes immediate, from the mechanism of the parts affected. A large quantity of fluid is often poured out in a very short time. The cavity of the pleura may fill in a few hours, and the lung may be compressed by it to a half or third of its bulk; and if both pleural cavities become thus affected, constituting *double pleurisy*, the patient must die from suffocation, if not at once relieved by allowing free vent to the fluid. This operation is called *tapping* the chest, or, technically, "*paracentesis thoracis*." Serous effusion into the areolar submucous tissue of the glottis may also produce almost immediate death from suffocation, unless the cavity of the larynx is immediately opened to admit the air to the lungs. The quantity of effusion also varies with the structure of the tissue inflamed. The effusion is largest from mucous surfaces and in serous cavities, and least in non-vascular tissues, such as cartilage and the cornea, where the exudation occurs to a small extent only from neighbouring vessels. In dense visceral organs, like the liver and kidney, the compactness of their structure prevents any large amount of effusion. In the kidney it may pass off by the uriniferous tubes, giving rise to albuminous urine.

2. *Blood Effusions, or Extravasations*, chiefly occur from rupture of the new vessels developed in the newly-formed material, which has just become vascular (ROKITANSKY); but Cohnheim has also shown that during the process of inflammation, at the same time that the white corpuscles permeate the veins, red corpuscles make their way through the capillaries. In the ordinary course of internal inflammations, extravasations of blood are rare, and betoken an unfavourable state of the constitution generally, such as exists in typhus fever, scurvy, purpura, or syphilis. The *post-mortem* evidence of such extravasations is the presence of a coloured cicatrix at the spot of rupture and effusion; and the colour is found to be due to the presence of hæmatoidine, generally in the form of a mass of aggregate crystals, composed of minute rhombic columns, which may be considered as the regular typical ultimate form into which hæmatine is converted in any part of the body where considerable masses of extra-

vasated blood continue to lie for any length of time—*e. g.*, apoplectic clots, and coagula in the Graëfian vesicle of the ovum after menstruation (VIRCHOW). We must not confound with hæmorrhages the cases in which the inflammatory products are merely blood-stained—*i.e.*, have acquired a more or less deep tinge of blood, through the oozing of some of its dissolved colouring matter (PAGET). The natural colour of inflammatory new formations is greyish or yellowish-white, and even when they have become vascular their opacity in the recent state prevents their having any uniform tint of redness visible to the naked eye. When inflammatory products present the tinge of redness, it is either because of hæmorrhage into them, or because they have imbibed the dissolved colouring matter of the blood; and when this imbibition happens during life, or soon after death, it is important, as implying a cachectic, ill-maintained condition of the blood, in which condition the colouring matter of the corpuscles becomes unnaturally soluble.

3. *Inflammatory Lymph or Fibrine*.—This product is so named to distinguish it from the lymph in the lymphatic vessels, with which it is probably not identical. It is a characteristic primary product of the inflammatory process, and was called originally "*lymph*," or "*coagulable lymph*;" and more recently it has been called "*exudation*," "*fibrinous*" or "*inflammatory exudation*." "It is at first probably always a pellucid liquid exudation which passes through the blood-vessels"—"sweats through them," as Simon hath it,—"and especially through the capillaries of the inflamed part. Its most characteristic general properties are, that it may become a *nidus-substance*, capable of taking some share, or of assisting in promoting the growth of new elements like the natural connective tissue of the body." (PAGET). But the nature of the products of this development in the "*inflammatory lymph*" varies much according to the part and the state of the constitution. Accordingly Bennett attempts to distinguish the products of *simple exudation* from those of *tuberculous* and *cancerous* exudations. But the typical elementary forms which may grow amongst inflammatory lymph vary according to a much greater variety of circumstances, which tend to modify the morbid appearances due to the inflammatory process, or impart to it a particular *tendency* in respect to the nature of the productive material, especially as to—(1.) The nature of the tissue in which the inflammation takes place. (2.) The period at which the product is examined after exudation has taken place and growth commenced. (3.) The state of the blood, and the nature of the general disease-process, or *constitutional* morbid state, which may be associated with the inflammation. (4.) The amount of vascularity which the affected part retains. (5.) The amount of the local exudation, and the extent of healthy tissue implicated. (6.) The suddenness of the phenomena of exudation and of growth. (7.) The persistence and extension of the inflammatory state in its vicinity. (8.) The amount of fluidity, serum, serous effusion, blood, or mucus, associated with the *inflammatory lymph*. These circumstances severally, or more or less collectively, influence the different stages of progression or degeneration, by which the local lesion may ultimately terminate in *resolution*, in *permanent organic mischief*, in *death* of the parts involved, or in *death of the patient*.

There are several typical forms, especially found growing amongst the

"inflammatory lymph," in the growth of which the material of the lymph may take some share—namely, the granular, molecular, or fibrillated development of *fibrinous* products and *corpuscular* forms. Rokitsansky describes these typical forms by the terms "*fibrinous*" and "*croupous*," and Dr. C. J. B. Williams by the names of "*plastic*" and "*aplastic*." Examples of each variety may illustrate the application of the terms. To the *fibrinous* or *plastic* variety belong the serous effusions already referred to, and perhaps also the granular, molecular, or fibrillated growths. The *corpuscular*, *croupous*, or *aplastic* forms of lymph are represented by those growths which never become consolidated, as in the early-formed contents of vesicles in *vaccinia* and *herpes*; in the *fluid of blisters* raised in cachectic patients; in some instances of *pneumonia*; and in some forms of inflammation of *serous membranes*. In by far the larger number of inflammatory products these typical forms are mixed in various proportions; and the larger the proportion of corpuscles in new growth, the greater is the probability of suppuration, or of some other degenerative process, and the more tardy is any process of development into tissue, such as that which constitutes adhesions, indurations, and the like. In other words, the preponderance of granules, molecules, and fibrillated material in the new growth, is generally characteristic of "adhesive or lymphic inflammations;" the preponderance of corpuscles, or their sole existence in a liquid medium, is a general feature of "suppurative inflammation." The hardness of inflamed parts is due to the former of these typical forms of inflammation, and is exemplified in the case of a *phlegmon* or *boil* before it suppurates; as also in a lung in a state of *hepatisation*, when its textures are enclosed by a lymph, "just as the stones of a wall are by the cement;" also in the hardening of a chancre. On the surfaces of inflamed membranes the new fibrinous growth forms a layer of a membranous firmness or consistence, to which the name of *false*, *adventitious*, or *pseudo-membrane*, has been given. By this new growth the naturally opposed surfaces of parts which are inflamed are apt to adhere. This is commonly seen to be the case between such serous surfaces as the *pleura*, the *pericardium*, the *peritoneum*, or the *edges of a wound*. The inflammation associated with this organisation is sometimes called "*adhesive inflammation*;" and Dr. John Thomson ascertained that this growth and organisation might be effected between the surfaces of wounds in less than *four* hours after they were inflicted. In the chronic course of an inflammation, the result of the process is to establish what has been termed "*fibroid substitution*," in which the inflamed part, instead of liquefying, becomes solidified from the gradual formation of new fibrous tissue, which takes the place of the natural structure; new vessels are gradually formed in it. This process takes place in cirrhosis or interstitial inflammations of the liver, lungs, and kidneys, as well as during the healing of wounds by the *first intention*. Its future tendency is towards contraction, and the formation of a cicatrix, characterised by puckering and depression over the seat of the interstitial inflammation—cicatricial-like contraction.

The "corpuscular" typical form of new growth as a result of inflammation may generally be seen on the surfaces of mucous membranes. It has little tendency to cohere, but grows in films, gelatinous masses, shreds, patches, or delicate casts of the surface upon which it was formed. The

new growths in chronic catarrh of the intestines are an example; so are the membranes sometimes passed from the cavity of the uterus, and called *dysmenorrhœal membranes*. In the "adhesive" form of inflammation the new growth of granules or of molecules may ultimately assume the form of *fibrous tissue* interstitial to the textural elements inflamed. Examples of this organisation are seen in the laminated and nodular thickening of the capsules of the spleen, the thickening and induration of the periosteum, or the capsule of the hip-joint in chronic arthritis; and by virtue of the peculiar tendency to contraction which fibrinous products possess, the contractions of parts are to be explained which have been the seat of such a form of inflammation.

There are instances also in which the new growth assumes the form of adipose tissue, elastic tissue, and epithelium (KIRKES, VIRCHOW, PAGET); and bone is a very frequent ulterior change which it assumes, especially when the new growth is interstitial to fibrous tissue; but these ulterior events only happen *after* the inflammatory process has ceased in the part. So long as the inflammatory phenomena continue, the tendency of the new growth is to assume the *corpuscular* form, such as pus, rather than the more adhesive forms of organisation. Hence rapid organisation accompanies, as a general rule, a minor degree of action; and by depressing the action of a part, we tend to prevent the threatened occurrence of suppuration.

The existence of the inflammatory state, associated with an interstitial exudation, influences the simplest *corpuscular* forms of organisation. *Lymph-cells*, or *simple primordial* forms, occur, which are represented by the corpuscles of chyle, lymph, the white corpuscles of the blood, and by those of granulations on the surface of a wound. These simple cells become developed amongst the lymph while it is still fluid, transparent, and apparently homogeneous. The first discernible organic form in the lymph of *herpes*, for example, is that of a mass of soft, colourless, or greyish-white corpuscles, about $\frac{1}{2500}$ or $\frac{1}{3000}$ of an inch in diameter, round or oval, pellucid, but appearing, as if through irregularities of its surface, dimly nebulous or wrinkled. It does not look granular, nor is it formed by an aggregation of granules; nor, in its earliest state, can any cell-wall be clearly demonstrated, or any nucleus on adding water. In a few hours, however, a pellucid membrane appears to have grown over its surface, permeable by water, which raises up part of it like a clear vesicle, while the contained mass retreats or subsides to the lower part of the enclosure, and appears more nebulous or grumous than before. A nucleus ultimately forms, and can be distinguished in this mass (PAGET). From these primordial cell-forms in the lymph either the adhesive or more corpuscular forms of organisation may proceed; and all the various forms of corpuscles described by authors as plastic cells, fibre cells, caudate cells, or fibro-plastic cells, and some forms of filaments, are developed from the germinal matter of the surrounding tissue by continuous development. Also, from the development of the primordial corpuscles, or white blood cells, all those elementary forms proceed which are known as "*pus-corpuscles*," "*granule-cells*," "*compound granule-masses or cells*," "*inflammatory globules*," and much of the *molecular debris-like matter* that makes inflammatory effusions turbid.

The modes of growth as well as of degeneration are well described by Sir James Paget, from whose work on Surgical Pathology the following examples and illustrations are chiefly taken :—(1.) The lymph may simply wither or waste, as may be noticed in the vegetations on the valves of the heart or large arteries when they become yellow, stiff, horny, elastic, and nearly transparent, or in the lymph deposited over a compressed lung, associated with empyema or hydrothorax. (2.) The fibrine of lymph may undergo changes similar to what is known as fatty degeneration,—changes similar to those which occur in the *primordial lymph-cell* when it is transformed into pus. The two changes generally go on together. To the former change—namely, the fatty-like degeneration of the fibrine—Sir James Paget has given the name of “*liquefactive degeneration* :”—the solid fibrine of inflammatory lymph that becomes again liquid when suppuration takes place—as may be observed in a hard mass of inflamed texture when it becomes soft. This is a degeneration which brings the new growth into a state favourable for its absorption, or to the *resolution* of an inflammation. Examples of such an absorption may be seen in *rheumatic iritis*, and the observations of the late Dr. Kirkes on the rarity of adhesions of the pericardium in comparison with the frequency of pericarditis may also be explained in this way. (3.) Melanic degeneration of lymph and new growths is not unfrequent, as in *peritonitis*. Concurrent with these degenerations of the lymph-granules and molecules are the degenerations of the *corpuscular elements*. (1.) They may wither, as in the dried-up pus of chronic abscesses. (2.) The fatty degeneration of cells is said to be shown in their transition to the *granule-cell*, known also as the *inflammatory globule* of Gluge, or the *exudation-corpuscle* of Bennett—a change which, I believe, is favourable to this re-absorption. The observations of Reinhardt, Dr. Andrew Clark, Paget, and Gairdner, place it beyond a doubt, that compound granular cells may result from a fatty degeneration of the textural cells of a part; just as calcareous or pigmental degenerations occur, and which are common to primordial cells. While there can be no doubt, therefore, that fatty degeneration of lymph or textural elements may lead to the appearance of compound granular cells, that process can scarcely be called degeneration which is associated with development, growth, and complete absorption, by which the indurated and confused parts of an inflammation, such as the solidified portions of a lung in pneumonia, are ultimately cleared up. Degenerate products are usually persistent, but the compound granule-cell is not. It seems, to me, to have an important function to perform in the removal of fluid, effete, or softened exudations, after which it too liquefies and disappears.

All these productive effects of inflammation, in the form of effusions or exudations, contribute, with the greater abundance of blood, to the *swelling* which attends inflammation of a part. The effusion may collect within a shut-sac, as in synovial and serous inflammations, or it infiltrates the solid organs. In mucous membranes the effusion is represented by the altered secretion which, as a catarrhal exudation, flows from its surface. The swelling in such cases is represented by the amplification of the epithelial layer; and hence the interference of respiration which belongs to the beginning of acute bronchitis, the blocking up of the kidney tubes with modified cell-growth in scarlet fever, and the diminished stream of urine

during the severity of gonorrhœa. It is these products of effusion and exudation which render inflamed parts so extremely juicy, so œdematous, that a cut or puncture into them during life allows the fluid to drain away long after hæmorrhage has ceased. All these effusions of inflammation differ from the mere exudation of serum from *liquor sanguinis* in the nutrition of texture. "Their specific gravity is high. Their proportion of albumen is great; and when they are not purulent, they probably give a fibrinous coagulum" (SIMON).

Local and General Symptoms of Inflammation.—Redness, or at least increased afflux of blood, swelling, increased textural productivity, pain, throbbing, increased sensibility, disorder of function, arrest and change of secretion, are the phenomena which are associated with this local morbid state, or with the textures in its immediate vicinity. Under all circumstances the inflammatory process involves a local production of heat accompanying the increased vital effort and the increased textural changes of inflammation. This has been recently proved to demonstration by the ingenious experiments of Mr. Simon and his colleague, Dr. Edmund Montgomery (*A System of Surgery*, second edition, edited by T. Holmes, M.A., Vol. I., p. 20). If the local process of inflammation, however, is carried on upon a minute scale, or in certain tissues, one or other or more of these symptoms may be absent; if, on the other hand, the local process proceeds on an extensive scale, and involves important and delicate textures of vital importance, then we have much more unequivocal expression given, not only to local symptoms of redness, swelling, heat, and pain, but to complex, morbid processes affecting the constitution generally. Of these the chief are:—

I. Inflammatory Fever.—Of the *constitutional symptoms*, as they are termed, the most prominent are those which indicate "*inflammatory fever, symptomatic fever, or sympathetic fever.*" These constitutional symptoms are of the greatest importance, not only by indicating the nature of the disease, as when the inflammation is connected with an internal organ removed from sight and touch; but they are highly important as a guide to treatment. The premonitory symptoms of coldness and shivering are usually very decided, but not of long duration, and rigors more frequently attend the commencement of spontaneous inflammation than of inflammation caused by external injury. They are succeeded by a stage of re-action. This fever is pre-eminently one of strong reaction and vascular excitement, and these characteristics may be said to constitute its *type*, the general symptoms of which may be summarised according to systems—thus:—(1.) *The Nervous System.*—There are aching, dull pains in the loins and limbs, restlessness, and much discomfort. The will and the power of exertion are diminished. Anxiety or foreboding of evil is felt, and expressed upon the countenance. The head is generally hot, the face flushed, the eyes suffused, the skin hot and dry. Special sensation is at first exalted, but afterwards the intellectual functions become more and more disturbed. Ultimately delirium is established, and coma may ensue. (2.) *The Vascular System.*—The pulse ranges from 80 to 130, or more, and the heart's action is proportionally rapid. The pulse is hard, rolling like a cord below the finger, and yielding but little to its pressure; or an irregularity of movement in the artery may exist, and thus a thrill or jar

is imparted to the finger. There is increased fulness, as if the vessel were itself enlarged, and held a larger quantity of blood at each impulse; the heart is acting not only more rapidly, but more powerfully than in health, and the circulation is truly accelerated. Frequency, hardness, and thrilling, are seldom, if ever, absent; but fulness may be wanting, and the pulse may be small instead of full. This latter modification is chiefly observed during serious inflammatory action, affecting important internal organs situated in the abdominal region. Hence it is sometimes termed the *abdominal pulse*; the artery resembling a hard, thrilling thread, rather than a cord. This pulse always exists in connection with great nervous depression and debilitated though rapid cardiac action, to which circumstance its smallness is probably due. In affections of the brain, on the other hand, producing coma, the pulse is commonly slow and full—the suspension of cerebral influence appearing to diminish the rapidity, without affecting the force, of the heart's action. There are idiosyncrasies also to be taken into account. The pulse may be naturally slow or rapid—fifty or ninety; and this must be allowed for, when previous inquiry has satisfied us that the patient is the subject of such peculiarity. (3.) *The Respiratory*.—Respiration is quickened; the breath is felt to be hotter than usual; and an oppression is complained of in the chest. (4.) *The Digestive*.—The tongue may be loaded, white, and moist; or the edges and central tip may be red and dry: the latter is probably the more frequent combination. (5.) *The Secerning*.—The secretions and excretions in general are materially diminished. The bowels are constipated—mainly from want of mucous secretion from their lining membrane; the skin is hot and dry; the mouth is parched; the urine is scanty, high-coloured, generally acid, sparingly aqueous, and holding much saline matter, with comparatively little urea in solution. (6.) *The Nutritive*.—Digestion is interrupted; so is assimilation; as the fever advances, so does emaciation; and strength is more and more prostrate. (MULLER, *Principles of Surgery*, p. 39.)

But the essential fact in the fever of inflammation is, that in proportion as any considerable part of the body becomes acutely inflamed, the patient's blood invariably rises in temperature above 98° Fahr.; and regarding this constitutional febrile state characteristic of inflammation, some important general conclusions, especially insisted on by Dr. Alison and Sir Thomas Watson, may be thus shortly stated:—(1.) There is no fixed relation between the degree or intensity of internal inflammations and the constitutional fever attending them; nor is the fever always proportioned in its degree of violence to either the size or importance of the part inflamed. In some cases where we are sure that we have had inflammation going on under our inspection to extensive effusion of pus, the pulse has been feeble, the skin cool and damp, and the patient exhausted and faint on the slightest exertion; while in others there is high and more inflammatory fever, and in some of these the organ inflamed has been so to no extent, and its function comparatively little affected, but yet the patient has become comatose nearly as in typhus, and died so (ALISON.) Laennec makes an observation of a similar kind, and Sir Thomas Watson observes that the fever may be high and very strongly marked in that common complaint, the *quinsy*, *cynanche tonsillaris*, or *tonsillia*, which can scarcely

ever be said to imply much danger. (2.) The situation, the extent, and the degree of the local inflammation being the same, the fever commonly runs higher in young and in plethoric persons, and in those of sanguine temperament, than under opposite conditions. (3.) Inflammatory fever is modified in its expression, and especially in the characters of the pulse, by the nature of the part which is inflamed. This has been already alluded to in regard to inflammations of the abdomen, where the action of the heart is depressed, and the pulse is changed accordingly, tending to death by asthenia; and also in regard to the brain, when the mode of death tends to be by coma, the pulse being slow, laboured, and full. (4.) The type of the inflammatory fever is very much modified by constitutional circumstances, such as the previous habits of the patient, and whether any zymotic disease is associated with the local inflammation. (5.) The inflammatory fever undergoes a further change of type, (a.) When suppuration takes place; (b.) when it continues long; and, (c.) when mortification or gangrene occurs to a large extent. (6.) The febrile state follows generally the local disease; but, (7.) There is also good reason to believe that the *pyrexial* condition, and the condition of *inflammation* in a part, may be excited in some instances conjointly; or, at all events, their periods of commencement may correspond so closely that it is difficult to conceive that one is the effect of the other. Observations are much wanted as to the exact ranges of temperature, as measured by the thermometer, in cases of inflammatory fever, and so to verify or set aside such general statements (see BILLROTH, in *Year-Book of Syden. Society* for 1861).

When inflammation proceeds to suppuration, a severe paroxysm of shivering is often the first indication of the formation of the pus, and the character of the fever undergoes a great alteration from that just described. The degree of the fever varies greatly; for a most copious formation of pus may take place from a mucous membrane, as that of the bronchi or urethra, and yet the constitution may hardly suffer in any appreciable degree; while a trifling amount of pus from a serous membrane may be associated with a fatal fever. In any case the character of the fever depends in a great measure on the constitution of the patient. If that be good, the fever is attended with a white tongue, with little tendency to become brown, also with much heat, and a full, strong pulse. On the contrary, if the patient's constitution be broken or impaired, the fever is of a low type—*asthenic*, as it is called. The event of *suppuration* is generally marked by a *rigor* of greater or less severity, while the fever hitherto has been *sthenic*. It is the occurrence of the *rigor* in the course of the inflammatory febrile state which gives it prominence and importance. It generally attracts the attention of the patient, and indicates to the physician that *pus* has been produced in the part or organ inflamed. As soon as suppuration is complete, and the abscess ripens, or pus approaches a surface to be discharged, and especially if any important organ is its seat, the fever tends to become *asthenic*, with a brown tongue and a rapid pulse, while the local pain in a great measure subsides. At this period the abscess must open spontaneously, or be opened by art, otherwise the patient may be in danger. The opening of the abscess, though attended with much pain from contraction of the inflamed walls, is usually followed by relief of all the constitutional symptoms; the pulse rises, the

tongue cleans, the appetite returns, and a visible and immediate amendment takes place. If, however, the patient has been exhausted by his sufferings in the earlier stages of the disease, the relief afforded is but transient, the pus degenerates into *sanies*, or is altogether suppressed, the fever changes its type, and the patient sinks, too enfeebled to establish the reparatory process.

II. The Typhoid Fever of Inflammation.—The *type* of fever during inflammation, known by this name, is *asthenic* or *adynamic*. Feeble and more feeble the patient becomes; the pulse sinks; there is great impairment of the heart's action, and tendency to collapse; the features become pinched, shrunk, damp, and ghastly; and the skin is covered with a cold and clammy perspiration. Sometimes these *adynamic* characters may pass into that typhoid state in which nervous symptoms, such as delirium, somnolence, and tremors, prevail. These characters are known as *nervous* or *ataxic*. The tongue becomes dry, black, and tremulous, sordes cover the teeth, and harden on the lips and angles of the mouth. Low muttering delirium, stupor, or coma prevail; tremors affect the voluntary muscles, and the fæces and urine pass unnoticed. This form of fever sets in as a consequence of some untoward or unhealthy tendency of the inflammatory process, such as when mortification of the part occurs. Any cause, however, by which the system becomes extensively vitiated, will bring about this form of fever. It is not necessary that the part should die. Putrescence of the infiltrated exudations in the inflamed part poisons the fluids circulating amongst them, and so, by absorption, may induce the typhoid state. If this happens with an internal organ, the event is generally indicated by a sudden cessation of all pain, at which the patient often appears very happy, and even joyous, while to the experienced physician its sudden cessation is assuredly an evil omen (WATSON). The most important vital functions are deeply impaired by a prolonged existence of this type of fever. It tends to death by a complete sinking of the circulation and diminution and loss of animal heat; or deepening stupor, with oppressed respiration, supervenes; or the patient dies by a combination of both conditions—*asthenia* and *coma*.

These are two distinct types or forms of fever, one or other of which is sure to be associated with inflammation.

Causes of Inflammation.—These have been described by Mr. Simon as:—

(1.) *Determining* or *exciting* causes, arising from several sources, and having different modes of operation.

Such exciting causes of inflammation do not take place without an irritating stimulus—something which irritates the nerves of the tissue which becomes inflamed: *irritation* being the starting-point in every form of inflammation.

The determining or exciting causes of irritation, classified according to the source whence they come, are:—

1. *Acts of mechanical and chemical violence inflicted from without*—*e. g.*, cuts, stabs, bruises, lacerations, abrasions, fractures, dislocations, burns, the action of caustics, or other chemical agents.

2. *Lodgment of matters foreign to the tissue*—*e. g.*, splinters, bullets.

3. *Morbid products of the body itself, and retained excreta*—becoming irritant to parts and cavities containing them; a bladder imperfectly relieved of urine, as in chronic paralysis; retained and decomposing excrement in balls or knobs (*scybalæ*), irritating the mucous membrane of the colon; renal, biliary, and salivary calculi; concretions in *appendix vermiformis*; concretions of blood, as in retro-uterine or pelvic hæmatocele; extravasations into brain, lungs, or muscles; masses of tubercle; dead tissue; a dislocated crystalline lens; a sequestrum of dead bone: all tend by contact, as foreign bodies, to provoke inflammation within a certain radius of their influence.

4. *An altered state of the local nerves*, as when injuries or diseases of the ophthalmic division of the fifth nerve lead to injection and inflammation of the conjunctiva, clouding and ulceration of the cornea, final destruction of the globe of the eye, and sometimes to ulceration of the neighbouring integument. Similarly, lesions of the pneumo-gastric nerve may induce pneumonia.

In explanation of how the nerve influence acts, there are some grounds for belief that certain portions of the nerve cords consist of centrifugal fibres exercising a special “trophic” function; and that it is probable they may be subject to some reflex action, as when a carious tooth produces superficial sloughing of the cheek, or ulceration of the neck (SALTER, in *Guy's Hospital Reports*, Vol. XIII., third series. SIMON).

5. *Abnormal properties of the circulating blood*, such as deficiencies in its constitution, or from its being charged with some specific irritant. The “poor blood” of persons who are ill nourished is popularly believed to be a source of inflammation. The tissues are badly nourished, so that wounds or injuries on them tend to inflame and fester, rather than to heal, and that out of proportion to their injury or hurt. Privation of food is therefore an active determining cause of inflammation; leading, most probably, to a certain quantity of textural death as the direct result of the privation.

The action of specific irritants with which blood may be charged, which lead to inflammation, may be exemplified in the fact, that *arsenic* applied to an external sore produces its characteristic inflammation on the mucous membrane of the stomach, just as efficiently as if it had been swallowed. So will the application of a cantharides blister produce strangury. From the contact of blood thus poisoned, the stomach or the kidneys inflame; and so also serous inflammations flow from blood in Bright's disease being poisoned with the elements of uneliminated urine, acting as an irritant to the serous membranes.

6. *Direct contagion* causes inflammation, as when the material secretion from a vaccine vesicle or a primary syphilitic sore, or from small-pox, is inserted into the skin by a scratch, and produces a train of inflammatory phenomena peculiar to each kind of inoculation. Also, when gonorrhœal pus comes into contact with a healthy urethra, inflammation, with a flow of similar specific pus, forthwith commences. And as with drugs, so with the specific contagia of morbid poisons, they exhibit affinities for some organs rather than for others.

(2.) *Predisposing causes or influences* are such as dispose the tissue to suffer from the visitation. “Of a given number of persons exposed to

the same exciting cause of inflammation, in comparatively few does the cause take effect, and the few are said to suffer in virtue of some predisposition. Those in whom the predisposition is great suffer more than those in whom it is slight. Again, the organ which becomes inflamed varies according to the individual predisposition; and thus it is that cold will excite in one person *tonsillitis*, in a second *pneumonia*, in a third *enteritis*, and in a fourth inflammation of the kidneys. Lastly, the quality of the inflammation produced by the same exciting cause will vary with the specific predisposition of the individual; thus, in one it will be rheumatic, in a second gouty, in a third syphilitic, and in a fourth it will show a tendency to gangrene." (MURCHISON'S *Lectures on Practice of Medicine*; *Brit. Med. Journ.*, March, 1872.)

The predisposing causes of inflammation are thus certain influences which co-operate with the determining causes. They embrace those influences which make one man at one time apter than another to initiate an inflammatory disease, or special forms or varieties of inflammation. Examples of such influences, Simon states, are to be found in chronic inanition, exhaustive diseases of old age, local disease of arteries, local obstruction of veins, local defects of innervation, previous inflammation in a part, over-feeding of the body, over-stimulation from alcoholic drinks, ingestion, by breathing or otherwise decomposing organic matters, climatic influences, such as heat. Morbid states of the blood and its contamination with such morbid poisons as small-pox, scarlet fever, typhus, and the products resulting from the pyrexia of these diseases. Secondary inflammations are thus apt to occur in these diseases; so also in the morbid states of the blood associated with constitutional syphilis, scrofula, rheumatism, and diabetes; the condition of the blood engendered by the abuse of alcohol; the retention of the excessive products of tissue-change owing to gouty constitution, or to the common habit of eating too much food with too little exercise. Such persons are prone to inflammatory "colds" from the slightest causes. (MURCHISON.)

ULCERATIVE INFLAMMATION.

LATIN EQ., *Inflammatio Exulcerans*; FRENCH EQ., *I. Ulcératione*; GERMAN EQ., *Geschwürige Entzündung*; ITALIAN EQ., *Inflammazione Ulcerosa*.

Definition.—*A form of inflammation which, eventually approaching a cutaneous or mucous surface, or internal surface of a tubular vessel, induces a breaking up of the surface, and the formation of what is termed an ulcer.*

Pathology.—When the process of inflammation takes place beneath a surface, that surface eventually gives way, or is directly transformed into a soft diffuent mass. A mode of suppuration or pus-formation is thus established, and *granulations* appear. These consist of a tissue, where, in a small quantity of soft intercellular substance, round cell-elements are embedded, proportionate in quantity to the proliferation of the granulations. The nearer the surface is approached, the more do the cells present divisions in their nuclei, which, in the deeper parts, were mostly uni-nucleated before; and quite on the surface they cannot be

distinguished from pus-corpuscles. If this mode of growth or proliferation continue abundant, the mass keeps constantly breaking up, the cells pour themselves out upon the surface, and a destruction takes place, which, making deeper and deeper inroads into the tissue, throws up more and more of its cells and débris upon the surface. Thus an open condition of the surface is brought about, to which the name of an *ulcer* is given. The term *ulceration* is now being restricted so as to express the removal of the superficial or exposed particles of inflamed parts; but when epithelium or epidermis of a part is alone removed, and none of the vascular or proper tissue beneath it, then the result is a mere "*abrasion*" or "*excoriation*" (PAGET).

Three processes thus progress simultaneously in order to effect ulceration, namely:—(1.) An exudation of *inflammatory lymph* and *serum* surrounds the mass of young cells, which constantly continue to grow and to break up (proliferation). (2.) Cells are thus continually growing on the surface, to be carried off by a fresh exudation. (3.) Liquefaction of the gelatinous interstitial material supervenes, and so destruction of tissue takes place continuously. Thus *ulceration* advances.

Ulcerative inflammation is always attended by a loss of substance, a destruction of parts, and a more or less abundant secretion of a puriform, ichorous, fœtid, sanious, or an otherwise variously-coloured fluid; but in the midst of this destruction and death of parts, the growth of *granulations* may eventually predominate as inflammation subsides. Thus an ulcer heals by *granulation*.

Granulation, therefore, is one of the modes in which a wound, or sore, or a part previously acutely inflamed, may heal. It is then said to do so by "second intention," and is always a more or less slow process of repair. Granulation may occur with or without suppuration. The first mode is extremely common. The latter is occasionally seen in the healing of syphilitic maculæ and ulcers of the cornea.

Granulation is associated with an exudation of inflammatory lymph, into which old vessels extend and new ones are formed. A new surface thus results, which is "granular"—the *granule* or *granulation* being a small conical tumor or growth, composed of a mesh of terminal loops, formed by capillary vessels shooting into the effused lymph. The figure and colour of the granulation are determined by the state of the circulation; when that is feeble and inclined to stagnate, the granulation is broad, flat, and spongy, and either pale or of a livid hue; when, on the contrary, it is vigorous, the granulation is conical or acuminate, and of a bright-red tint (TRAVERS). The vessels prolonged into the granulation are more or less tortuous, and so numerous as to require a high magnifying power to exhibit their distinctness after successful injection. These vessels become contracted to obliteration as the period of cicatrisation approaches. Granulation may take place from a surface, or from the sides of an abscess. If from the cutaneous tissue, the sore heals by a process of skinning, the skin always springing from the edges of the wound. Again, if granulations spring from the walls of an abscess, their opposite surfaces may unite. Granulations sometimes form with great rapidity. Mr. Hunter recorded, after trephining a patient, that the dura mater strongly united to the scalp in twenty-four hours.

Granulations, however, have not in all cases an equal disposition to unite. Thus the granulations of fistulous abscesses are little prone to adhere, their surfaces being often as difficult to unite as those of a mucous membrane; indeed, it is often impossible to produce adhesion except by exciting a considerable inflammation. A part having healed by granulation uniformly contracts. This contractile force is so great, that although the sore made by the amputation of a thigh is seldom less than seven or eight inches in diameter, yet the cicatrix left on healing is hardly more than an inch or an inch and a half. From this cause we find, in parts that have been the seat of abscess, a marked depression at the point of cicatrization:—cicatricial contraction.

The reproductive energy of parts which heal by granulation, however, is not great. It is rare that the original tissue is perfectly reproduced. No fat, for instance, is regenerated in ulcerated adipose tissues; a muscle, being divided, unites by a cicatrix of connective tissue, no muscular fibre being reproduced; and a divided cartilage unites by tough fibrous tissue, but not by a cartilaginous bond of union. The skin, when destroyed, may be reproduced, especially by the method of *grafting* or *transplantation* of germs of the *rete mucosum*. After small-pox, the *rete mucosum* is either slow in forming or never forms at all, so that the cicatrix or *pit* remains whiter than natural. Neither the smooth muscular fibres, nor any of the glandular structures of the skin, are formed in its scars; but its fibro-areolar and elastic tissues, its papillae and epidermis, are all well formed in them. The reparation of the mucous membrane is equally imperfect, the villi being always wanting. The reparation of a flat bone, such as the cranium, is so slow, that ten, twenty, and even fifty years pass away before a small trephine hole is filled up with bony matter. In like manner a healed cavity of the lungs is always marked by a cicatrix of areolar tissue altogether different from the original structure; neither, as far as we know, is the proper tissue of the liver, of the spleen, or of the kidney, restored. A nerve simply divided is united by nervous matter in about twelve months or more; and the union is quicker and better in all tissues if air is excluded from the healing of the part.

It is a rule of all cicatrices that the newly-formed part is harder and of greater density than the original structure. Muscle, for instance, unites by coarse, dense, connective tissue; tendon most frequently by a harder and less pliant, but not tougher tissue, and sometimes by bone; and bone after a fracture is a more compact substance, and contains more phosphate of lime than before the accident; but, notwithstanding this addition, the new bond of union is not so strong, nor the living actions so energetic, as in the original structure. For when the constitution becomes enfeebled by severe disease, of a scorbutic kind especially, an old sore has been known to open, and the ends of a once-broken bone again to separate. It is equally a rule that a part having been once inflamed, the liability of the part to that form of inflammation is greatly increased; and also, when new membranes or tissues have formed, that these tissues are infinitely more prone to disease than the original membrane.

The sites of ulcerative inflammation with which the physician has

most frequently to contend, besides the skin, are especially the cervix uteri, duodenum, larynx, pharynx, cesophagus, stomach, intestines, gall bladder, and urinary bladder. The form and mode of ulceration in each of these parts are peculiarly influenced by the anatomical nature of the textures implicated. Sometimes a distinct and limited slough is first cast off, and leaves the ulceration beneath—sloughing ulcers—as in those of dysentery, where the smallest and most superficial ulcers of the gut are preceded by the death and detachment of portions of the mucous membrane, with its epithelial investment (BALY).

SUPPURATIVE INFLAMMATION.

LATIN EQ., *Inflammatio Suppurans*; FRENCH EQ., *I. Suppurative*; GERMAN EQ., *Eiterige Entzündung*; ITALIAN EQ., *Inflamazione Purulenta*.

Definition.—*A form of inflammation resulting in the formation of pus.*

Pathology.—One of the most frequent and important results of inflammation is the formation of pus. If a phlegmon or boil be observed, when it is a firm, hard, and solid mass of texture and exudation, we may feel in a few days that the solid mass has become fluid, and that it has not increased in bulk. The solidity and hardness are due to the inflammatory changes and effusion, the softening is due to the growth of pus-cells developed from the germinal elements of surrounding tissue (VIRCHOW, BEALE), or from the white blood-corpuscles. So it is with vesicular eruptions which become pustular. The new cells there also become pus-cells—a change which may be accomplished in twelve hours or sooner (PAGET).

The following circumstances point to the development of pus from pre-existing germinal matter—namely, that,—(1.) A preliminary lymph-cell cannot always be discerned; (2.) The modification of the suppurative process which occurs in the inflammation of mucous surfaces (shows the formation of pus at once taking the place of the natural cell-growth, without any apparent distinction or alteration of the membranes of the mucous cells), corresponding in this instance to the most simple idea one can have of what Virchow terms *parenchymatous inflammation*, as described at p. 64. Ultimately the natural mucous secretion undergoes a change. The characteristic cells on its surface drop off in all stages of abortion. Impaired cohesion of parts results,—an invariable expression of the inflammatory tendency. The epithelial covering becomes less characteristic, and gradually declines to small and simple cells, mingled with many primordial cells, which appear to have been hurried from the surface before they had time to undergo their legitimate development into the perfect mucous cells. From this sketch of what occurs, “it will be obvious,” as Mr. Simon writes, “that the anatomical distinction between pus and mucus must be as useless as the so-called chemical tests. Infinite gradations between the two destroy all practical value in such criteria. Mucus, as a copious fluid secretion, has no existence in health: the only natural secretion of a mucous membrane is its epithelium, which ought not to exist in quantity sufficient for any evident discharge. If the

secretion be hurried, as in *catarrh*, it immediately begins to assume the forms and physical characters of pus, even to the splitting of its nuclei with acetic acid." In short, the essential process of inflammation has been established *in the very cell itself*, by the abnormal nutritive morphological relations which take place between it and the blood in the processes of life. Inflammations of mucous membrane with a *mucinous* exudation (quite as characteristic of inflammation as *fibrinous* exudations) appertain to certain surfaces—*e.g.*, the gastric catarrhal inflammations. Such mucus is loaded with *mucine*, as a characteristic product of the inflamed mucous membrane, and which gives the tenacious, stringy character to the discharge. Between healthy pus and healthy mucus there can thus be no confusion; but there are conditions between the two which yield neither "*praiseworthy*" pus nor healthy mucus.

Suppuration—Formation of Pus.—Well-formed, perfectly elaborated pus is a smooth, viscid, yellowish or cream-coloured fluid, specifically heavier than water, averaging generally about 1·030, having little or no smell, and of an alkaline re-action. Microscopically, it is seen to be composed of certain essential constituents—namely, the pus-cell, and often minute clear particles, which seem to have some relation to the pus-cells as rudiments or nuclei of them. These constituents float in a fluid or serum called the *liquor puris*, which closely resembles *liquor sanguinis*. The *pus-cells* are about $\frac{1}{2500}$ to $\frac{1}{3000}$ of an inch in diameter, pellucid, filled with semi-fluid albuminous contents, and sometimes containing a few minute oil-globules, which give the cells a granular appearance. They cannot be distinguished from the white corpuscles of the blood, or *leucocytes*—a term now used to include all masses of contractile protoplasm, such as pus, lymph, or white blood cells; all of which are endowed with the power of spontaneous movement, which may undergo continuous alterations of form (amœboid), which may migrate into the tissues, and which may multiply by division. Their shape appears to depend upon the density of the *liquor puris*. Sometimes a distinct, circular, dark-edged nucleus may be seen in the paler corpuscles, and sometimes two, or even three particles, like a divided nucleus. The minute clear particles often seen are not more than $\frac{1}{10000}$ of an inch in size. Such are the components of *good, healthy, or praiseworthy* pus—the *pus laudabile* of the older authors—literally, the *pus to be commended*, as showing a benign form of inflammation, indicating that the process, though a morbid one—a disease—is going on regularly, and promises a fortunate issue (WATSON). It is the *laudable pus* of surgical writers. When, however, the process deviates from the healthy state of pus-formation—deviates from the usual and regular course of the morbid action in a person apparently otherwise healthy—then we find not only variations in the pus-cells, but multiform mixtures of withered cells appear, with molecular and fatty matter, escaped and shrivelled nuclei, blood-corpuscles, and fragments of granular matter like shreds of fibrine. The *liquor puris* becomes unduly liquid, and the pus is then said to be *watery* or *ichorous*. It may even, in weak and tuberculous patients, consist chiefly of a thin serum, mixed with flakes or curdled matter, when it has been called *serous pus*. When the colouring matter of blood is mixed with it, it is called *sanious pus*. Chemical or vital changes of various kinds bring about a peculiar decomposition in pus while

yet in contact with living parts, although it is probable that atmospheric air, or gases from an internal cavity, may have to do with the change; but *hydrosulphate of ammonia* is frequently developed, especially in abscesses about the alimentary canal, near the tonsils or the rectum. The stench is then most offensive when the fluid is set free. Pus, besides possessing certain chemical properties, may possess certain *specific disease* properties: thus, it may be impregnated with certain specific poisons, as that of syphilis, or of small-pox; it is also, in certain constitutional states, loaded with such foreign matters as *urate of soda*.

The formation of pus is termed *suppuration*. It takes place under three conditions, namely,—(1.) Circumscribed; (2.) Diffused; (3.) Superficial; or, (4.) Deep. The following are examples:—

(1.) Of the *circumscribed formation* of pus, may be mentioned an *abscess*, a *boil*, or a *phlegmon*, in which the suppuration is enclosed within a cavity whose walls are composed of connective areolar tissue, and into which interstitial exudation of inflammatory lymph and serum has extended over a certain area. It happens that while the central portion of an area has become purulent (*i.e.*, has produced pus-cells as a result of the continuous premature proliferation of tissue), the peripheral part has maintained its firmness and solidity by activity of nuclear growth; and sometimes a “thin, opaque, yellowish-white layer, easily detached,” separates the suppuration area from the denser part. This has been called a “*pyogenic membrane*,” from the supposition that its function is to secrete the pus, whereas the nuclei and cells of the denser parts are growing by continuous but premature development into pus-cells. Abscesses are sometimes formed without any of the usual accompanying signs of inflammation being present. They are generally slowly formed, and are named *old* or *chronic* abscesses. When suppuration happens in the natural cavities of the body, it is still circumscribed by the cavity. It is not then, however, called an *abscess*, but a *purulent effusion* into the cavity.

(2.) Of *diffuse suppuration*, as in *phlegmonous erysipelas*, or the *purulent infiltration* of an organ. In such cases the inflammation extends through a wide extent of tissue, and from first to last the boundaries are ill-defined. The growth of pus-cells is distinctly interstitial. They are generally rapidly formed, and the tissue becomes thoroughly infiltrated, as if soaked in pus. The usual want of cohesion in the elements of tissue involved in inflammation prevails from the first, and ultimately large *sloughs*, or death of portions of texture may take place. In some textures of a loose kind the pus may spread about or infiltrate parts by its own gravity, thereby leading to secondary destruction of tissue and the formation of what are called *sinuses*. The incipient progress of diffuse suppuration is probably not dissimilar to that of a phlegmonous abscess, but the inflammation is generally of a different type, and all the processes are less complete; thus, no fibrinous lymph circumscribes the limits of the abscess, nor does any membrane form to limit the pus. The process of suppuration is less perfect, so that the abscess often contains shreds, or even large portions of mortified and loose connective tissue. The pus is less healthy, is thinner, containing a larger portion of serum, and oftentimes portions of loose fibrinous lymph. The *pointing* of this form of abscess

differs also from that of the phlegmonous abscess, for the pus readily passes from its original seat, by infiltration, and gravitating towards the most depending position, presents a soft, broad surface, without any indications of *pointing*. Such collections of matter are always of greater extent than phlegmonous abscesses, for the free transmission of pus from part to part occasions a great extension of the original disease. When these diffused abscesses open, the phenomena which result depend very much on the nature of the opening, and how it has been effected. "I have seen," says Mr. Hunter, "large lumbar abscesses open of themselves on the lower part of the loins, which have discharged a large quantity of matter, then close up, then open anew, and so go on for months, without giving rise to any disturbance; but when opened, so as to give a free discharge to the matter, inflammation has succeeded, fever has come on, and, from the situation of the inflamed part, as well as from the extent of the lesion, death in a very few days has been the consequence." How different are the results now under Mr. Lister's antiseptic method of opening such abscesses! In erysipelas, however, which so often gives rise to this form of abscess, a free opening is often necessary, to allow of the escape of the portions of loose areolar tissue—openings which ought to be made under Mr. Lister's method.

(3.) *Superficial suppuration* may be observed in *gonorrhœa*, *purulent ophthalmia*, and generally in *inflammation of mucous and cutaneous surfaces*. There the growth of pus can be clearly traced where stratified, as well as columnar, epithelium naturally exists. Upon the skin the development of pus may be seen to proceed from the *rete Malpighii*, as a growth by continuous premature development of cells from this part of the young cuticle. In proportion as these young cells give birth to younger germs (proliferate), a separation of the harder layer of epidermis ensues, and a vesicle or pustule is the result. The exact spot where the growth of pus occurs corresponds to what would be the superficial layer of the *rete Malpighii*; and if the membrane of the vesicle be stripped off, the cells of the *rete*, in process of conversion into pus, in place of epithelium, will adhere to the epidermis, and be stripped off with it (VIRCHOW). In the deeper layers the cell-elements, which originally have only single nuclei (centres of nutrition, growing or germinal centres), divide so that their nuclei (or centres of growth) become more abundant. Single cells have their places taken by several, which, in their turn, again provide themselves with dividing nuclei, and so the process of multiplication goes on.

Dr. D. R. Haldane, of Edinburgh, has observed and recorded the continuous development of pus-cells from the cylindrical variety of epithelium. In a case of small-pox he found the larynx and trachea coated over with a soft, dirty-looking deposit, which was found to consist of pus-cells. On gently scraping the surface, the cells were found enlarged, and, in place of containing a single nucleus, each contained several—three, four, or more. These were derived from the proliferation of the original nucleus. External to the cells were young ones in all stages of development (*Edinburgh Medical Journal*, Nov., 1862, p. 439).

The more completely the epithelium is of the stratified kind, the less is the surface liable to ulceration (*e.g.*, the urethra in gonorrhœa); but those mucous surfaces where the epithelium is of the cylindrical form

scarcely ever produce pus without ulceration (*e.g.*, the intestines). Pus-cells, mucous cells, and epithelial cells, are now regarded pathologically as equivalent elements, which may replace one another; but physiologically they are not equivalent elements, inasmuch as they cannot perform each other's functions. Deeply seated pus-formation may proceed from *connective tissue*, or from the nuclei of vessels or sheaths of tissue. An enlargement of the *connective-tissue* germs occurs (OTTO WEBER) which divide and subdivide, and so multiply excessively by divisions of the germinal masses or cells. Round about the irritated or inflamed parts, where single cells lie, masses or groups of cells are formed, a large new formation grows, and towards the interior of this growth heaps of little cells accumulate (*proliferation*). These little accumulations occur at first as diffuse "infiltrations" of roundish masses, encircled by an intermediate growth, which continually liquefies as proliferation of the cells extends. Virchow regards this liquefaction as of a chemical nature; the intermediate substance (which yields gelatine) becomes transformed into mucus, and, being ultimately converted into an albuminous fluid, is thus rendered liquid.

Thus two different modes of pus-formation are distinguished, according as (1.) the growth of the pus-cells proceeds from the germs of *superficial* tissue, like *epithelium*, or (2.) from *connective tissue*; and two forms of inflammation can in like manner be separated from each other, namely—(1.) The *parenchymatous inflammation* (p. 64, *ante*), where the process runs its course in the interior of the tissue-elements (*e.g.*, connective-tissue cells or germ masses, hepatic cells), without our being able to detect the presence of any free fluid which has escaped from the blood, but where softening and fluidity are due to the process above described. (2.) The *secretory* (exudative) *inflammation* of superficial tissue-elements, where an increased escape of fluid takes place from the blood, and conveys the new products of growth and altered secretion along with it to the surface.

The *parenchymatous inflammation* has from its outset a tendency to alter the elements of tissue and their special functions. Whereas the *secretory inflammation*, with a free exudation, in general affords a certain degree of relief to the part. Witness the relief which follows the free flow of mucus in catarrh. It conveys away a great mass of noxious matter, and the part appears to suffer much less than a part which is the seat of a purely *parenchymatous inflammation*. In gonorrhœa, also, we have an example of how the pus resulting from the *secretory* form of inflammation is carried away by that transudation of fluid (exudation) which removes the pus-cells from the surface without the slightest appearance of ulceration (VIRCHOW).

The description here given regarding the formation of pus is based on the great fact, demonstrated originally by Goodsir, that all new cells proceed from "centres of nutrition," or bioplasm from other cells, or from the nuclei of them; and, as Dr. Haldane justly observes,—“We must not expect to be able, in the case of every abscess or purulent discharge, to trace thus distinctly (as has been done in the preceding paragraphs) the origin of the pus-cells. There is only a certain stage in pathological as in physiological growth in which the actual mode of development can be followed. We might as well expect to be able to discover, by an examina-

tion of the mature foetus, the different steps by which its organs had been formed, as to be able, in a ripe abscess, to determine in what way normal had been converted into abnormal tissues."

There are especially three events which, with more or less frequency, accompany or follow inflammation in a part. These are—*softening, ulceration, and mortification.*

Softening, or diminished cohesion of tissue, is an almost constant result of inflammation. It may be due not merely to mechanical separation by infiltration of the component elements of tissue, but to a loss of the vital cohesive properties and impaired functions of the tissues themselves, which tend towards their liquefaction and degeneration. Examples of this may be seen in the inflammation upon mucous surfaces already referred to, also in the inflammatory red softening of the brain and spinal cord, and in the lungs, where a peculiar brittleness and rottenness are imparted to their fibrous substance or skeleton texture. Such softening is due to vital changes in the proper tissue, often independent of any interstitial infiltration. The most remarkable example of inflammatory softening is that which occurs in bones. An acutely inflamed bone is so soft that it may be cut with a knife (STANLEY, PAGET).

But while some parts are *softened*, others are removed altogether by the process of *interstitial absorption*, as it has been termed. This phenomenon is best seen in bones which have been inflamed. Such absorption gradually precedes the extension of the inflammatory process, and leads, in the case of abscesses, to their spontaneous evacuation, commonly called the "*pointing of an abscess.*" The inflammation continues, and the growth of pus moves along in a definite direction, towards the cutaneous or mucous surfaces of the body in its vicinity; but as the integuments are generally the more prone to inflammation, it is probable that they thus become soft, and yield sooner than the mucous surfaces do.

Hectic Fever.—If suppuration continues beyond the powers of the constitution to supply the process with material to form inflammatory lymph and pus—if the inflammation continues, and becomes chronic as to time, inflammatory lymph continuing to be exuded, and pus continuing to form in profuse quantity, especially if an internal organ is its site—a characteristic type of *febrile* symptoms is apt to supervene, constituting *hectic fever.*

It is not to be supposed, however, as was once believed and taught, that *hectic fever* is due, in every case, to the continued formation of pus. There are forms of *hectic fever* unconnected with suppuration anywhere, but associated with some analogous wasting of the bodily substance; for example, a prolonged secretion of milk in mothers who suckle their infants beyond the natural period. In all cases where a drain upon the system is established beyond its means, such a complex morbid condition of the body as *hectic fever* may be thus induced, and the mischief may not be revealed by any other symptoms.

This type of fever is particularly distinguished from the inflammatory and typhoid forms of fever (described at pp. 75–78), by its remarkable intermissions, which are usually periodical; a period of remission and a period of exacerbation occurring once, and sometimes twice, in the twenty-four hours. It is also characterised by an excessive waste of the

tissues of the body; and the sweating which attends the paroxysms causes great exhaustion. The assimilative and nervous functions are comparatively unimpaired, so that it is a febrile condition generally of very long continuance. The mind remains perfectly clear—often vigorous and active—even when the body is extremely debilitated; and if the intervals between the paroxysms are tolerably free from febrile excitement, the *hectic* type of fever may be protracted much beyond what at first sight might appear credible. Thus it is sometimes within our power to alleviate greatly this condition. If, however, the fever does not abate during the remissions of the excessive paroxysms, when sweating continues profuse, and when suppuration or other wasting discharge is excessive, the fatal termination approaches rapidly.

The leading symptoms of this form of fever have been watched and described minutely by many observers, non-professional as well as professional. The *fever* creeps on insidiously, and almost imperceptibly; and the physician is at first led to suspect its existence only by a very slightly increased frequency of pulse, and a small degree of heat of skin, occurring generally towards evening, and subsiding before the beginning of the next day. The pulse is subject to temporary quick excitement from slight causes, such as by exertion, by emotion, or by food, as after meals. The heat is especially felt in the palms of the hands and soles of the feet. The excitement of the pulse gradually begins to be more and more easily induced throughout the day, daily loses in power and increases in frequency; and towards evening the general exacerbation of the febrile state becomes regular, and is unmistakable. *Periodic* exacerbations or febrile paroxysms occur almost invariably towards evening, and remissions now become distinctly marked. The exacerbation reaches its height about midnight, and terminates by a profuse perspiration or sweating stage towards the morning. This sweating is sometimes called *colliquative*, and may be replaced or accompanied by *diarrhœa*. Occasionally a second paroxysm occurs in the morning after breakfast (WOOD), or at noon, as described by Cullen; and as a mid-day meal was common in his day, it is probable that these slighter paroxysms may be attributed to such causes as the simple taking of food. Generally, however, in the earlier periods of this type of fever, the interval from morning till towards the afternoon and evening is free from fever; but in the advanced stage the fever becomes nearly constant, while the evening exacerbations and the morning sweats remain characteristic to the end. The following diagram (Fig. 7) shows records of temperature in a case of suppuration of knee-joint and abscesses in kidney, described by Mr. John Croft (HOLMES' *System of Surgery*, Vol. I., p. 288). The pulse of the *hectic* patient is scarcely ever so hard and full as the pulse in *inflammatory fever*; nor is it so soft and compressible as the pulse of the *typhoid* patient. It expresses a middle condition between the two, of very variable character, both as to quickness and strength, according to the degree of exhaustion of the patient and the amount of febrile re-action. Often during the paroxysm, or during temporary excitement from slight causes, it reaches 120 beats in the minute, the beat being performed with a jerk, as if the result of irritation upon a weakened heart (WOOD).

The heat of skin during the paroxysm is often considerable, and always

DIAGRAM SHOWING RECORDS OF TEMPERATURE IN A CASE OF HECTIC FEVER (CROFT).

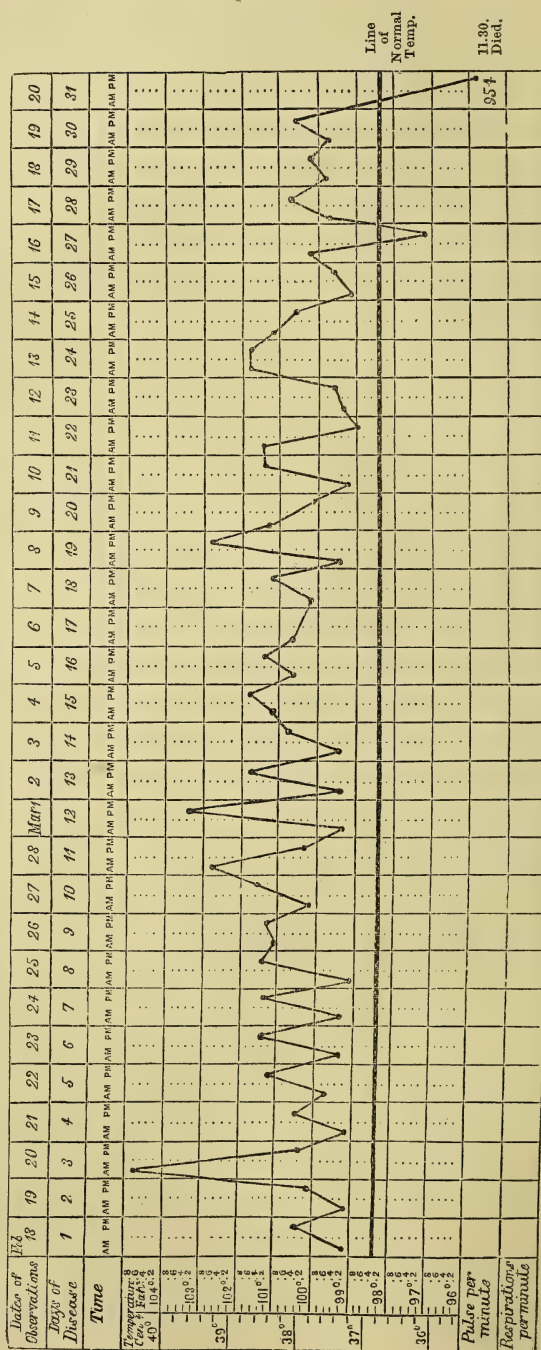


Fig. 7.

distressing, so that little more than the slightest covering can be endured. The respirations are quick and short. The appearance of the face is so characteristic by its florid circumscribed suffusion, that this *hectic flush* of the cheek, limited to a spot in its centre, is now well known. The delicate bright-red colour and circumscribed form of the flushed spot contrast strongly and often beautifully with the pale cheek and the bright and sparkling eye, with its sclerotic of pearly whiteness. The surface of the skin is harsh and dry, and towards the close of life the region of the ankles is apt to become cedematous. The patient loses flesh rapidly, and as death approaches he becomes exceedingly emaciated. It is then that *diarrhœa* is apt to supervene, and to aggravate the sweating, so as completely to exhaust the remaining strength. The mind, unclouded before, gently wanders now, and the functions of life cease, generally without a struggle. It is often one of the closing symptoms, most strongly marked, in pulmonary consumption; and the non-professional pen of the great novelist, Charles Dickens, has beautifully portrayed its more striking features in the death of Smike:—

“But there were times, and often too, when the sunken eye was too bright, the hollow cheek too flushed, the breath too thick and heavy in its course, the frame too feeble and exhausted, to escape their regard and notice. There is a dread disease which so prepares its victims, as it were, for death; which so refines it of its grosser aspect, and throws around familiar looks unearthly indications of the coming change,—a dread disease in which the struggle between soul and body is so gradual, quiet, and solemn, and the result so sure, that day by day and grain by grain the mortal part wastes and withers away, so that the spirit grows light and sanguine with its lightening load; and feeling immortality at hand, deems it but a new term of mortal life,—a disease in which death and life are so strangely blended, that death takes the glow and hue of life, and life the gaunt and grisly form of death.”

PLASTIC INFLAMMATION.

Definition.—*A form of inflammation in which the material produced not only tends to solidify as fibrine, but is loaded with elementary colourless corpuscles; and is, for the most part, peculiar to serous surfaces, to the cut surfaces of wounds, and flaps of amputated parts.*

Pathology.—In the early stages of inflammation of a serous membrane, the free surface, which ought to be smooth and glistening, becomes covered with a soft “lymph,” capable of being moulded into form, and hence described as plastic. It is seen in most characteristic perfection in some forms of pericarditis. On the free surface of any serous membrane so inflamed, the epithelial cells become swollen, opaque, and loosened. They fall off altogether into the cavity of the serous sac. A rapid multiplication of their contained nuclei ensues, set free by the breaking down of the enclosing cells.

Productive changes at the same time occur in the corpuscles of the sub-epithelial connective tissue, and groups of rudimentary corpuscles appear, by germination from the nucleated protoplasm. Continuing the

description, the best ever given, in the words of Sir James Paget, it is found that, "as the inflammation advances, these rudimentary cells multiply with great rapidity, so that adjacent groups run together and form masses of immature corpuscles, which are so numerous in the focus of the inflammation that the part seems to be altogether composed of these new productions, and all trace of its natural structure is lost. Not unfrequently the more superficial layers of this corpuscular lymph become detached and fall into the liquid exudation, which is formed with more or less abundance into the serous cavity, where they may be mingled with the fibrinous coagula which usually form in that fluid" (*Surgical Pathology*, p. 247). It is impossible, without isolation of the cells composing the thick layer of corpuscular lymph, to recognise their forms. Towards the free surface the cells are usually pale round corpuscles, possessing a well-marked nucleus, invested by soft, faintly granular protoplasm. Beyond these the nuclei tend to subdivide, and the cells to elongate into fibre-cells. In the sub-epithelial tissue proliferation in the corpuscles of the connective tissue goes on with an activity proportionate to the inflammatory action. An enlargement first takes place of the single nucleated cells;



Fig. 8.*

then the nuclei divide, and for some time multiply excessively. These changes are very soon followed by divisions of the cells themselves; so that round about the inflamed part, where single cells only were found, pairs, or even multiple groups of cells, are subsequently found. This process is named *proliferation*, and is well shown in Fig. 8, from an inflamed pleura, but the appearances are similar from below any

serous surface; or in connective tissue, as in the flap of an amputated limb (VIRCHOW, PAGET, TURNER).

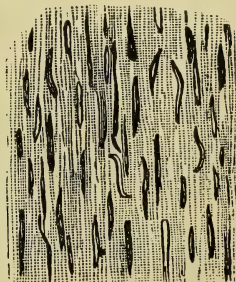


Fig. 9.†

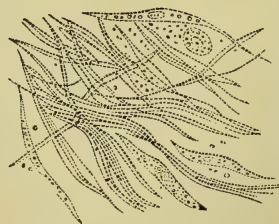


Fig. 10.‡



Fig. 11.§

* From inflamed pleura (after PAGET and TURNER).

† Nuclei in the fibrinous product, developing themselves into fibres (BENNETT).

‡ Fibro-plastic cells developing themselves into fibres (BENNETT).

§ Perfect white fibrous tissue (BENNETT).

In addition to this local multiplication of material derived from the elementary structures of the part, it is at present a subject of inquiry how far the observations of Cohnheim give grounds for belief that much of the purely corpuscular forms on the free and soft parts of the lymph may not have migrated from the blood-vessels.

Examined under the microscope, the lymph of plastic inflammation usually possesses two distinct elements—namely, a filamentous basis, composed of very delicate threads crossing one another in all directions. Amongst these are entangled the cell or corpuscular elements of the lymph. These elements are described as plastic-cells, fibre-cells or fibro-cells, caudate or fibro-plastic cells; all of which may become so elongated and condensed as to assume the appearance of white fibrous tissue. Figs. 9, 10, and 11, after Bennett, show nuclei (Fig. 9) in the fibrinous product developing into fibres; fibro-plastic cells (Fig. 10) developing into fibres; and into (Fig. 11) white fibrous tissue.

Eventually this new matter becomes organised; that is, it becomes supplied with blood and blood-vessels, and is vascular in proportion to the amount of such supply. New blood-vessels sprout up in loops from the vessels of the adjacent texture. When these fibrinous products form on free surfaces, like the pericardium or peritoneum, or on a mucous surface, as in croup or dysentery, the film of lymph is usually called a *false membrane*.

RHEUMATIC INFLAMMATION.

LATIN EQ., *Inflammatio Rheumatica*; FRENCH EQ., *I. Rhumatique*; GERMAN EQ., *Rheumatische Entzündung*; ITALIAN EQ., *Inflamazione Reumatica*.

Definition.—*Inflammation concurrent with an attack of acute rheumatism; or occurring in a person liable to such attacks.*

Pathology.—As the subject of rheumatism will be afterwards fully considered, a notice merely of the peculiarities of rheumatic inflammation is all that is required here. Certain textures are especially liable to rheumatic inflammation—namely, the fibrous tissues of the joints, aponeuroses of muscles, sheaths of tendons, neurilemma, periosteum, muscles, and tendons. The inflammation is attended with great pain (but not so great as in gout), and its severity is probably owing to the dense and unyielding nature of the textures especially implicated, and which are subjected to stretching and pressure in their elements by the dilated capillary vessels and local inflammatory oedema. Such inflammation is generally held to be rheumatic when these fibrous tissues are implicated in an idiopathic way, and independent of any traumatic origin. There is also a great tendency for the inflammatory lesion in rheumatism to pass from the part first affected to others of analogous structure and function.

In rheumatic inflammation of joints, the synovial capsule of one or more is generally the texture most deeply implicated; but the inflam-

mation seldom reaches a great degree. Fluid exudation into the cavity of the joint is not apt to be copious, nor fibrinous, nor very purulent. Inflammatory oedema of the connective tissue round the joint accounts for any visible swelling. A predisposition to rheumatic inflammation is certainly inherited, and those who have had an attack are very liable to others. Recovery is the usual termination, by a gradual and not always regular diminution of symptoms.

GOUTY INFLAMMATION.

LATIN EQ., *Inflammatio Podagra*; FRENCH EQ., *I. Goutteuse*; GERMAN EQ., *Gichtische Entzündung*; ITALIAN EQ., *Inflammazione Gottosa*.

Definition.—*A form of inflammation characterised especially by the intensity of pain, a shivering surface, by oedema of the part, and desquamation of the cuticle when superficial structures are attacked; and occurring in those predisposed to gout.*

Pathology.—As the nature of gout will be afterwards fully considered, the character of the inflammation attending that general disease alone requires a short notice here. The pain of the inflammation is very intense, and differs from that produced by injury. The oedema also is peculiar, and may not at first be obvious on account of the great tension of parts, when inflammation is violent and the skin is distended and shining. When the inflammation begins to subside, pitting is then easily produced, and the presence of fluid made evident. In this respect it differs from rheumatic inflammation, in which oedema is not local. After complete subsidence of the gouty paroxysm, desquamation of the cuticle takes place. It is most usually observed from about the feet and hands, and but rarely from the knees; and, according to Dr. Garrod, the desquamation seems to bear some relation to the amount of the previous oedema, and consequent distension of the skin. Now and then the nails have been shed after a severe attack of gout. The presence of much excrementitious matter in the blood of gouty people tends to influence the products of inflammation; but it is rare indeed that inflammation in gout is followed by suppuration, though, at first sight, when the disease is most intense, and the part is swollen, red, hot, and tender, suppuration looks as if it were inevitable.

The cases which come to suppuration are those in which the gouty concretions (of *urate of soda*), acting as a foreign body, or local irritant, establish suppuration. The inflammation of gout is indeed "characterised by an explosive local appearance of *uric acid*; and on consideration of the circumstances under which this explosion occurs, the inference suggests itself, that materials transformable into *uric acid* were previously accumulated in the texture which inflames" (SIMON).

GONORRHOEAL INFLAMMATION.

LATIN EQ., *Inflammatio Gonorrhœica*; FRENCH EQ., *I. Blennorrhagique*;
 GERMAN EQ., *Gonorrhœische Entzündung, Tripperartige Entzündung*;
 ITALIAN EQ., *Inflammazione Blennorrhagica*.

Definition.—*A form of inflammation arising about five days after the direct contagion of gonorrhœal pus within the orifice of the urethra.*

Pathology.—Although the process of inflammation in the mucous membrane of the urethra, which constitutes a gonorrhœa, is not different from the process of inflammation in any other portion of a mucous membrane, and is an example of a catarrh, yet as the inflammation is peculiar in being due to a specific cause, and gonorrhœa, therefore, is a specific disease, the inflammation requires a short notice here.

The peculiarities of gonorrhœal inflammation are :—

1. Its cause distinguishes it from all other catarrhs which affect the mucous membrane of the urethra or of other regions, and the disease never arises otherwise than by contagion.

2. The nature of the specific poison is as little known as that of small-pox, or other communicable poison; it is only certain that it produces a specific result, invariably the same, case after case. The *virus* is a something fixed and specific, conveyed in the secretion of a diseased mucous membrane, by contact with which secretion, in those susceptible of infection, a similar complaint is transmitted from person to person.

3. Between the period of contagion and the obvious expression of the disease, there is a certain interval—three to eight days—known as the period of incubation; earlier or later periods are exceptional.

4. The susceptibility to gonorrhœal inflammation varies greatly in different persons; and the causes of increased or diminished susceptibility are quite known.

5. Certain portions only of mucous membrane seem susceptible of gonorrhœal inflammation. These are,—the urethra, the female genitals, the conjunctiva, and the rectum; all other parts of the mucous membrane remain incapable of undergoing the specific inflammation of gonorrhœa. Different portions of the urethra also are more susceptible to the influence of the poison than others; and although the infecting secretion acts first upon the orifice of the urethra, the chief site of the gonorrhœal inflammation is in the *fossa navicularis*. During the first and second week of the disease, the anatomical changes are entirely confined to this portion of the urethra, a portion which is very vascular, and very richly furnished with glands. Many complications may be associated with gonorrhœal inflammation, all of which will be fully noticed when the disease is treated of in a subsequent part of this work.

Besides these forms of inflammation, a large number of diseases, specifically distinct as to their causes, symptoms, and progress, are linked together by the common occurrence of inflammation; and in proportion as our knowledge of the causes of inflammation extends, so may we disentangle from the “unassorted heap of inflammations” many varieties hitherto undistinguished (BRISTOWE).

GANGRENE.

LATIN EQ., *Gangræna* ; FRENCH EQ., *Gangrène* ; GERMAN EQ., *Brand*—
Syn., *Gangrän* ; ITALIAN EQ., *Gangrena*.

Definition.—*Incomplete mortification, or death of a portion of a soft organ or tissue, while the rest remains alive.*

Pathology.—When complete death of the part is accomplished, the condition is termed *sphacelus*. In the hard parts, as in bones, a somewhat similar distinction obtains in using the terms *caries* and *necrosis*. Particular names are also given to the dead parts. *Necræmia* means a corresponding death of the blood. A dead piece of tissue is called a *slough*. A dead piece of bone is called a *sequestrum*. Progressive gangrene of soft parts is usually called *sloughing*.

Mortification of the soft parts may be white or black in appearance, humid or dry. The mortified part has a black aspect when the blood is extravasated through the walls of the blood-vessels into the affected tissues, giving to the part a purple or dingy hue, while to the touch it is soft, inelastic, and doughy. It may appear white when, by the action of cold, the blood has been driven from the part before its death commences, which subsequently freezes perfectly white.

Humid mortification occurs when the blood transudes in a fluid state, and after its exudation separates into its constituent parts, so that the serum, set free, dissolves in it the red globules, raises up the cuticle in bladders, and forms what are termed "*phlyctenæ*." Air, generated by a process of commencing putrefaction, is not unfrequently contained in the *phlyctenæ*, and gives to the finger touching the part a sensation of crepitation.

Dry mortification is rare, and has sometimes been caused by the ergot of rye, or rather diseased grain, used as food, giving rise to the condition known as *ergotism*. In the year 1716, dry mortification appears to have been to a certain extent epidemic at Orleans, fifty cases having been treated at the Hôtel Dieu of that city. Dodard described it as beginning generally in one or both feet, with pain, redness, and a sensation of heat or burning like that produced by fire. At the end of some days the part became cold, as black as charcoal, and as dry as if it had been passed through fire. Sometimes a line of separation was formed between the dead and the living parts, and the complete separation of the limb was effected by nature alone. In one case the thigh separated in this manner from the body at the hip-joint. In other cases amputation was necessary. Mr. Solly has given an interesting case of this description, which occurred in the practice of Mr. Bayley, of Odiham. The patient was a child three years and seven months old, from whom, by this spontaneous process of nature, both arms were removed above the elbow, the left leg below the middle of the thigh, and the right foot above the ankle-joint, being a remarkable instance, in modern times, of this destructive disease (see "*Ergotism*," and *Med.-Chir. Trans.*, Vol. XXII., p. 23).

The bones, the brain, the lungs, the liver, the spleen, and the kidney, are all liable to *sphacelus* and *gangrene* ; so are the different tissues, as the

areolar, cutaneous, nervous, and serous. The muscles, tendons, aponeuroses, and blood-vessels, are likewise all liable, but in a less degree.

It is necessary to distinguish the incomplete death of soft parts, or *gangrene*, from the condition afterwards to be described as *degeneration*; and which sometimes precedes complete death of the degenerate part.

The degeneration of a part is to be distinguished from *gangrene*, or actual death, by this,—“that the degenerate part never becomes putrid; and that no process ensues for its separation or isolation, such as we can see in the cases of a dead part” (PAGET). A tissue, however degenerate, remains in continuity with the parts around it, or is absorbed. If the tissue were dead, the parts surrounding it would separate from it, and the dead portion would be ejected from them.

But there are also some conditions of parts where it is impossible to say whether they are dead or alive; a condition which may be termed “suspended animation.” Thus, the end of a finger, in a case of diseased heart, may be cold, livid, insensible, and shrunken for three days; or the foot of an old man cold, livid, purple, mottled, and numb for a whole week; and during these times it could not be told whether the parts were alive or dead. But, as both parts afterwards regained all the signs of life within the days mentioned by Sir James Paget, time alone showed that both parts had been in a state of “suspended animation.” In the same way parts that are frozen, as by the mode of inducing local anæsthesia by the ether-spray, introduced by Dr. Richardson, or parts that are crushed or otherwise severely injured, have their “animation” or vitality “suspended” for a time.

PASSIVE CONGESTION.

LATIN EQ., *Congestio Passiva*; FRENCH EQ., *Congestion Passive*; GERMAN EQ., *Passive Congestion*; ITALIAN EQ., *Congestione Passiva*.

Definition.—*Over-fulness of blood in the capillary vessels of a part; associated with impairment of the vital relations between the blood and minute elements of the texture, as the cause of the sluggish flow of the blood in the capillaries.*

Pathology.—The term *congestion* or *hypercæmia*—meaning, literally, a mere excess of blood in a part, or overbloodedness—has always been regarded as of two kinds, namely, *active* and *passive*. The former, or *active congestion*, cannot be separated from inflammation of a texture, of which it forms a stage or part of the morbid process, as already described, and, in fact, leads up to it; while the latter, or *passive congestion*, merely predisposes to inflammation from trifling injuries, obstructs recovery, tending to molecular degeneration of tissue, and to atrophy; and further, it leads to dropsy.

Much confusion exists as to the use and interpretation of the term *congestion*; and as no one has put the subject in a clearer light than Professor Rokitsky, and still more recently Dr. Moxon, in his very interesting lectures, at Guy's Hospital, on Analytical Pathology, the account here given is based mainly on his exposition, in *Med. Times and Gazette*, July 16, 1870, and that of Rokitsky, in Vol. I., p. 107.

The relations between *active* and *passive* congestion have been described as simply that, *too much* blood being brought into a part, *active congestion*, or *hyperæmia*, is established; on the other hand, when *too little* blood passes out of a part in proportion to what passes into it, the state of *passive congestion* is established. But, in the first case, the description falls short of the truth, inasmuch as too much blood passes out of the part as well as into it; and the congestion is not only active, as regards the flow of blood to the part, but also as regards its flow out of it. In the second case, the description of *passive congestion* involves two forms of congestion, differing in their causes, and also in their results. The causes of one form are purely *mechanical*, the causes of the second are truly *passive*.

Examples of purely mechanical congestion are to be studied in the following morbid conditions:—Narrowing of the mitral orifice of the heart, by the mechanical stoppage of the blood at the obstructed valve, inducing a very intense congestion of the lung. Condensation of the tissue of the lung is the consequence of the persistence of the mechanical obstruction; and, at last, some of the minute vessels of the lung tissue may burst, causing pulmonary apoplexy.

The results of congestion due to mechanical obstruction of the venous current are,—

(a.) Distension of the veins behind the obstruction, leading to stretching and straining of their walls, and so, apparently, to leakage of serum through the distended veins into the tissue, producing *œdema* in connective tissue, or *dropsy* into serous cavities. Habitual use of a tight garter will occasion such results of mechanical congestion of the leg; so will a tumor in the popliteal space or the groin, or habitual distension of the lower bowel by fæculent matter.

(b.) Effusion of blood, either from rupture of over-distended vessels behind the obstruction; or, reasoning from the observations of Addison, Waller, Cohnheim, and others, who contend that the blood-corpuscles find their way through the tissue of capillary vessels without any rupture of them, those cases are capable of explanation where a free and fatal hæmorrhage may come from the stomachal mucous membrane, without any trace of a ruptured vessel or solution of continuity of the mucous surface.

Examples of effusions of blood from vessels behind the mechanical obstruction are to be seen in pulmonary apoplexy, in the free escape of blood into the stomach in cases of cirrhosis of the liver obstructing the portal vein, or in cases of obstructive heart disease, in the cerebral hæmorrhage that results from infantile convulsions, and in hæmorrhage from varicose veins.

(c.) A *thrombus* or *coagulum* of blood may form during life in the obstructed vein. Such clots or thrombi are to be found in the portal veins in cases of cirrhosis of the liver; and in the veins of the bend of the thigh, and prostatic plexus, in cases of cardiac dropsy. These clots begin to form behind the valves of the mechanically distended veins, in the stagnant eddies of blood which occupy the hollows behind the valves. Portions of clots so formed in these veins may break away, and pass into the current of the circulation, and passing up the veins through the right side of the heart, may lodge in the pulmonary artery, and cause either

sudden death or pulmonary apoplexy. This is one form of *thrombosis*; and the primary formation of the clot in this form is to be distinguished from formation of the clot in veins during life, which is due to inflammation of the vein, or close to its wall. Such inflammatory coagula constitute another form of thrombosis. They form wherever veins are subjected to irritation, as in the cerebral sinuses in cases of traumatic or other inflammation of the cranium, the veins of unhealthy stumps after amputation, such clots being sometimes mixed with true pus; and when such clots pass into the lung, inflammation of the part of the lung supplied by the artery is the result.

(d.) Gangrene may be a result of mechanical congestion, as in the limbs and scrotum, from obstructive disease of the heart, or the too tight bandaging of a limb may lead to gangrene of finger or toe.

These four results of congestion succeed each other as the results of successive increments of mechanical force, and are the natural consequences of such mechanical obstruction.

Passive congestion differs from this mechanical congestion both in its cause and in its seat. Severe emphysema of the lungs and chronic bronchitis furnish examples of passive congestion of different parts of the body. In emphysema there is a general lividity of the surface, so constant that it constitutes a valuable sign in diagnosis between emphysema and heart-disease, especially when the presence of a tricuspid murmur in emphysema, or bronchitis in mitral disease, makes the one very like the other. In the case of mitral disease, there is free entry of plenty of air into the lungs, but there is little blood passing through them. The flow through the mitral orifice is a small stream; but it is well aerated blood, and as such freely passes through the capillary vessels and into the veins, where it begins to meet the first mechanical impediment to its farther progress, and is detained in the veins, then in the right heart, and then in the lungs. These being the parts congested, and not the systemic capillary vessels, lividity is generally absent in mitral disease. On the other hand, in *emphysema* of the lungs, the blood circulates freely through the lungs; but not so the air. It is with difficulty, and in an imperfect way, that air is renewed in the lungs. The blood, therefore, is insufficiently aerated, and so passes to the systemic capillaries in an impure state, where a true *passive congestion* is the result. The normal vital relations between the impure blood and the minute elements of tissue are impaired, by the unfitness of the blood to nourish the textures. Hence the skin becomes livid, as the capillaries are gorged with a slowly moving purple current of blood.

Thus the seat and the cause of true *passive congestion* are different from the seat and cause of *mechanical congestion*. The seat of *passive congestion* is the capillaries; and the congestion is largely due to deficient activity in the vital interchanges between blood and texture of the part. In *mechanical obstruction*, the seat of the congestion is in the veins, and the cause is not vital, but mechanical.

The two forms may be combined in cases of general languid action of the heart, where imperfect circulating force is at the same time a mechanical obstacle to the current of blood, and congestion is at the same time both passive and mechanical.

Other examples of passive congestion are seen in the blueness of cold

hands, redness of the extremities, ears, and noses of people with languid, weakly, or feeble circulation; in the prolonged redness or lividity of fauces following severe tonsillitis; hypostatic hyperæmia of the dependent parts of the lungs in fever, where the air fails to pass into the air vesicles during the imperfect respiration. In all these cases the congestion is passive, and secondary to local inactivity in the vital changes between the tissue-elements and the blood. Rokitansky believes passive congestion to depend upon direct palsy of the *nervi vasorum*, wherewith is commonly associated a depressed energy in the remainder of the nervous system. Such palsy may originate in the nerve-centres, or it may be peripheral, and is often determined by a morbid condition of the blood, especially by its decomposition. The passive congestion of asthenic inflammation in organs exhausted by excess of functional activity, enfeebled by active congestion, or paralysed—so also hypostatic hyperæmia of the lungs, abdominal and pelvic organs, and the common integuments of dependent parts developed under diminished impulse from the heart—are examples of such paralysis, causing congestion. The direct results are, that the tissue becomes depraved, becoming atrophied with more or less molecular change of a degenerative kind, and such texture is thereby greatly predisposed to fall readily into inflammation under trifling injuries, when the passive congestion will be replaced by the active congestion of inflammation, and the power to recover from such lesions is very greatly reduced.

The redness of passive congestion is always of a dark or livid hue. Little or no heat is felt in the part; a sense of weight or dulness is experienced rather than pain; and there is neither obvious tension, induration, nor increased proliferation in the part.

The causes of passive congestion may be thus shortly stated, as—(1.) Previous perverted vascular function, such that vital interchange between the textures and the blood is diminished. It may thus come to be a result of previous inflammation or active congestion. Thus local debility of a part from any cause favours the depression of textural vital function, and therefore to passive congestion. (2.) Diminution of the normal proportion of fibrine in the blood renders its fluid portion more transudable, and so favours passive congestion in dependent parts, as in the lungs in fever. (3.) General debility is favourable to passive congestion.

EXTRAVASATION OF BLOOD—HÆMORRHAGE.

LATIN EQ., *Suffusio Sanguinis, Hæmorrhagia*; FRENCH EQ., *Extravasation de Sang*; GERMAN EQ., *Blutextravasation, Hæmorrhagie*; ITALIAN EQ., *Stravaso di Sangue, Emorragia*.

Definition.—*Any discharge, transudation, or effusion of blood in its entirety from the blood-vessels along which it ought to flow.*

Pathology.—Hæmorrhage may be *spontaneous* or *traumatic*. The first kind of hæmorrhage concerns the physician, the second concerns the surgeon. Blood escapes from the containing vessels amongst the interstitial elements of an organ or tissue, either by rupture of a blood-vessel,

by solution of its continuity from disease, such as an ulcer, an abscess, or from injury. As a rule, there is effusion, hæmorrhage, or extravasation of all the blood-constituents; except, perhaps, in those cases of transudation or sweating through the capillaries, as the old term "exhalation" was meant to express, and which may now be explained by the experiments of Cohnheim, Bastian, and others. Virchow's description of a capillary being as homogeneous as a layer of collodion, abundantly testifies to its capacity for "exhalation" in the sense of Cohnheim's experiment. (See page 59, *ante*.)

These recent experiments, reviving those of Waller and Addison, point to hæmorrhages from the capillary vessels of a part taking place without any rupture of vessel or visible breach of surface. Such hæmorrhages, where no breach of surface could be found, were wont to be described as *hæmorrhages* by *exhalation*, and were believed to occur in ninety-nine cases out of a hundred.

Congestions due to mechanical stoppage of the various currents are amongst the most frequent causes of hæmorrhage, probably from rupture of over-distended vessels; and when the hæmorrhage takes place into the substance of any organ, or into any texture interstitial to its elements, the lesion receives the name of *apoplexy*.

But there are also hæmorrhages from free mucous surfaces under various morbid conditions. For example, the free escape of blood into the stomach in cases of cirrhosis of the liver obstructing the portal vein, or in cases of obstructive heart disease, are hæmorrhages from rupture of vessels behind mechanical obstructions. Also cerebral hæmorrhages (or apoplexy) sometimes result from infantile convulsions, and are due to rupture of vessels from obstruction; and so are hæmorrhages from *varicose* veins. These are all examples of hæmorrhage from *mechanical* obstacles to the circulation. Valvular disease of the heart; hypertrophy of the left ventricle of the heart, with capillary impediments to the passage of blood; affections of the liver, like cirrhosis, which obstruct the flow of blood through the portal vessels, producing congestion of the whole portal system, cause hæmorrhage in the stomach and bowels. "Time was," says Dr. Moxon, "when we could speak strongly concerning these hæmorrhages. Thus, the notion of blood passing through the walls of a vessel without a lesion of the latter, is one that could only belong to times when the structure of minute vessels was unknown. Capillaries naturally possess delicate but perfectly solid and continuous walls; and it is just as possible for a single blood-corpuscle to get through that wall without a hole being made for it, as it would be impossible for the entire man to sink through the floor of an apartment without an aperture of his own size existing for his passage. But all this is changed now. If the femoral vein of a frog be tied or pinched in forceps, and the capillaries of the foot well watched, corpuscles of blood are seen to make their way through the capillary walls. They evidently go through holes much smaller than themselves, so that in the middle of their passage, they are hour-glass-shaped, half in and half out of the vessel, constricted much where actually passing. Whether the walls of the capillaries are composed of cells adapted to each other so as to leave pores between them; or whether, rather, our minute tissues, during their life, possess some of that yielding and closing

capacity that one sees in the amœba, which can take in a fellow-creature of its own size through its skin, and then close over it, so that there is no remaining opening,—I think we don't know." "It is, indeed, very surprising to find a very free and even a fatal hæmorrhage from the stomach while the mucous membrane from which the blood must have come is entire; yet no doubt this sometimes occurs. Nevertheless, its occurrence must not be accepted without very careful search over the mucous surface, for the opening of a vessel must be very small." Dr. Moxon instances a case where cirrhosis of the liver led to total obstruction of the portal vein, by *ante-mortem* coagulation in it; and in consequence of this obstruction, a varicose œsophageal vein ruptured close to the cardiac orifice of the stomach. A small hole was found leading into the vein channel, from which the patient had bled to death. The occurrence was equivalent to rupture of œsophageal piles, and is an interesting link, connecting common small hæmorrhoids, that arise from hepatic obstruction, with the dilated vessels on the cheeks and in the mouth—all signs of obstructed hepatic circulation—and which were really facial hæmorrhoids (*Med. Times and Gazette*, July 16, 1870, p. 58).

Hæmorrhages have also been distinguished into *active* and *passive*; and, according to Broussais, all *spontaneous* hæmorrhages are *active*—i.e., they are due to increased action and excess of irritation of the blood-vessels of the part. Those, on the other hand, are *considered* passive which result from external lesion of the vessels, as when hæmorrhage takes place from a blow, which impairs the vitality of the part and allows the escape of blood into the surrounding tissues. Active hæmorrhages are typified in the menstrual flow of the female, which is natural, and in vicarious hæmorrhage and epistaxis, common in the young and plethoric. The predisposing causes are,—good living, the excessive use of fermented drinks, excessive exercise, as well as a too sedentary life. Heaviness and increased pulsation, which makes itself sensibly felt in the part, commonly precedes the flow of blood, owing to the local congestion, and also coldness of the feet. The blood evacuated is generally of a florid red colour.

There are a number of *passive* hæmorrhages which acknowledge some general cause, and which seem to result from some constitutional weakness, combined with an altered state of the blood. Protracted disease of kidneys, heart, liver, or spleen; degeneration of vessels, poor diet, or unsuitable food; long watching, excessive evacuations, previous active hæmorrhage in excess, scurvy, purpura, are instances of conditions under which such passive hæmorrhages are apt to occur. Hæmorrhage from the laceration of vessels and parts of organs diseased in texture, pulpy and friable, as in laceration of the heart, are of this kind. Hæmorrhages are also common when the blood contains less fibrine than in health, as in typhus and in scurvy, while they are rare in diseases in which the blood contains an excess of fibrine. In hæmorrhage from plethora, the fibrine remaining the same, or being diminished, the blood contains a larger proportion of red globules than in health; while in scurvy, or other depressed states of the system, the fibrine is alone diminished, the red globules remaining in normal proportion. In general, in hæmorrhage the blood is not buffed, has a large soft clot, and if the hæmorrhage has been considerable, with difficulty coagulates, showing a diminished quantity

of fibrine. Many substances also which directly contaminate the blood seem to have the power of inducing hæmorrhage. A solution of sub-carbonate of soda injected into the veins of animals deprives the blood entirely of the power of coagulating, and disposes to hæmorrhages. Many morbid poisons, also, as that of typhus fever and of small-pox, have a similar tendency. Hæmorrhage, therefore, may be caused by an altered state of the blood as well as by a diseased condition of the tissue, and in many instances, perhaps, is referable to both causes. Such passive hæmorrhages are not preceded by any signs of local congestion or excitement of an active kind. They are usually associated with pallor of the countenance, feeble pulse, and a tendency to faintness.

Plethora and *anæmia* are thus alike proved to be conducive to hæmorrhages. In youth, plethora is apt to predominate, and then the seat of hæmorrhage is generally the mucous Schneiderian membrane, and is named *epistaxis*. In adult age, hæmorrhages from the lung and bronchial tubes are called *hæmoptysis*; from the stomach, *hæmatemesis*; and from the urinary passages, *hæmaturia*; from the uterus, *menorrhagia*. In old age, apoplexy and hæmorrhoidal discharges of blood prevail, especially from fifty to the end of life. If such hæmorrhages are not suddenly fatal, as may be the case with apoplexy, repeated hæmorrhages from piles, often occurring, tend to embitter existence.

The effects, and even the appearance of hæmorrhages, are generally alarming. The effects must be estimated after due consideration of the cause and the seat of the hæmorrhage. The signs of danger and grounds for anxiety are as follow:—

(a.) Depression, restlessness, deep inspirations (expressed by the term *anxietas*), with rapidity of pulse.

(b.) Pallor of countenance.

(c.) Loss or imperfection of vision.

(d.) Syncope, on attempting to sit up.

(e.) Coldness of the extremities.

(f.) Wandering or delirium.

} Ease or contentment with
desire to be left alone and
undisturbed.

Secondary Hæmorrhage.—Effusions of blood are also apt to occur in connection with the inflammatory process; but such hæmorrhages are generally from rupture of the vessels of the inflammatory products which have recently become vascular (ROKITANSKY). These new vessels are peculiarly delicate, and being apt to rend, like the vessels of new granulation, with a very slight force, especially when they are made turgid or dilated by an attack of inflammation of the newly-formed material in which they lie, they may be sources of considerable bleeding, especially in the stages of congestion and of stasis (PAGET). Such is probably the explanation of the conversion of a *hydrocele* into a *hæmatocele*; the inflammatory products of the *hydrocele* becoming vascular, and being subjected to slight violence, the new vessels are ruptured, and blood pours into the sac. *Hæmorrhagic pericarditis* acknowledges a similar mode of causation. To these effusions of blood Sir James Paget gives the name of *Secondary Hæmorrhages*. Primary effusions of blood in inflammation, *i.e.*, the effusion of blood direct from the ruptured vessels of inflamed parts, mingling with the inflammatory products, are common in *pneumonia*, in which the extravasated blood gives the sputa their characteristic rusty tinge in

that disease. In the inflammatory red softening of the brain, the blood is also effused direct from the vessels of the inflamed part.

Other morbid conditions are also liable to hæmorrhage; for example, such as are highly vascular, as encephaloid and other cancers, and the highly vascular walls of cysts. In all these, the hæmorrhage is generally capillary.

Hæmorrhagic Diathesis—a disposition or liability to habitual hæmorrhage—has been described under the names of *hæmophilia*, *hæmorrhophilia*, *hæmorrhophilis*, and occurs in some persons as a constitutional peculiarity. The tendency is congenital, and is sometimes made manifest immediately after birth by the difficulty with which bleeding from the umbilical cord can be subdued, and in some cases death of the infant takes place from such loss of blood. In after life, and shortly after dentition is completed—*i.e.*, after the sixth or eighth year—the tendency is expressed by the obstinacy of traumatic hæmorrhages, profuse and dangerous bleeding from very slight wounds, by spontaneous bleedings from the gums, nose, bronchi, stomach, intestines, or kidneys; also, as bruises or ecchymosis into the texture of the skin and subcutaneous areolar tissue. The tendency expressed by bruises has been noticed as early as one and two months old, and even ten days old. They occur on any part of the tegumentary system, often so large as to extend from shoulder to elbow, or from hip to knee; and sometimes very painful, of a paroxysmal kind, with violent nightly exacerbation. From these bruises, like ecchymosis, much blood is lost to the system, producing great weakness, and by frequent repetition blanching the skin and mucous membrane. Such spontaneous hæmorrhages, however, do not usually occur till after the patient has suffered repeatedly from traumatic hæmorrhages—such frequent losses of blood tending to hypinosis (diminution of fibrine of the blood), and to impair the coagulability of the blood. Such spontaneous hæmorrhages are generally preceded by certain phenomena moving or struggling towards the crisis (*molimina*) which eventuates in the hæmorrhage. Such phenomena constitute the "*molimina hæmorrhagicum*," and are expressed by cardiac palpitation, tendency to stupor or indifference, signs of cerebral congestion, pains in the limbs, and, in some cases, painful tumefaction of the joints, especially of the wrists, knees, and ankle-joints (NIEMEYER), with ecchymosis and fever (MILLER),—symptoms which continue about a fortnight. These joint-swellings are extremely painful. Mr. J. W. Walker, of Spilsby, is of opinion that serum rather than blood is effused in these parts; and he has given a very interesting account of four children, all bleeders, and males, out of a family of seven—(*Brit. Med. Journ.*, June, 8, 1872)—the three healthy children being girls.

The accidents of a traumatic kind which give rise to these alarming hæmorrhages, are usually extraction of a tooth, a leech-bite, biting the tongue, puncture, slight cut, abrasion, or laceration, as of the hymen; and sometimes the slighter the apparent wound, the more obstinate and dangerous is the bleeding from it. The internal surface of an abscess sac (from a suppurating submaxillary gland) has been known to exude blood; and in the same case, during the cutting of teeth the blood seemed to flow, as it were, from all parts of the gingival mucous membrane. This child died at the age of 16 months, the surface of the body being

thickly covered with petechiæ. The blood oozes from the surface, or wells up from the puncture or cut, as if from a sponge, or like a continuous spring of water, although no bleeding vessel can be discovered. No styptic can staunch the flow; the bleeding may persist for days; and many cases even prove fatal by syncope. At first the blood may appear normal, but gradually it grows thin and watery, and coagulates loosely, if at all. The complexion becomes pallid, waxy, and anæmic; the colour passing from the lips and mucous membrane of the mouth.

When the bleeding ceases, as it may after a period of from three to fourteen days, the patient is left in a state of the most extreme exhaustion, approaching collapse, and recovery is very slow, from the effects of the enormous loss of blood, which sometimes amounts to many pounds.

The slightest contusion, bruise, or pressure on the skin of such hæmorrhagic patients, sometimes leads to extensive extravasations beneath the skin.

The peculiar state of the system is either congenital, or becomes developed afterwards as life advances. Those born with the diathesis have usually inherited the constitution, chiefly through the male line. It usually descends from a family, one of the members of whom, in previous generations, has suffered from the same affection. In its hereditary transmission it furnishes, like gout, instances of *atavism*,—i. e., it may, in transmission through four generations, skip over or miss one, the grandchildren inheriting the disease, but not the children. Thus, lost in one generation, the disease re-appears in the next. But all cases are not so inherited, and even when an inheritance, it is rare for every member of the family to be affected, the males being more liable than the females of the family.

Many cases die young, and, if the tendency to bleeding is great, the period of childhood is rarely survived. On the other hand, cases disclose themselves at a very early age, and, abating as age advances, the tendency to bleeding ceases altogether, and the patient lives to a good old age. The tendency to bleeding also seems to fluctuate; the least abrasion or scratch may at one time threaten fatal loss of blood; at another time bleeding from any similarly slight injury will scarcely attract attention.

Distinct periods of remission and exacerbation are also experienced. The "*molimina hæmorrhagicum*," already noticed, marks the periods of exacerbation, which continue about a fortnight.

The disease has points of resemblance, on the one hand, to scrofula, and, on the other, to scurvy or purpura; but with marks sufficiently characteristic to separate it from both. The most prominent are an obviously delicate constitution—usually a fair complexion—a thin transparent skin, irritability of the circulation at all times, occasional attacks of fever. But the cause does not lie in the constitution of the textures only, for there is a morbid condition of the blood as well as of the blood-vessels. There is a preternaturally delicate and vulnerable structure of the coats of the vessels, associated with a thin and watery condition of the blood. It is deficient in the due proportion of fibrine, and in the power of coagulation. Even when wholly at rest, it is incapable of forming a dense firm coagulum. An undue tendency to congestion of the capillary vessels is a

consequence of these changes in the blood and in the vessels; so that when they are cut, not only do they contain too much blood, upon which they are unable to contract, remaining open and uncontracted, passively pouring out their thin contents; but that blood is deficient in the most important of nature's hemostatics,—the power of coagulation. The minute arterial twigs seem to be devoid of any middle substance. They are of a thin feeble appearance, and unusually capacious, impaired in contractile power and tone. They are friable and easily torn; hence slight bruises produce serious and extensive ecchymosis; coughing may induce a dangerous hæmoptysis, a sneeze may bring on uncontrollable epistaxis, and extravasations within internal cavities not unfrequently follow a very slight cause.

The assemblage of facts shows that in *hæmophilia* we have to do with a substantive disease as much so as Bright's disease, where the blood-vessels are characteristically affected, as Dr. Johnson has shown. Hence *hæmophilia* ought to have a place in our systematic nomenclature.

It is necessary in all such cases to find out, if possible, the source of the depraved blood and constitutional impairment of texture. Some of the organs having to do with nutrition may be contributing a permanent supply of noxious material to the system. Hence it is of importance to discover the definite tissue or organ from which the derangement in the constitution of the blood proceeds. Numerous instances of the hæmorrhagic diathesis have pointed to a definite organ as its source—namely, either a morbid condition of the spleen or the liver; and in cases of *leukæmia*, usually towards the close of life a genuine hæmorrhagic diathesis is developed, and hæmorrhages ensue, occurring with special frequency in the nasal cavity (as an exhausting epistaxis), and also in or from other parts of the body, as in apoplectic clots in the brain, or melæna from the intestinal canal.

The liver, spleen, and lymphatic system of glands require special investigation in all cases of hæmorrhagic diathesis.

The following are the special hæmorrhages, extravasations of blood, or hæmorrhagic lesions, which call for special notice in this text-book; namely,—

1. Cerebral hæmorrhage, or the sanguineous form of apoplexy.
2. Spinal hæmorrhage, or spinal apoplexy.
3. Choroidal hæmorrhage, or choroidal apoplexy.
4. *Epistaxis*, or hæmorrhage from the nose.
5. *Hæmoptysis* (*a.*) from passive congestion; (*b.*) from pulmonary extravasation, or pulmonary apoplexy.
6. Hæmorrhage into the pericardium.
7. *Hæmatemesis*, or hæmorrhage from the stomach.
8. Intestinal hæmorrhage.
9. Hæmorrhoids, internal and external.
10. Hæmorrhage from the rectum.
11. *Hæmaturia renalis*, or hæmorrhage from the kidneys.
12. *Hæmaturia vesicalis*, or hæmorrhage from the bladder.
13. *Hæmatocele*, or hæmorrhage into the tunica vaginalis.
14. Hæmorrhage into ovary.

15. *Peri-uterine*, or *pelvic hæmatocele*, or hæmorrhage underneath the peritoneum, or within the fold of the broad ligament of the uterus.
16. *Pelvic hæmatocele*, hæmorrhage into the areolar tissue of the pelvis. [15 and 16 are sometimes described under the name of *retro-uterine hæmatocele*.]
17. Uterine hæmorrhage, and menorrhagia.
18. Hæmorrhagic cysts.

Some of these forms of hæmorrhage will be considered in detail under the respective organs implicated in the lesion.



Fig. 12.*

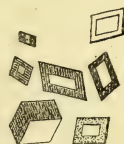


Fig. 13.†

There remains now to be noticed the results of blood extravasations.

The immediate effects of hæmorrhage, besides the anæmia consequent upon great effusion either out of the body or into its cavities, are lesions of continuity in textures, impairment or destruction of function, paralysis of the organ affected, as in *cerebral* and *muscular* hæmorrhage. The irritation, too, of the extravasated blood, acting as a foreign body, may set up inflammation of surrounding textures, with eventual organisation of the effused products, resulting in a callous condensation of texture surrounding the lesion, and the isolation, by enclosure of the hæmorrhagic clot in a more or less dense capsule. It is rare for the hæmorrhagic lesion to become purulent.

A slight hæmorrhage is usually cured by absorption of the effused fluid, so that the injured and distended textures recover their resiliency. The liberated red pigment, however, frequently resists absorption, even in slight hæmorrhages, remaining in a state of minute molecules, scattered over a membrane, or between the minute elements of a texture, as a minute or black pigment. Every extravasation may also leave behind it a contingent of hæmatoidine crystals (Fig. 13), which, when once formed, remains in the interior of the organ in the shape of compact bodies endowed with the greatest powers of resistance. From the numbers of such remains

* Pigment from an apoplectic cicatrix in the brain (VIRCHOW'S *Archiv.*, Vol. I., pp. 401, 454, plate iii., fig. 7). *a*. Blood-corpuscles which have become granular, and are in process of decolorisation; *b*. Cells from the neuroglia, some of them provided with granular and crystalline pigment; *c*. Pigment-granules; *d*. Crystals of hæmatoidine; *f*. Obliterated vessel, with its former channel filled with granular and crystalline red pigment: 300 diameters (VIRCHOW, *Cellular Pathology*, p. 144).

† Crystals of hæmatoidine in different forms: 300 diameters (l. c., p. 143).

any number of old apoplectic attacks may be counted, with remains like Fig. 12.

Prognosis in cases of hæmorrhage is unfavourable under the following conditions:—

- (a.) Into serous cavities, unless limited by other lesions.
- (b.) Into the substance of vitally important organs.
- (c.) Under the influence of the hæmorrhagic diathesis. But in cases of *hæmophilia* the natural history of the disease shows that its tendency after a certain time is to recovery, after, as it were, a certain amount of blood has been drained off.

Treatment.—As the details for treatment of each of the hæmorrhages already named will be considered under the account of the respective diseases, there only remains to indicate here the treatment suitable for cases of the *hæmorrhagic diathesis*.

Nutritious diet, out-door life, and tonic remedies generally, express the line of treatment.

Medicinal tonics are of first importance, and must be patiently persevered in, consistently with the capacity to assimilate food with them. Smart purgative doses of *sulphate of soda* are of benefit in two ways: first, as a purgative and hydrogogue, diminishing the amount of serum in the blood; second, as a chemical salt, which has the effect of increasing or promoting the firm coagulation of the blood. *Acetate of lead* and *opium* also favour coagulation, and tend to calm the circulation. They must be administered in full and sustained doses. *Opium* seems to have a tonic and astringent effect on the capillary vessels, and tends to sustain life under the great depression from loss of blood.

Sulphate of alum and *sulphate of potass*, in doses of fifteen to twenty grains; or *gallic acid*, in doses of twelve grains, frequently repeated, *i.e.*, every two, four, or six hours; or *infusion of matico*, or *oil of turpentine*, may each be used by turns, and according as the stomach will tolerate one rather than another; but the *sulphate of soda* must not be given in conjunction with the *acetate of lead*. The efficacy of *gallic acid* is increased by combining it with fifteen or twenty minims of *aromatic sulphuric acid*. It may also with benefit be combined with *alum*. *Gallic acid* should have a fair trial before giving *acetate of lead*. From two to five grains of *ergot of rye* (*secale cereale*), or the *liquid extract*, in doses of twenty or thirty minims, frequently repeated, say every hour or half hour, has been of use. The *oil of turpentine*, to the extent of ten to twenty drops, is to be given in mucilage every two or three hours. In four cases described by Mr. West Walker, it was the only remedy which was uniformly satisfactory; so much so, that whenever any appearance of bruising was more than usual, the use of turpentine was at once commenced. One drachm of this medicine was beat up with the yolk of two eggs; and one-sixth or one-eighth part of the whole was given twice or thrice daily; or in the form of a gum-cased medicine known as “Thevenot’s globules” of turpentine.

Nutritive but non-stimulant food must be given in small quantities, and frequently. It is best given as fresh meat, fresh animal jelly, coagulated soups, or soup in the jelly form, rather than in the fluid watery form. Pure milk and eggs are to be taken freely, ordinary vegetables in moderation, ripe fruit in excess. Alcoholic stimulants are to be avoided, except

during convalescence, when wine is useful, and good ale and porter are called for as articles of diet (WEST WALKER). Dr. Walker also considers "spruce beer" of value, as it contains some of the terebinthinate constituents of the fir, thus combining in a pleasant form food and medicine. One well-selected plan must be persevered in, so as to avoid capriciously and rapidly shifting from one remedy to another.

As to local remedies, the actual cautery should never be employed, but trust may be placed in gentle pressure, long continued over-applications of strong *tincture of matico*, and *solution of the perchloride of iron*. Subcutaneous injection of *ergota* to the amount of *one grain* of the liquid extract at a time.

As a last effort in cases apparently hopeless, *transfusion of blood* may still be tried.

Consult also the articles on *Scurvy* and *Purpura*; although the usual remedies in these diseases generally fail in cases of hæmophilia.

DROPSY.

LATIN EQ., *Hydrops*; FRENCH EQ., *Hydropisie*; GERMAN EQ., *Wassersucht*; ITALIAN EQ., *Idropisia*.

Definition.—*Dropsy (a contraction for Hydropsy) signifies the accumulation of a watery fluid, occurring independently of inflammation, in one or more of the serous cavities; or a diffusion of watery fluid through the areolar tissue of the body, or its organs; or a combination of all these conditions.*

Pathology.—The distinctive characteristic of dropsy is in the nature of the fluid which forms the accumulation. The fluid is not *liquor sanguinis*, but water containing more or less constituents of the blood-serum. It is devoid of all coagulating elements, or of elements tending to growth or development, and when removed from the body does not undergo spontaneous coagulation. The liquid is clear, like the serum of the blood; and in the so-called "fibrinous dropsies," where the fluid contains coagulated fibrine in greater or less amount, the fluid is not the fluid of dropsy, but is generally the result of more or less inflammation, involving the texture of the serous sac in which the fibrinous fluid is found. In all true dropsies the texture of the part where the fluid exists is unimpaired by inflammatory changes; and the lesions giving rise to the dropsy must be sought for elsewhere than in the dropsical part. The fluid collections in the pleura of *pleuritis* and *hydrothorax* contrast in these respects. The fluid of the former is the result of inflammation, and generally contains flakes of coagulated fibrine; the fluid of *hydrothorax* is a true dropsical effusion. Dropsies receive different names according to their situation, with the word "hydro" prefixed to the name of the cavity. Thus dropsy of the belly, where the peritoneum is distended with watery fluid, is sometimes called *hydroperitoneum*, but is usually now called *ascites*; dropsy of the brain or head, where the ventricles are distended with watery fluid, is called *hydrocephalus*; dropsy of the chest, where the pleural sac is filled with serous fluid, *hydrothorax*; dropsy of the pericardium, where that sac contains the fluid, *hydropericardium*. These are all true dropsies; but the

following are of a different pathological nature, and are not true, but "spurious" dropsies:—dropsy of the eye, *hydrophthalmia*; dropsy of the womb, *hydrometra*; and dropsy of the *tunica vaginalis testis*, termed *hydrocele*.

Dropsy of the areolar tissue of a part, when infiltrated with fluid, is termed *œdema*, and is a lesion common to the areolar tissue of internal organs, as well as to the subcutaneous areolar tissue of the body generally. Hence there is *œdema* of the lungs and of the liver; and when fluid effusion exists in the tissue of these organs, they are said to be *œdematous*. When *œdema* of the subcutaneous areolar tissue is general over the body, this dropsy receives the name of *anasarca*; so that *anasarca* is a *general œdema* of the connective tissue throughout the body. It is distinguished from swelling due to other causes by the absence of redness or tenderness, by the fact that the swelling shifts its place in obedience to the law of gravitation, and by the circumstance that pressure displaces the swelling, causing a pit to remain for some time after the pressure is removed. "Pitting" on pressure is thus a sign of the presence of fluid, or *œdema*; and the combination of *anasarca* with dropsy of one or more of the large serous sacs, is usually named *general dropsy*.

The water of dropsy has the serum of serous membranes as the basis of the fluid accumulation, and is in excess of the normal absorption in the part, which in a state of health balances the exhalation of fluid. When this balance is not maintained, either an accumulation of fluid takes place amounting to *dropsy* somewhere, or a *flux* occurs from some mucous surface. The exact constituents of the dropsical fluid differ in different cases as regards the ingredients held in solution; and, generally in all, more of the water of the blood than of the solids of the serum passes out of the vessels in true dropsies. The specific gravity of the fluids also varies in different parts, the fluid of *hydrocephalus*, 1006·7, and *spina bifida* being the least, *ascites*, 1015, the greater, the serum of the blood being 1029·5.

Generally, the fluid of *hydrothorax* will be lighter than that of pericardial fluid; and the fluid of dropsy is but an augmentation of the kind of effusion or secretion natural to the part, and not merely a sweating or escape of serum. In its re-action, the fluid of dropsy is mostly alkaline, but may be faintly acid or neutral; in its purity it is colourless, or pale straw colour, and of a clear limpid appearance; in the quality of its ingredients it is analogous to the serum of the blood, although in consistence it is thinner, because the proportion of solid matter is much less, being scarcely more than one-third or one-fourth. Chemically, it consists of water, albumen, fat (as cholesterine, in rare cases), pigments, extractive matter, and salts (*chloride of sodium* preponderating over the *carbonates* and *phosphates of alkalis*), and of alkaline earths. Urea is sometimes present when the kidneys are at fault, a fact of much practical importance. The albumen is subject to the greatest fluctuation; and is generally pure as the albumen of blood-serum, or it exists as an *albuminate of soda* or other well-known forms, as well as in forms and modifications as yet unknown. The fluid of pleural dropsy (*hydrothorax*) is richer in albumen than the fluid of *ascites* (LEHMANN); and there is still less albumen in the fluid of arch-noid dropsy, or that of the cerebral ventricles (*hydrocephalus*), than in the fluid of *ascites*. The fluid of dropsy into the subcutaneous areolar tissue (*anasarca*) is the poorest of all in albumen. A red coloration is due to

blood-pigment; a yellow or yellowish green, generally to bile-pigment; a milky whiteness, opalescence, or whey-like turbidity, may be due to accidental mixture with epithelium or fat, as fine cholesterine crystals (often seen glistening in the fluid of old hydroceles, but which are not true dropsies), or it may be due to albumen precipitated by an excess of water relative to the saline contents.

The origin of dropsy is due to several causes, acting singly or in two or more combinations; and, like congestion, it may be sometimes purely mechanical, as—(1.) When it is the *result of retention of blood in the veins through mechanical hinderance to the circulation*. The interruption to the venous circulation as a chief cause of dropsical effusion, was first experimentally demonstrated by Lower, who tied the *vena porta* of a living dog, thereby causing its death by dropsy. The extensive distribution of the dropsical effusion varies in accordance with the site of the obstruction, and is great in proportion as the centres of the circulation are implicated. The fluid also will be in abundance proportionally to the fluidity or excessive wateriness of the blood (*hydræmia*), and is exuded mainly from veins. (2.) In dropsy resulting from *general debility, hydræmia in parts that are paralysed*, effusion takes place mainly from capillary vessels.

Thus four important conditions may act singly or in combination in the production of dropsy; namely,—(1.) Mechanical impediment to the circulation, and especially the free passage of blood through one or other or all of the great organs, namely, the heart, lungs, or liver. Diseases of the heart and lungs cause obstruction to the systemic circulation; those of the liver or portal vein cause obstruction to the portal circulation. The minute veins of these systems then become gorged with blood (venous repletion); the watery portion of their contents transudes through their coats, absorption is diminished, and dropsy is the result. (2.) Altered condition of the blood, by excess of water diminishing the density of blood serum; or by uneliminated excreta, such as biliary or urinary elements in hepatic or renal diseases. (3.) A poor, watery, exhausted blood. (4.) Diminished exhalation in one part of the body, inducing a compensating exhalation in excess in another.

With regard to those numbered (2) and (3) of these conditions, Dr. Murchison is of opinion that, although they may assist in the production of dropsy, it is not certain that alone they can produce it; for when the blood is in an attenuated state, as in anæmia and hyperæmia, its liquid portion passes through the capillaries with increased facility. This is the condition after malarious and other fevers, starvation, hyperlactation, hæmorrhages, and other great drains of fluid from the system, and hence the frequency of dropsy as a common symptom of such conditions. But as the propelling power of the heart is also weakened under such circumstances, the chief cause of the dropsy, as Dr. Murchison justly argues, is no doubt venous repletion.

With regard to the condition numbered (4.), Dr. Murchison observes that “the water which is taken into the body is eliminated by the kidneys, the skin, and the lungs, but mainly by the two former channels, which stand in a vicarious relation to each other. In warm weather, when there is free transpiration from the skin, the quantity of urine is diminished; whereas cold weather, which checks the transpiration of the

skin, increases the flow of urine. When transpiration by the skin is suddenly arrested in consequence of a chill, as when a man, drenched with perspiration from working in a close heated room, goes out without his coat to cool himself in a frosty atmosphere; or, when similarly heated, he is exposed to a heavy rain which wets him to the skin, or when he lies down to sleep upon the damp grass, the quantity of blood which is repressed from the surface may be greater than the kidneys can get rid of, and the result is either an undue flux from the mucous membrane of the alimentary canal, or the effusion of serum into the areolar tissue, or into a serous cavity. These are the cases which some writers have designated *active* or *febrile* dropsy, but which, so far as my experience goes, are invariably associated with acute congestion or inflammation of the kidneys. The results of a chill just referred to are all the more likely to occur if the eliminative powers of the kidneys have been already impaired by disease. For example, in the third and fourth weeks of convalescence from scarlet fever, the kidneys are congested, and their secreting tubes are crammed with desquamating granular epithelium-cells. These organs are not in a condition to respond to any sudden stimulus to increased function, particularly if the stimulus produce increased congestion, which deranges still further their secreting power. When, then, under these circumstances, increased work is thrown upon the kidneys in consequence of a chill, they do not respond, and the patient is attacked with diarrhoea or with dropsy. Similar results may ensue from a chill independently of scarlatina; but even then there will be reason to suspect the existence of some organic change in the secreting structure of the kidney antecedent to the chill. But, irrespectively of any check to the cutaneous functions, diseases of the kidneys furnish other illustrations of dropsy produced in the manner we are now considering. The scarlatinal and other poisons may excite inflammation of the kidneys without any chill of the surface, or the secreting epithelium may undergo a chronic fatty degeneration, the starting-point of which may, or may not, be inflammation. In both of these morbid states—the one acute, and the other chronic—the uriniferous tubes become gorged with epithelium; the elimination of water by the kidneys is impeded; the quantity of urine diminishes; and what may be called a compensatory dropsy is a common result, the transudation of serum through the walls of the vessels being facilitated in the more chronic cases by the impoverished or anæmic condition of the blood. On the other hand, there are chronic diseases of the kidneys (the contracted or granular, and the waxy kidney), in which the uriniferous tubes remain patent, or (in the granular kidney) are even abnormally so from the denudation of the lining epithelium; and where, therefore, there is no obstacle to the escape of the water discharged by the Malpighian tufts. As might have been expected, we find that in these forms of renal disease there is no diminution in the quantity of urine, which, on the contrary, is often greatly increased; that, consequently, dropsy is rarely observed, except in the very last stages, when a large proportion of the uriniferous tubes have been destroyed; and that the skin also is often abnormally dry.” (*British Med. Journal*, 11th May, 1872.)

In addition to these conditions there is derangement of the *osmotic* circulation, between the blood on the one hand, and the free surfaces

of the mucous and serous membranes and tissues on the other. Of this osmotic circulation in relation to mucous membranes, Dr. Parkes writes as follows, and his statement is similarly applicable to that of the serous membranes and connective tissue:—"It is now known that, in varying degrees, there is a constant transit of fluid from the blood into the alimentary canal, and as rapid reabsorption. The amount thus poured out and absorbed in twenty-four hours is almost incredible, and of itself constitutes a secondary or intermediate circulation never dreamt of by Harvey. The amount of gastric juice alone passing into the stomach in a day, and then reabsorbed, amounted in the case lately examined by Grünewaldt, to nearly twenty-three imperial pints.* If we put it at twelve pints we shall certainly be within the mark. The pancreas, according to Kröger, furnishes twelve and a half pints in twenty-four hours; while the salivary glands pour out at least three pints in the same time. The amount of the bile is probably over two pints. The amount given out by the intestinal mucous membrane cannot be guessed at, but must be enormous. Altogether, the amount of fluid effused into the alimentary canal in twenty-four hours amounts to much more than the whole amount of blood in the body; in other words, every portion of the blood may, and probably does, pass several times into the alimentary canal in twenty-four hours. The effect of this continual outpouring is supposed to be to aid metamorphosis; the same substance, more or less changed, seems to be thrown out and reabsorbed, until it be adapted for the repair of tissue or become effete." (PARKE'S *Guln. Lectures on Pyrexia*.)

As a proof also of the rapidity with which fluid in a serous cavity can be taken up into the blood currents, Dr. Richardson injected four pounds of water into the peritoneum of a dog, which weighed twenty-three pounds. Twelve hours afterwards the dog died, and when examined an hour after death, not a drop of fluid was found in the peritoneum. It had all been taken up, and caused death by diluting the blood. This experiment he repeated three times, with a similar result in all the cases. (*On Coagulation of the Blood*, 1856. Exp. 36, 37, 38, 39.)

Dropsy is never a primary affection or substantive disease, but only a symptom of disease, like *jaundice* or *diarrhœa*, and always dependent on some antecedent morbid condition, the mechanism of whose action is implied in the physical conditions just mentioned. To find out the primary cause of dropsy is the practical end in view, and necessary to the successful treatment of every individual case.

An important distinction is made between *general dropsy* and *local dropsy*. In the former, more or less extensive *anasarca* is associated with effusion of fluid into one or more serous cavities, especially the *peritoneum* or the *pleura*. The subcutaneous appearance of the fluid is generally expressed first by a puffiness of the face, especially in the morning, under the eyelids, and next in the feet and ankles in the evening, or in the hands and fore-arms, particularly the left; or it appears at once in all of these

* Grünewaldt's observations were made at Dorpat, on a robust woman, 35 years of age, who had a fistulous opening through the abdominal parietes into the stomach. An account of these observations, abstracted by Dr. Murchison from Grünewaldt's Latin Memoir, will be found in Beale's *Archives of Medicine*, 1858, Vol. I., p. 270. The quantity of gastric juice secreted was equivalent to 14,016 kilogrammes (30·27 lbs. avoirdupois) in twenty-four hours.

seats, and gradually extends throughout the body, associated with which the accumulation in the serous cavities may be also considerable.

In *local dropsy*, one serous cavity, or one organ, or one part of the subcutaneous areolar tissue, is the seat of the dropsical accumulation. The *peritoneum* is the most frequent serous sac so affected, and the accumulation is generally the result of obstruction to the circulation through the liver by the *vena porta*, a result of hepatic disease generally in the form of the complex lesion known as *cirrhosis*, under which this form of dropsy will be more particularly described. But it may be here stated that the effect of the obstruction in the liver, as in cardiac dropsy, is carried back till its effects are felt in the remotest capillaries of this section of the venous system; and hence the *ascites* which is the usual expression of such hepatic obstruction. An example of local dropsy may also be seen in the œdema of the lungs, resulting from the cardiac lesion of *mitral obstruction*. Fibrinous concretions obstructing the *vena cava* and internal iliac veins, or tumors, or a psoas abscess pressing upon these vessels, are also sometimes causes of local dropsy. Disease, by enlargement of the spleen, gives another cause for local dropsy, and is often the result of malaria.

Special details regarding dropsies will be found under the several lesions of which dropsy is a result.*

FIBRINOUS DEPOSIT.

LATIN EQ., *Fibrina Deposita*; FRENCH EQ., *Dépôt Fibrineux*; GERMAN EQ., *Faserstoffablagerung*—Syn., *Fibrinöse Ablagerung*; ITALIAN EQ., *Deposito Fibrinoso*.

Definition.—*A morbid condition in which fibrinous matter separates from the blood, more or less rapidly, and is deposited in various parts.*

Pathology.—The exact nature of this form of lesion is not known, but if the quantity of fibrine be considerable at any one time, it forms a solid mass, of which, perhaps, the best examples are those large yellow pyramidal blocks often seen in the spleen. If, on the other hand, as is more common, a slow and gradual separation occurs, a formation of fibroid tissue takes place, causing thickening of membranes, or similar changes

* *Pneumatoses, or the accumulation of various gases, both within textures, and more particularly in every cavity of the body and of its organs, ought to find some notice here. It does not find a place in the new nomenclature of the College of Physicians. Rokitsansky describes the modes in which gas accumulations originate, as follows:—*

(a.) Gas accumulated in the textures or in cavities is generally *atmospheric air*, which has penetrated from without. For example, most kinds of emphysema, gas accumulations in the pleural sac, gas accumulations in the stomach, and perhaps also, the more rare instances of gas collections in the uterus and urinary bladder; also gas in the blood, after injury to veins, particularly those of the neck, are collections of atmospheric air. Interstitial emphysema and pneumothorax are results of lesions of continuity, through wounds or ulcerations in the bronchial passages, or in the lungs. If air be long retained in any of those situations, it undergoes changes similar to what it undergoes in the lungs. Its oxygen becomes exchanged for carbonic acid, with the superaddition of aqueous vapour.

But some gaseous accumulations are the products of decomposition; as, for example, gas development in stomach and intestines, the result of impaired and imperfect

in more solid parts (H. JONES, *Path. Trans.*, Vol. VI., p. 96). These fibrinous deposits present a light red or yellowish white colour, and occur in a more or less wedge-shaped form, with the apex centripetal and the base of the wedge towards the periphery of the organ. Microscopically the deposit consists of a granular material.

Two views are entertained regarding the production of such deposits—namely, either that they result from minute particles of fibrine being continuously detached at points of the circulating system, more or less remote from the local deposit, but carried there by the current of the circulation (embolism); or they have been regarded as an exudation of fibrine from the capillary circulation. (See *Path. Trans.*, Vols. III., VIII., XIII.)

There are cases where a tendency, either on the part of the blood to precipitate its fibrine, which seems increased in quantity, or there is a tendency to the spontaneous coagulation of the fibrine, it being natural as to quantity, but thus altered in quality. Instances of this exist in cases of *phlegmasia dolens*, especially those occurring as a sequence to typhoid fever. The tendency also exists in those cases where decomposing clots of fibrine are found at times in the cavities of the heart, especially in the appendices of the auricles (J. W. OGLE, *Path. Trans.*, Vol. VI., p. 32).

Fibrinous deposits, or concretions of fibrine, within the heart, are called *polypi*, or *fibrinous vegetations*. When such fibrinous deposits are found in blood-vessels, having been carried from a distance into them, and not formed where they are found, the term *Emboli* has recently been applied to denote such deposits.

ALTERATION OF DIMENSIONS.

LATIN EQ., *Magnitudo Mutata*; FRENCH EQ., *Altérations de Dimension*; GERMAN EQ., *Veränderungen der Gestalt und Grösse*; ITALIAN EQ., *Cangiamenti di Dimensione*.

Definition.—*Alteration of dimension is expressed by an increase or decrease in the volume or bulk of an organ when compared with the normal volume as expressed by the cubic inches of water it will displace.*

Pathology.—Such alterations of dimensions are comprehended under the following lesions—namely, *dilatation, contraction, hypertrophy, atrophy*.

digestion. Its morbid increase constitutes *flatulency* and *tympanites*; the former term implying movement of the gas (borborygmus), and its final expulsion by mouth or by anus; the latter term implies its retention, causing distension, with augmented resonance on percussion of the abdomen. This tympanites is sometimes witnessed to a painful extent in cases of lientery, where the great bowel gets so distended with gas and paralysed by distension, that death has ensued with symptoms similar to intestinal obstruction. The mucous membrane itself is also believed to secrete gas.

Such putrefactive gas development also takes place out of blood mass; or from decaying normal textures or morbid products, such as sloughing cancers.

It has also been observed that the subcutaneous areolar tissue, when slightly inflamed, may secrete air in such abundance as to produce emphysema. Dr. Graves relates a case in which gas was secreted to a considerable amount in the cavity of the pleura.

The terms *dilatation* and *contraction* are usually applied to hollow organs, such as the heart; while *hypertrophy* and *atrophy* have reference to an increase or diminution of the specific texture composing any organ or part.

(a.) DILATATION.

Definition.—*Expansion of the walls of a cavity, so as to enclose greater contents.*

Pathology.—It is generally coupled with hypertrophy of the walls of the dilated organs, when it is known as *active dilatation*. When the enclosing walls of the cavity are of normal thickness, the dilatation is known as *simple dilatation*; conjoined with thinning, attenuation, or atrophy of walls, it is known as *passive dilatation*.

The causes of dilatation of hollow organs may be expressed as follows :—1. *Mechanical impediments*, obstructing the free passage and egress of the contents of the different canals and reservoirs, and occasioning dilatation either beyond or behind the impediment.

They are expressed in the following lesions, namely :—(a.) Constriction of calibre, through pressure from without—*e.g.*, by abscess or tumors. (b.) Coarctation or stricture, consequent upon hypertrophy and change of texture in the walls of the organ. (c.) Foreign bodies, or secretions obstructing canals.

2. *Paralysis of the contractile elements in the walls of the organ.*

3. *Degenerative lesions.*

4. *Inflammations.*

Dilatation tends to destroy life through paralysis of the organ simply, or by the concurrence of asthenic inflammations, or by gangrene, the retained contents of dilated cavities contributing, by decomposition, to *pneumatoses* and farther dilatation, as in paralysis with distension of the large intestine and urinary bladder.

(b.) CONTRACTION.

Definition.—*Contraction, coarctation, or stricture, are also terms applied to diminished capacity or calibre of hollow parts or passages,—such, for example, as local contraction of bile ducts, or of urethra, or œsophagus, stomach, or any part of intestinal canal.*

(c.) HYPERTROPHY.

Definition.—*A lesion in which the enlargement of a part is effected by increase, growth, or development of its natural tissue, with proportional retention of its natural form, and with increase of power in proportion to increase of growth (PAGET).*

Pathology.—Hypertrophy of the heart is a common example; and in its genuine form, the muscular tissue is developed to more robustness. Its fibres become not only larger or more numerous, but firmer, more highly

coloured, and stronger. It is an instance in which the individual elements of the structure take up a considerable amount of matter, and thereby become larger; by which, and in consequence of the simultaneous enlargement of a number of elements, at last the whole of an organ may become swollen or enlarged. When a muscle becomes thicker, all its primary fasciculi become thicker. So also a liver becomes enlarged by *hypertrophy*, simply in consequence of a considerable enlargement of its individual cells. It is a genuine hypertrophy without new formation (VIRCHOW). In the pregnant uterus also such fibres are formed as are not seen in the unimpregnated state. They are not a new kind of fibre, but they differ in size and shape, and are much more powerful than those which compose the uterus in its unimpregnated condition. It is an enlargement of the organ effected through increase by development of its natural tissue. It is the *hyperplasia* of Virchow. Such hypertrophy of pregnancy is natural; but it is imitated in disease, when, by the growth of fibrous tumors in the uterus, the womb attains the size, the structure, and full capacity of action of the pregnant organ, so that even the course of labour is imitated, and the fibrous tumor is expelled by the contractile power of the uterus, as if it had been a foetus (PAGET). Hypertrophy involves an abnormal activity of nutrition, and also at the same time preserves the proportional natural form of the part. Thus simple hypertrophy is scarcely to be distinguished from the results of nutritive irritation; but, on the other hand, mere nutrition increase, *without* irritation (which is equivalent to exercise), will not produce hypertrophy. Sir James Paget, therefore, has well stated the essentials for hypertrophy to consist of the following conditions, namely:—

1. *Increased exercise of a part in its healthy functions (or irritation short of exciting inflammation).*
2. *An increased accumulation in the blood of the particular materials which a part appropriates to its nutrition or in secretion.*
3. *An increased afflux of healthy blood.*

As examples of the first, the arm of a blacksmith, whose muscles acquire bulk and power from constantly recurrent and vigorous contraction by exercise in the use of the hammer; or the great robust heart of a man who has suffered from some disease producing an obstacle to the movement of the blood. In the great majority of cases of hypertrophy of the heart, the lesion is due to valvular disease, which presents an obstacle to circulation of blood through the organ. So also is the urinary bladder hypertrophied in consequence of stricture of the urethra. "The bladder," says Mr. Hunter, "in such cases having more to do than common, is almost in a constant state of irritation and action, by which, according to a property in all muscles, it becomes stronger and stronger in its muscular coat; and I suspect that this disposition to become stronger, from repeated action, is greater in the involuntary muscles than the voluntary; and the reason why it should be so is, I think, very evident; for, in the involuntary muscles, the power should be in all cases capable of overcoming the resistance, as the power is always performing some natural and necessary action; for whenever a disease produces an uncommon resistance in the involuntary parts, if the power is not proportionally increased, the disease becomes very formidable; whereas, in the voluntary muscles there is not that necessity, because the will can stop whenever the muscles cannot

follow; and if the will is so diseased as not to stop, the power in voluntary muscles should not increase in proportion" (Sir JAMES PAGET'S *Catalogue of College of Surgeons*, Vol. I., p. 3; and HUNTER'S work, Vol. II., p. 299). Thus it is that the œsophagus, the stomach, or the intestinal canal (as often as any portion is the seat of stricture) becomes hypertrophied as to its muscular coat above the seat of stricture.

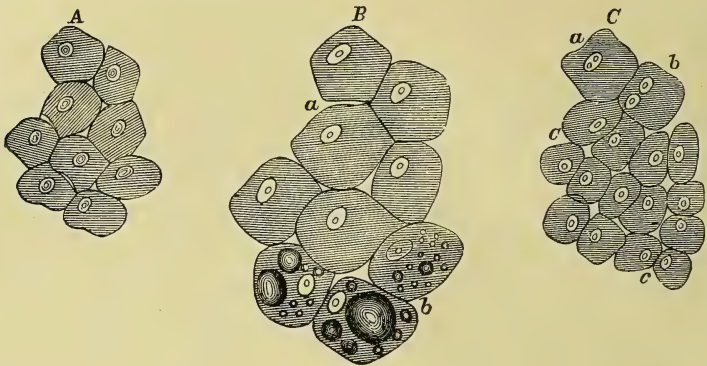


Fig. 14.*

It is still undecided whether, in muscular hypertrophy, the increase of size is owing exclusively to enlargement of primitive fibrillæ, or whether new fibrillæ are produced. If new fibrillæ are produced, the enlargement, according to Virchow, would be *hyperplastic* and not *hypertrophic*. In *hyperplasia* new or more numerous anatomical elements are generated, which contribute to the enlargement of a part by real increase of new material, and which must be distinguished from that enlargement of a part which is a genuine hypertrophy. It will thus appear obvious that mere enlargement of a part is not necessarily hypertrophy of the part; and, therefore, it is important pathologically to distinguish *real hypertrophy* from *apparent* or *false hypertrophy*. A liver or spleen enlarged by lardaceous disease is an example of *false hypertrophy*; such liver or spleen, as to structure, being really in a state of *atrophy* from wasting of the normal elements. Cases in which an enlargement takes place in consequence of an *increase in the number of the elements*, are examples of *hyperplasia*. Thus, a liver may become enlarged by a very abundant development of a series of small cells in place of the ordinary cell development (c, Fig. 14). *Hyperplasia* may therefore be considered as a *numerical hypertrophy*, due to a proliferation or reproduction of tissues similar to the original part; and the condition of the pregnant uterus is thus one of *hyperplasia* rather than genuine *hypertrophy*.

* Diagrams of hepatic cells.—A. Their simple physiological appearance. B. Hypertrophy: a, simple; b, with accumulation of fat (fatty degeneration, fatty liver). C. Hyperplasy (numerical increase or adjunctive hypertrophy): a, cell with nucleolus and divided nucleolus; b, divided nuclei; c, c, divided cells. (After VIRCHOW.)

(d.) ATROPHY.

Definition.—*A deficiency of the formative process, by which a part simply wastes and is reduced in size, with little or no change of texture, or with gradual and continuous degeneration (PAGET).*

Pathology.—As there are two forms of hypertrophy—the one with growth, the other with development (the *hyperplasia* of Virchow); so there are two modes of atrophy—the one with simple decrease, the other with gradual and continuous degeneration of tissue. In both forms, there is invariably a loss of functional power in the part: but in the one form—that of simple decrease—the loss is due to deficient quantity; in the other form—that of degeneration—the loss is due to deteriorated quality of the tissue. Atrophy does not necessarily imply diminution in the bulk of an organ. The atrophied organ may, in reality, be increased in size, as in the false hypertrophies already noticed, especially fatty and lardaceous degenerations. Atrophy is sometimes a natural process, as in the decline of gland textures when the need for their existence, or natural term of their special life, has ceased; for example, the thymus gland, Peyer's patches after forty or fifty years of age, the ovary, testicles, and genital parts generally in old age. Progressive atrophy of all the organs and textures is incident to old age, and is called *senile atrophy*. In some instances certain tissues waste continuously or progressively; for example, the muscles in that form of disease which will be described as "*progressive muscular atrophy*," and where the volume of the affected muscles is markedly diminished as they become successively implicated in the disease. Atrophy, with diminution of volume, is also illustrated in certain chronic diseases of the kidneys, marked by contraction of their bulk; and also in cirrhosis of the liver. The emaciation of scrofula, with or without tubercle, is an example of atrophy affecting the fatty or adipose tissue especially; and it is especially expressed by the terms *phthisis*, *consumption*, *marasmus*. It is thus a general atrophy; and its effects exhibit themselves in the wasted appearance of the body as a whole.

The conditions giving rise to atrophy may be shortly stated as the opposite or reverse of those producing hypertrophy, already noticed.

DEGENERATION.

LATIN EQ., *Degeneratio*; FRENCH EQ., *Dégénérescence*; GERMAN EQ., *Degeneration*—Syn., *Entartung*; ITALIAN EQ., *Degenerazione*.

Definition.—*A granular disintegration or detritus within the minute elements of tissues; or, any deterioration by exudation or deposit, in the texture; or, where one tissue or textural element is replaced by something else abnormal to the part.*

Pathology.—The separation of degenerations from substantive diseases is one of the greatest advances in modern Medicine (Sir WILLIAM JENNER).

Degeneration implies a retrograde metamorphosis. In health we are always changing, and *something* is always being removed and replaced; but

in degeneration a passive change goes on, distinct from these living processes, and the result is a granular disintegration of the minute elements of tissue. (Fig. 15.) A gradual transformation and final destruction of anatomical form in the most minute elements of texture go hand in hand with impairment and final extinction of the functions of the parts. Hence these



Fig. 15.

processes have been described as an "*involution of the tissues*."

This is especially expressed in the form of fatty degeneration, which leads to softening and rottenness, and in the form of calcification or petrification. Similar changes occur *post-mortem*, and after tissues are removed from the body and preserved in the bottles of the museum.

Degeneration also implies changes accompanied by thickening of tissues, or by hyperplasia generally, and diminished elasticity (tendons, ligaments, lens, &c.), occurring especially in advancing life.

The general diffusion of these degenerations is characteristic of advancing age. The circumstances under which degenerations occur are of the nature of decay and death. For example, degeneration occurs to an immense extent in the tissues of the aged, especially in the heart and arteries, and to a less extent in the voluntary muscles and the hard textures. Towards the close of the life of a part of the body, degeneration takes place; as, for example, in the textures of the placenta, when utero-gestation is nearly complete—of the mammæ, of the testicles, of the ovaries, and of Peyer's patches. To such degenerations Virchow has given the name of *necrobiosis*, because death and degeneration seem to be brought about by altered life at the close of natural existence. In this respect it may be truly said, that "as we begin to live we begin to die." "To degenerate and to die are as normal as to be developed and to live." A spontaneous wearing out of living parts goes on, so that destruction and annihilation are immediately consequent upon life.

Alterations in consistence are marked characteristics of degeneration. *Elasticity* is impaired, and *softening* is often the ultimate result of such degeneration. Such softening becomes palpable chiefly by the decided friability of the parts, or by letting a gentle stream of water play upon the part, which will wash away the softened matter. The minute elements of tissue lose their coherence, and at last really liquefy, so that pulpy or almost fluid products take their place. When it is remembered, also, how abundantly a granular fatty transformation occurs after death, the nature of degenerations becomes more intelligible; and my friend Dr. Lyons, Professor of Medicine in the Catholic University of Ireland, instituted a series of observations which beautifully demonstrated a process of morphic changes of tissues through dissolution and decay, till the mortal parts of our body return "ashes to ashes" and "dust to dust." To these morphic changes he has given the name of "*Histolysis*." To the same end are the demonstrations of Dr. Quain, regarding the conversion of muscle into fat, and of crude flesh generally into *adipocere*, accounting for the enormous fattiness of certain geological strata in which animal remains are abundant (MICHAELIS, quoted by SIMON). Such experiments and observations as those of Panem, Melsens, Ascherson, Gluge, Lyons, Simon, Burdach, Wagner, Michaelis, in which granules, vesicles, and cell-

forms appear to rise spontaneously out of homogeneous albuminous fluid, will go far to explain many of the conflicting accounts which are given of the nature of the inflammatory products just described, and of the degenerations. Such forms may undoubtedly arise, as these observers show, and, having arisen, they decompose and advance changes such as Dr. Lyons has described under the name of *histolysis*. On the other hand, the *productive* results of inflammation undoubtedly grow from pre-existing tissue-elements, as already described. From this point of view, structural changes in the valves of the heart are the result of one of three conditions:— (a.) Imperfect development. (b.) Endocarditis. (c.) Degenerative changes. These last rarely occur till middle life; not usually till advanced life in civilians. They seem to be frequent in soldiers at relatively early ages. Dr. Lawson's statistics (*A. M. Rept.*, 1866) show a greater frequency of aorta compared with mitral disease in the service; and disease of aortic valves with *hypertrophy* is the typical form of heart disease in the army.

Our knowledge of such degenerations enables us to appreciate lesions of cerebral textures following degeneration of arteries and the capillaries of the brain. Degenerative changes in the coats of larger arteries can be traced in series up to aneurisms through all stages of local dilatations. They do not occur in childhood or early youth; they are concomitants of old age especially, but are seen at early ages amongst soldiers.

All the degenerations are examples of *atrophy with* changes of texture (PAGET), as distinguished from *atrophy* resulting from simple decrease of bulk, the organ or tissue otherwise retaining its usual form, and to some extent its function.

To recognise the following degenerations of tissue after death, the employment of the higher powers of the microscope is essential. Their recognition has very much modified our practice and opinions of recent years, especially as regards the diagnosis and treatment of degenerative heart diseases, also of certain forms of apoplexy. In illustration of this Sir William Jenner gives the following apt illustration in one of his most terse and suggestive lectures:—"Although with regard to the virtues of this or of that particular drug, and to the mode of action of this or of that particular class of remedies, there are and always will be differences of opinion—the evidence that satisfies A being insufficient from the constitution of his mind to satisfy B—with regard to the value of drugs in the abstract, with regard to the value of treatment, there is really little difference of opinion among physicians equally well informed as to the present state of medical knowledge, and equally experienced in practice.

"I say, among men equally well informed. Let me illustrate my meaning. I was one of three who met in consultation concerning a case of apoplexy. In the opinion of one of my colleagues and myself, the only treatment to be adopted was as follows:—To place the patient in the recumbent position, with head and shoulders raised, to enforce absolute rest, to keep the bowels so far loose as to prevent excitement and straining; to apply cooling substances to the head in the event of any heat of the part occurring; to support the patient with light nutritive food, having regard to his habits. The third gentleman protested against the modern system of doing nothing; he was anxious to bleed, to purge, to blister; and, when opposed, was not sparing of the term sceptic, &c.

"Now, the difference of opinion in this case was not due to scepticism on the one side and justifiable faith—*i. e.*, faith justified by knowledge—on the other; but to knowledge on the one side, and absence of knowledge on the other.

"The case was one of *degenerative change*, retrograde *metamorphosis*, of the arteries; one had become so rotten that its wall had given way, its contents had escaped, a clot had formed, and by its mechanical effects had given rise to the symptoms. The heart shared in the degenerative changes; the bleeding had ceased. To those who understood the real nature of the case the lesions present, and the mode in which they had been produced—in short, the pathology of the case—belief in the efficacy of so-called active treatment appeared to be not merely unjustifiable faith, foundationless faith, faith without knowledge, but to be faith in opposition to knowledge, which in Medicine is the worst form of scepticism, inasmuch as it is doubt of truth and belief in error—doubt which may prevent the saving of life, and belief which, embodied in practice, may kill" (*The Practical Medicine of To-day*, p. 4).

(a.) *Fatty Degeneration.*

Amongst the degenerations which are brought about by the spontaneous wearing out of living parts, the most widely spread, and the most important, is unquestionably *fatty degeneration*. It is attended by a continually increasing accumulation of fat, which replaces the minute elements of tissue in different organs; and the presence of such oil or fat in textures uninfamed, is essentially a sign of weakness or of death, representing decomposition of effective material (SIMON). In such *necrobiosis* the elements of the normal tissue completely perish, and are replaced by fat-granules. Examples of this degeneration may be seen in the minute elements of muscle, especially of the heart; in the acini of the liver, contiguous to the capillaries into which the branches of the portal vein break up. In such degeneration the cells ultimately disappear, leading to loss of substance and atrophy of the gland. It may be seen in the blood-vessels, in the *corpora lutea* of the ovaries, in the renal epithelium, and in many pathological products, such as pus, tubercle, cancer, and the like, when in process of decay; and, in short, in nearly all cell-structures this degeneration is known to occur.

In every texture fatty degeneration becomes evident in a similar manner. Isolated, extremely minute globules of fat appear in the substance of the cells, and, becoming more abundant, they gradually replace the normal cell-element. Usually the fat granules appear at some distance from the nucleus; but ultimately they lie as close to each other as in the *colostrum corpuscles* of milk (Fig. 15, p. 120). At last the nucleus is no longer visible, and the membrane of the cell finally disappears, probably by a species of solution. *These minute granules never flow together* in larger drops (as in fatty infiltrations), but remain separated from each other by thin layers of protoplasm. The more these granules increase, the less of the normal protoplasm or cell contents will remain. Finally, the fatty granules replace the whole structure, and reach the limits of the protoplasm mass,

or of the contour of the cell. The nucleus, which hitherto might be recognised as a clear spot in the midst of the dark mass of fat globules, and might be tinted with colouring reagents, now becomes invisible. During this process the protoplasmic mass or cell greatly increases in size, to three or four times its usual diameter—hence the large livers and kidneys in fatty degeneration—and whatever may have been the original shape of the cell or mass, it now tends to become spherical. From their granular appearance the masses are often called “granular corpuscles” each being a globular aggregation of fat-granules held together by an albuminous intermediate substance. This large amount of granular fat gives a yellowish white or butter-yellow appearance to a fluid or to tissue in which it abounds.

The last process of involution in fatty degeneration may be described as a process of “*lactification*.” The granular corpuscles disintegrate—*i.e.*, the albuminous substance which cemented and held together the granules, dissolves away in the constantly alkaline juices which surround them; and the granules scatter themselves uniformly throughout the fluid, which is now “milk-like,” and the granular corpuscle finally disappears—*e.g.* *cancer juice*. Still further changes go on where the products of degeneration are enclosed in a cavity which is in a state *fit to absorb*, or in a state of inflammation, and *not fit to absorb*. Saponification may then take place and finally an abundant deposit of crystals of cholesterine—(fluid of hydrocele)—contained in bile and brain and spinal marrow tissue. Its crystals are very difficult of solution.

If the degeneration occurs in the more rigid structures—as, for example, in the walls of arteries—the fatty granules retain the form of the cell-structure which they replace. Such degeneration in arteries is first seen in the tissue composing the innermost layer of the internal coat. Afterwards the intermediate substance softens, the degenerate fat-granule masses fall asunder, and the current of blood may carry away the particles of fat with it. Thus a number of uneven places (cicatricial-like loss of tissue) may be produced upon the surface of the larger vessels without any appearance of ulceration (VIRCHOW).

In fatty degeneration of the substance of the heart there is discoloration of its whole substance. It assumes generally a pale yellow hue, with peculiar spots on the papillary muscles. Short yellow streaks, which communicate with each other, are to be seen in the direction of the primitive fasciculi, and pervading the substance of the papillary muscles.

Yellow softening of the brain is a form of fatty degeneration; and the yellowness is due to the accumulation of finely granular fat. At every point where fatty degeneration attains a high pitch, great opacity always presents itself. The primitive cells of tissue are always transparent in their normal state, but fat in excess renders them opaque. Thus a transparent part becomes opaque, as in the cornea, where the fatty clouding marks the *arcus senilis*, described by the late Mr. Canton, in persons past middle life, and regarded by him as an index to the existence of fatty degeneration of other more important organs, although the importance of the sign may have been exaggerated. In some form of Bright's disease the uriniferous tubules become filled with fattily degenerated epithelium, which appear as opaque spots on the surface of the kidney.

Additional examples of this fatty degeneration are to be seen in the *fatty liver*, and in *mollities ossium*, atrophied renal capsules, and thymus gland, and the muscles—voluntary as well as involuntary—the fatty degenerations of the placenta, of cartilage, of bone, and of morbid growths; indeed, there is no kind of tissue, healthy or morbid, which may not undergo fatty degeneration.

When the normal structure of the part is thus transformed into fat, the place of the histological elements is gradually occupied by a purely emulsive mass—a kind of milk or fatty *débris*—that is, an amorphous accumulation of fatty particles in a more or less highly albuminous fluid (VIRCHOW).

Practically it is of importance to know what leads to such degeneration. The conditions are mainly as follow :—

(a.) Impediment to the flow of blood to the textures, due to calcification or petrification of the coats of arteries. (b.) Anything damaging to *nutrition of a part* favours such degeneration.

Thus the hypertrophied heart ceases in time to yield the proper physical signs of hypertrophy, more so when the hypertrophy is due to aortic valve regurgitant disease; because the valve ceases to act as a structure sending the blood through the coronary vessels of the heart. Degeneration comes on, and the signs of hypertrophy are obscured and ill-expressed, being overlaid by those of degeneration, which then becomes really a preservative lesion; and from this point of view we ought to discriminate clinically valve lesions arising from endocarditis, as distinguished from degenerative changes due to old age and other causes, and also distinguish the results of changes due to structures damaged from acute inflammation.

Fatty heart and fatty arteries are generally concomitants.

Fatty degeneration and fatty infiltration are lesions which require to be carefully distinguished from each other. In fatty infiltration the fat is but a superfluous constituent of the cell—a substance conveyed from without and retained in the protoplasm. The fat globules, while they become more numerous, at the same time flow together, forming fat cells; so that one seldom sees more than two, or at most three, isolated globules in a cell, and these tend to flow together into a single large drop—the protoplasm with the nucleus being pressed aside, so that the remains of their existence can scarcely be seen, although they may be made to reappear on the occurrence of the fat absorption. The lesion does not thus necessarily tend to destroy function, as does fatty degeneration. A fattily infiltrated liver still yields bile, its function being impaired but not abolished; and with absorption of the fat the function returns. Next to the liver cells, other cells of the organism are disposed to fat infiltration, especially the cells of areolar connective tissue, thus becoming true adipose tissue. The subcutaneous and subserous connective tissue are most commonly so infiltrated; next comes the interstitial connective tissue of the muscles, especially of those which are but little used; and next comes the subfacial, subsynovial, and submucous infiltration of fat into the connective tissue. Such infiltration is apt to occur when the blood carries a superabundance of minutely divided fat—when its *serum* is cloudy, opalescent, and whitish. Fat particles, or minute granules, may be recognised microscopically; but the emulsion may be so fine that nothing is visible. In any case the test

of agitation with ether is the best; it clears every kind of fatty serum, so that when it is allowed to stand the fat rises to the surface as cream. Such serums may be found about three hours after a meal; and fat accumulates in the blood of those who drink much ale, brandy, and such like alcoholics, and in those diseases of the lungs where more fat is ingested and conveyed to the blood than is consumed, the liver being the principal *dépôt* for such surplus fat (*RINDFLEISCH*).

(b.) *Mineral Degeneration—Petrification.*

The process followed by tissues undergoing this form of degeneration is very similar to that described in the previous paragraphs; but it is necessary to distinguish forms of *mineral degeneration* as distinct from *ossification*. Formerly every kind of tissue condensed to the same degree of hardening as a bone was considered to be ossified, and the condition was described as "*ossification*." But although a part may have lime in its intercellular substance, and although stellate cells may be present in it, yet it may be merely "*calcified*" or "*petrified*" tissue, and this condition Virchow briefly describes as "*petrification*."

Pathological ossification presupposes that the tissue or part which ossifies is called into existence by growth; and not that a previously existing tissue or part merely assumes the form or hardness of bone by absorbing calcareous salts. Ossification always begins by a growth of new tissue; and deposition of calcareous salts in its substance does not take place till a comparatively late period.

Calcification, or *Petrification*, is a degeneration comparatively more frequent in the peripheral arteries, and occurs most commonly in cases where there is a tendency to calcifications generally, and where calcareous salts are set free at other points in the system, to circulate with the juices (*VIRCHOW*).

The lesion in its purity, or genuine form, is to be distinguished from the results of *atheroma* of the arteries, which implies a combination of the fatty with the calcareous material—the so-called *ossification*. In both conditions the artery may be felt to be a hard and rigid tube, with a calcareous feel to the knife or the touch. A careful examination microscopically will show that the degeneration is in the middle coat, that *calcification* or *petrification* of the minute muscular cell-elements has taken place, and that the fibre-cells of the circular fibre coat are transformed into calcareous spindle-shaped bodies, mixed with more or less fat. The degeneration may also invade surrounding parts, while the internal coat of the artery may be unchanged. The larger arteries are often brittle, from the mineral degeneration of their tissue—associated with fatty degeneration (*atheroma*). Patches or plates of the mineral substance may be seen imbedded in the middle coat after the inner membrane is stripped off. When the smaller vessels undergo the mineral degeneration, the deposit resembles particles of oil; and the nature of such an appearance can only be determined by the microscope after the application of mineral acids, which will dissolve the mineral matter with effervescence.

Nerve-cells, the fibrous membrane of the brain, the pia mater, and the

choroid plexus, are all liable to undergo the mineral degeneration. Exudations and new growths are similarly liable. The gall-bladder has been seen converted into a calcareous shell, and the pericardium into an unyielding box of mineral matter enclosing the heart. The cardiac valves are thus often covered with mineral encrustations. Cancer and tubercle-growths may be transformed by the mineral degeneration; and the late Dr. J. H. Bennett has shown how the calcareous transformation of such masses is the natural mode of arresting their advance.

The degeneration may follow upon the metastasis of calcareous salts not excreted by the kidneys, in cases of caries of the bones, necrosis, or osseous cancer. I have seen specimens in the interesting collection of Professor Virchow which show that metastatic deposits of bone-earth have taken place in the lungs and in the stomach under such circumstances. Considerable portions of the pulmonary tissue were *calcified* or *petrified*, without any apparent injury to the permeability of the respiratory passages. The lesion in the lung looked like a portion of fine bathing sponge. The mucous membrane of the stomach was in like manner transformed into a *calcified* or *petrified* material. It felt like a rasp, and grated under the knife, so that the stomach-tubes seemed imbedded in a stiffened mass. The basis of such degeneration in which the lime-salts find a resting-place, are the fine fibrous or connective tissues; and hence the degeneration is seen to occur in fibrous tumors, serous membranes, the parenchyma of lungs and stomach, cicatrix tissue of the skin, the valves of the heart, the connective tissue of muscle sheath (as well of the heart as of common muscle), the tunica albuginea, the fibrine coagula found in the heart's cavities and aneurismal sacs, and the thyroid and pineal glands. The certifications of fibrine, of pus, of tubercle, of cancer, of vegetations, of coagula, all pertain to this form of degeneration; and the process may be traced through all stages of progressive degeneration, from the pulp-like condition to cement-like, compact, calculous concretion, as in the phlebolite of veins; also in the turbid, chalky, speedily condensing juice of the cysts of the choroid plexus, and the cell-incrustations of the pineal gland concretions, as well as in the calcification of *sarcomata* and cancers. With regard to the degeneration as seen in tumors, Sir James Paget describes two methods by which it advances—namely, a *peripheral* and an *interstitial* calcification. The former is the rarer of the two. In this form of degeneration the fibrous tumor is seen to be coated with a thin, rough, nodulated layer of chalky or bone-like substance. In the interstitial form the degeneration is interspersed throughout the tumor, and so arranged that by maceration a heavy hard mass is obtained, variously knotted and branched, like a lump of hard coral (*Surgical Pathology*, Vol. II., p. 139).

In the *Glasgow Medical Journal*, for August, 1872, two cases are described by Dr. Coats of calcareous infiltration of the muscular fibre of the heart, by the impregnation of lime salts in a minutely granular form. The salts in one case were dissolved by hydrochloric acid, without the evolution of gas, and were probably phosphate of lime; in the other case the salt was in the form of carbonate of lime, the affected fibres of the heart being converted into rigid cylinders, having a crystalline texture, and effervescing with hydrochloric acid.

(c.) *Pigment-Degeneration—Pigmentation.*

In this degeneration *pigment* takes the place of the minute tissue elements, as fat or lime did in the previously described conditions. It is seen in mucus-corpuscles (as in catarrhal pneumonia), in the pulmonary epithelium, in the acini of the liver, in the epidermic tissue, in the corpuscles of the blood in *ague* and *melanæmia*. As in the former degeneration, so in this one, a distinction must be carefully made between fat-granule cells and pigment, for in both cases apparently the same image is offered to view. The fat-granule cells appear as brownish-yellow corpuscles, but their individual particles have no positive colour; whereas the pigment-cells contain unquestionable grey, brown, or black molecules of pigment, which are opaque (VIRCHOW). The diagnosis between the two is important, as in the brain, for example, where both sorts of granule-cells, namely, pigment-cells and fat-cells, may exist side by side. The former points to apoplexy having existed, the pigment originating probably in a solution of the colouring matter of the effused blood, the fat to cerebral softening. Therefore it is of importance for the pathological interpretation of the diseased condition to distinguish between pigment and fat in the granular form. Such pigment or colouring matter is insoluble in potash and acids, even in nitric acid.

In mucus-corpuscles or catarrhal cells the pigment exists in the form of greyish-black granules. They give rise to the smoky grey spots which are brought up in great quantity in the sputa in catarrhal states of the pulmonary passages; and to an extreme degree where accumulating masses of proliferating epithelium take place, as in catarrhal pneumonia and in the phthisis of colliers, so well described by the late Dr. William Thomson, Professor of Medicine in the Glasgow University (*Med. Chir. Trans.*, Vols. XX. and XXI.)

In the condition known as *melanæmia* (which, like *leukæmia*, has cells circulating in the blood, having made their way into it from definite organs) the cells contain black pigment; in the latter case (*leukæmia*) the cells are colourless. In *melanæmia* coloured elements are met with in the blood which do not belong to it (STIEBEL, VIRCHOW, SCHONLEIN, HEINRICH, MECKEL, FRERICH, and TIGRI). These pigment-cells in the blood were first seen to occur in melanotic tumors, and were supposed to be due to the passage of particles from the tumors into the blood. This is not yet verified by observation. On the other hand, it is to enlarged spleens pervaded by black pigment that the change in the blood is to be ascribed in such cases, the colour being due to the absorption of coloured particles from the spleen. The class of cases which are the most fruitful source of black pigment in the blood are those of malarious diseases—*e.g.*, *intermittent fevers*, and especially in persons who have been long afflicted with a considerable enlargement of the spleen. In such cases Virchow found cells containing such pigment in the blood of the heart, and the cells that bore the colour resembled in size and form the colourless blood-corpuscles; but there were also other cells of an oblong form and nucleated, within which a greater or less number of large black granules were to be seen. It is in the more severe forms of intermittent fever that such pigment-degenera-

tion occurs. Such pigment is seen to accumulate in the minute capillaries of the brain, attaching to the points of division of the small vessels, and sometimes associated with the comatose and apoplectic forms of intermittent fever. Such pigment is also seen in the minute hepatic vessels (FERRICH'S), where it ultimately gives rise to atrophy of the parenchyma of the liver. I have frequently had occasion to examine livers of soldiers who died at Netley, in which a deposition of melanotic pigment in a granular form was visible amongst the interlobular connective tissue, following mainly the course of blood-vessels in an irregular manner; and, as in all the cases yet recorded, associated with a large black spleen. The contamination of the blood in these cases seems due to a degeneration commencing in the spleen.

In *post-mortem* lesions the textures are thus seen to be variously tinted, red, yellow, brown, green, or black, generally resulting from chemical alteration in the colouring matter of the blood or bile. The red pigments, as a rule, are due to the altered hæmatine, originally of a yellow colour. It is the common origin of three different kinds of crystals, namely:—

(1.) Crystals of *Hæmatoidine*—the most frequent products of blood-degeneration (VIRCHOW) (Fig. 13, p. 107, *ante*), are formed spontaneously in the body out of hæmatine; and in their most perfect form present the shape of oblique rhombic columns, of a yellow-red colour, or, in thicker pieces, of a deep ruby-red. In little plates it frequently bears a considerable resemblance to uric acid. In the majority of cases the crystals are of extreme minuteness—difficult to see clearly, even with a power of 300 diameters. They are insoluble in alcohol, ether, dilute mineral acids, and alkalies; and exhibit a peculiar play of green, blue, rose-tint, and yellow colours, under the action of concentrated mineral acids. If large masses of extravasated blood continue to lie for any length of time, this is the substance into which the blood is transformed. An apoplectic clot in the brain, for example, is repaired by a large portion of the blood undergoing this transformation, and the colour of the resulting cicatrix is due to the crystals of *hæmatoidine*. Also, when a young woman menstruates, the cavity of the Graëfian vesicle, from which the ovum escaped, becomes filled with coagulated blood, and ultimately *hæmatoidine* crystals are the last memorials of the event (VIRCHOW). *Hæmatoidine* is allied to the colouring matter of the bile.

(2.) Crystals of *Hæmine*, arising out of *hæmatine*, differ from *hæmatoidine* in this, that hitherto they are only known as artificial products which have not yet been seen in the human body. They are of a dark-brown colour.

(3.) Rectangular crystals or spicules of *Hæmato-crystalline*.

The yellow pigments are due to blood very much dissolved or dispersed, as in ecchymosis, or to bile, when it is absorbed in the blood and tinges all the textures. Colouring matter due to bile may be recognised in the urine by the play of colours it gives with nitric acid. A small quantity of acid gives a green hue; and, as more acid is added, blue, purple, violet, and a red or brown yellow colour will ultimately appear. Of the brown and dark pigments there are two kinds. One kind loses colour on the addition of nitro-muriatic acid or chlorine water; the other resists not only these agents, but even the action of the blowpipe. This latter pigment consists of carbon. The former is a peculiar secretion formed within

cells, or is a transformation of the colouring matter of the blood (BENNETT). Blue and purple pigments have been seen in urine containing *uroxanthin*, or the *Indican* of Schunk; and illustrate the close connection subsisting between animal and vegetable colouring matters (PARKES *On Urine*, p. 198). For much more interesting observations on the nature of pigmentation, consult BENNETT'S *Principles and Practice of Medicine*, p. 249.

(d.) *Fibroid Degeneration.*

This degeneration implies a very gradual transformation of tissue, with scarcely any preceptible exudation of material capable of growth, into a material having a fibre-like appearance.

This fibroid transformation (to be distinguished from "fibroid substitution" already noticed) is chiefly found in membranous structures. It takes part in the gradual thickening of serous membranes and areolar tissue; and on the surface of such organs as the spleen and pericardium covering the heart (white spot) it very much resembles cartilage, by its dead-white appearance, as if the capsule of spleen or covering of the heart had undergone cartilagification (ROKITANSKY). But there is no resemblance beyond appearance between the degenerate formation and cartilage. The capsule of the liver is sometimes similarly thickened, and so are the sheaths of the vessels composing the capsule of Glisson similarly impaired in some forms of *cirrrosis*. The degeneration is the result of long-continued pressure (condensation), perhaps with friction; or may be a sequence of chronic inflammation with an exudation. It is a form of *sclerosis tele cellulose* of new-born children (*hide-bound*). It takes part in the wheals and knolls of skin in elephantiasis, and constitutes cicatrix tissue. In synovial membranes it appears first as a fibro-serous plate, of milk-white hue, from which the serum is ultimately expelled, leaving a dense fibroid band of union, or a thickened, opaque, bluish-white, tough patch, as in the arachnoid, pleura, peritoneum, pericardium, and endocardium. It is especially so on the heart's valves, which become rough, indurated, and thickened under the influence of this degeneration. Milk spots on the heart are of this nature.

LARDACEOUS DISEASE—SYN., AMYLOID DISEASE, WAXY DISEASE.

LATIN EQ., *Morbus Lardaceus*—Idem valent, *Morbus Amyloides*, *Morbus Cereus*; FRENCH EQ., *Lardacée*—Syn., *Maladie Amyloïde*; GERMAN EQ., *Speckige oder Amyloide oder Wachsartige Degeneration*; ITALIAN EQ., *Lardacea*—Syn., *Malattia Amiloidea*.

Definition.—*A lesion in which the normal textural elements of many organs and tissues are transformed into a peculiar substance, suggesting, on the one hand, an alliance (in some respects only) with the chemical characters of amyloid compounds, and, on the other hand, with albuminous substances similar to those which pervade the tissues of fetal life.*

Pathology.—The London College of Physicians no longer regards this lesion as a degeneration, but classes it with other local lesions as a substantive disease.

Professor Virchow, of Berlin, was the first to collect the facts regarding this peculiar lesion, and to put them prominently forward. He proved the frequent occurrence in the animal economy of the peculiar substance, which gradually takes the place of the normal elements in the tissues so diseased. But Drs. Gairdner and Saunders, of Edinburgh, had anticipated many of the views and descriptions of the distinguished Berlin Professor, and, quite independently of him, they initiated in this country the first steps in the elucidation of this very remarkable lesion. They showed that the waxy-like condition of the liver and kidney was due to the same change as that which was seen to take place in the spleen. These valuable communications were made to the Physiological Society of Edinburgh; and an account of them may be read in the *Edinburgh Monthly Journal of Medical Science* for February, 1854, p. 186, and also in May of the same year. Notwithstanding these researches, and those of Drs. Harris, Aldridge, and others in this country, we have much still to learn regarding, (1.) The conditions under which this disease occurs; (2.) The forms in which it exists; and (3.) The symptoms of it.

This disease has been long known by a variety of names. For many years the morbid anatomist has been familiar with a "bacon-like" or "lardaceous" infiltration of several solid organs of the body, and especially of the spleen and the liver. Portal and Abercrombie described the morbid condition in the liver as a "*lardaceous degeneration*;" and Hodgkin and Bright described the same disease as an "*albuminous infiltration*." In 1842 Rokitansky was the first to give a clear account, and to describe in detail the "*lardaceous*" infiltration of the kidney with an "*albuminous*" transparent substance. The lesion so described constitutes his eighth form of "Bright's disease." But Rokitansky made no chemical examination of the infiltrated material. He simply assumed, from its general appearance, that it was of an *albuminous* nature, and he rightly recognised its pathogenetic relations to certain cachexiæ. Budd has described the disease as "*scrofulous enlargement of the liver*;" Oppolzer and Schrant, by the name of "*colloid*;" Baron by the name of "*carnification*;" and the pathologists of this country under the term of "*waxy degeneration*."

Such are the names, derived from appearances generally, under which the peculiar lesion has been described before microscopic examination demonstrated the condition of the structures implicated.

Chemistry and micro-chemical investigations have modified the views regarding the nature of the substance formed in this disease, and now and then have led to modifications in the nomenclature. Under this kind of inquisitive investigation it has been described, (1.) By Virchow under the name of "*animal amyloid*," he believing, from the behaviour of the new substance with iodine and sulphuric acid, that it must be classified with the vegetable carbo-hydrogens—cellulose and starch. (2.) Meckel retains the name of "*lardaceous*" or "*cholesterine disease*," believing that the essential character of the disease consists in the development of a peculiar fatty or lardaceous matter, of the nature of cholesterine. (3.) The more extended and definite examinations by Friedreich and Kekulé have shown

that the substance in its purest form more closely resembles the *albuminous principles* than any other substance we know of; and, (4.) Schmidt has arrived at the same conclusion. The question, therefore, is not yet definitely settled as to the exact nature of the substance into which the tissues are transformed, but the weight of evidence points to its being *albumen* in some form; and the albuminoid deposits in the spleen of children, so well described by Sir William Jenner, must be classed as examples of this lesion, and probably also the special lesions in *rickets*.

Investigations relating to lardaceous disease have taken especially three directions. Pathologists have endeavoured—

(1.) To trace the extension of the process throughout various tissues and organs of the body, and so to find out if it is an infiltration of a new substance or a degeneration of tissue.

(2.) To determine the essential nature of the material which characterises the disease.

(3.) To determine the conditions under which the disease is brought about.

Virchow first stated that the large Malpighian sacculi in the spleen (which, in some instances, looked like boiled grains of sago) were sometimes composed of a substance which gave the chemical reactions of cellulose, as seen in plants. Cellulose and starch are both vegetable constituents—"isomeric" forms of some common material; and what gave special interest to the observation of Virchow, was the discovery that cellulose is also an element in the covering or skin of the *Tunicata*—a genus of acephalous mollusca—and therefore not a constituent of vegetable organisation only. Dr. Robert M'Donnell, of Dublin, has also shown that the blood-vessels of the fœtus, at a certain stage of development, are of the same albuminoid matter. Thus the discovery of cellulose in animal tissue induced Virchow to look for it or its analogue—namely, "starch," in the human subject. He recognised it in the *corpora amylacea* of the brain. These contain a substance chemically related to starch or cellulose; and these bodies were first seen and named by Purkinje, who gave them the name they have, not on account of chemical characters, but because he observed them to be laminated like starch. Of these *corpora amylacea* there are two kinds, namely,—(1.) Mineral bodies with concentric circles more or less soluble in mineral acids; (2.) Others which assume a blue tint with iodine, and a violet colour on the subsequent addition of sulphuric acid. The relations of these two kinds to each other are still unknown. The first are the calcareous particles known as brain-sand; and both were at first described under the name of "*corpora amylacea*" by Virchow, which has led to some confusion. The term ought to be restricted to those bodies which, by physical and chemical characters, are assimilated to starch. The mineral bodies erroneously described as *corpora amylacea* are chiefly found in the cysts of the choroid plexus and in the pineal gland. On the other hand, the starch-like bodies have been found by Virchow, Rokitsansky, Scherer, Kölliker, Busk, and other observers, in the *ependyma* of the ventricles, the *septum lucidum*, the *fornix*, the *auditory* and the *optic nerves*, and also in the *prostatic ducts*. Concentric lamination of these bodies is not always present; nor is the re-action with iodine and sulphuric acid constant. For these reasons Virchow began to

examine those organs whose morbid state was described by the names already mentioned as having been given to the fatty or waxy spleen. He applied solutions of nitric acid, which, when hot, gave a yellow hue; he applied caustic ammonia, which gave a brown colour; and from behaviour with re-agents generally, he concluded that the substance was "ALBUMINOID" in its nature. Iodine and sulphuric acid were subsequently tried. Iodine alone gave a strong yellow-red; sulphuric acid being added, developed a blue colour, passing into a strong violet hue. An excess of acid destroyed the violet hue, causing a dark brown-red colour, passing into yellow. Meckel, subsequently to these observations of Virchow, came to the conclusion that there were four forms of this waxy material—that the basis of them all was a peculiar fat allied to cholesterine rather than to starch—that various saponaceous products are formed, ending in the development of cholesterine; and although he did not sustain his statement by anything like sufficient proof, he made the important discovery that it was the system of small arteries and capillaries which first suffered in this disease.

The inquiry into the chemical nature of the lesion becomes still more interesting when connected with the observations and discoveries of Bernard, Pavy, and others, on the "sugar-producing" functions of the liver, and on the material so formed, which may be separated by chemical processes, and which has been recently shown by Dr. Robert McDonnell to be a substance entering largely into the constitution of most of the tissues of the embryo (*Proceed. Royal Society*, Vol. XII., p. 476). The results of these inquiries bring the "starchy substances" of animals in very close physiological alliance, and also in alliance with morbid products. The material so found has been called indifferently "glycogen," "amyloid matter," "zoo-amyline," or "animal-starch." It owes its origin, not to any direct function of the organ, but its formation seems to take place almost immediately upon contact with albuminous matter, when this remarkable product is the result, and which may be obtained as a white powder. It seems capable of being produced in greatest abundance by the hepatic tissue; but its formation may proceed at any part of the vascular capillary system. If, therefore, it is thus formed normally, it may also be formed, retained, or transformed in a morbid way. In *diabetes* we have an instance of the transformation of the product into sugar discharged by the urine at the expense of the tissues at large.

The analysis of the pure matter is very defective. Such as it is, it shows the substance to be *albuminoid*, and combined with *nitrogen* rather than *starch*; and those who describe the re-action of *cellulose* and *starch* with iodine and sulphuric acid, seem only to agree with each other in giving singularly diversified descriptions of colour; which, perhaps, to those familiar with the writings of the late Dr. George Wilson and others on colour-blindness, may be accounted for. Such diversity may be explained in some measure, also, by the fact that the degree of concentration of the re-agents materially concerns the results; for, as Virchow correctly observes, the blue coloration is only got after a considerable period, and in practised hands, and it may pass from a bright purple to a very deep blue or even black colour; in fact, the blue-black colour is a result of the decomposition of the iodine solution,

by excess of sulphuric acid, throwing down the iodine, which blackens the tissue. Nevertheless, the action of iodine solution on the lardaceous tissue is peculiar and definite, independently of a blue colour. It is of the nature of a chemical re-action, which gives a hue different from the mere dyeing with the iodine, and which *suddenly* deepens in tone, from the moment it begins to take effect, to a deep brown-red colour. The appearance of such a chemical reaction is alone sufficiently characteristic. When this takes place with the solution of iodine *ALONE*, it distinguishes at once the substance from *cellulose* and *cholesterine*.

Morbid Anatomy and General Characters of Tissues affected with Lardaceous Disease.—The cut surface of an organ so affected has a semi-transparent appearance. It feels like a piece of soft wax, or of wax and lard combined (WILKS). It cuts into portions of the most regular outline, with sharp angles and smooth surfaces; and the thinnest possible slices may be removed by a sharp knife for microscopical examination without any special preparation. The tissue is abnormally translucent. Water, alcohol, and acids do not produce any change upon the transformed parts, which may be kept for a length of time without decomposition. The organs affected are increased in volume, in solidity, and in weight, absolute and specific. Anæmia is predominant; but the colour of blood or of tissue shines through the semi-transparent morbid substance.

Lardaceous disease is generally widely diffused; so much so, that a constitutional state of ill-health is always associated with its production; and in cases preceded by a local disease, such as caries of a bone, the lesion may be found in the adjacent lymphatic glands only (BILLROTH). This is the earliest appearance of the disease yet recognised in connection with local lesion.

The small vessels of the tissue—the more minute arteries in particular—are, as a rule, the first structures attacked. The coats of the blood-vessels become granular and thickened, apparently by exaggeration of their transverse fibres, and at last pellucid, transparent, and hyaline. Their calibre is reduced, and their cut section remains patulous. It is the transverse fibres of the middle or muscular coat of the vessels which first change. Each fibre-cell becomes a compact hyaline, pellucid, transparent particle, with an indistinct outline, and all the tissue involved becomes at last uniform, clear, and transparent. The diseased artery looks like a compact, homogeneous, silvery cord or thread, of a clear and glassy appearance, with a lustre like molten glass without polish, or like rough ice. This colourless, hyaline, diseased tissue is very tough, but not hard nor brittle, like the calcareous degenerate parts. All degenerations tend to obscure the original texture, by making it more opaque. This lesion, on the contrary, renders the affected tissue more transparent and pellucid. The specific cells of the functional parenchyma (when the disease affects a solid organ like the liver or kidney) next undergo the change, which finally spreads to the nutrient vessels amongst the connective tissue. The morbid matter seems to penetrate the coats of the minute vessels, and gradually works its way into the surrounding tissue; and the changes which thenceforth result vary according to the organ affected. In the solid viscera the lardaceous material remains about the vessels, and fills the

interstices of the texture. Thus, the organs so affected, especially the liver and spleen, increase in size, becoming hard, grey, and semi-transparent, as if uniformly infiltrated with wax. The kidneys, supra-renal capsules, and lymphatic glands, are all apt to assume the same firmness and wax-like translucency. In the spleen the new material exaggerates the Malpighian sacculi, till they are larger even than grains of boiled sago—more like boiled tapioca, as I have seen them in many instances. In the mucous membranes of the small intestines the vessels are similarly altered in appearance; but the exudation in this form of the disease, instead of being retained, is passed off by diarrhœa; or, if the stomach is affected, by vomiting (DICKENSON).

When a solution of iodine is brought in contact with the affected part, a very deep violet-red colour is produced. This deep red colour seems to be *alone* a sufficiently characteristic test of the existence of the disease, especially when in a few seconds the colour increases in depth from the moment it begins to take effect. The morbid material seems to have a strong affinity for the re-agent, "absorbing it readily, holding it tenaciously, and assuming its full colour," while the healthy parts take only a faint and superficial yellow tinge. Hence the contrast which the deep reddish brown of the morbid parts presents against the uniformly faint yellow of the normal tissue in which it may be placed (DICKENSON). It is of the nature of a chemical re-action which ensues between the iodine solution and the morbid matter in the part. The best test-solution is composed as follows:—*Twelve grains of Iodine* are to be dissolved with *twenty-four grains of Iodide of Potassium*, and mixed with *three ounces of Water*. Such a test-solution ought always to be at hand on making a *post-mortem* examination anywhere.

The elements of tissue in which lardaceous disease has been demonstrated are,—(1.) *Nervous system*.—Ligamentum spinale cochleæ: atrophied parts of brain and spinal cord: gelatinous softening, and tumors. (2.) *Spleen*.—Cells of the Malpighian sacculi and pulp: thickened walls of the arteries in all stages: the trabeculæ. (3.) *Liver*.—The hepatic cells and intralobular vessels, and intercellular tissue. (4.) *Kidneys*.—Malpighian tufts and afferent vessels, the walls of which become enormously thickened: areolar tissue in the vicinity of the papillary ducts. (5.) *Muscular tissue* of the heart and the uterus. (6.) *Blood-vessels* of the villi and mucous membrane of the alimentary canal. (7.) *Osseous tissue*. (8.) *Lymphatic glands*. (9.) *Old deposits* in serous membranes, having lost their fibrous character, becoming dense, more vascular, and semi-transparent, undergo this metamorphosis (GAIRDNER). (10.) *Tubercle* also becomes lardaceous (GAIRDNER). (11.) The *cancerous nodules* in a waxy liver also become lardaceous (GAIRDNER). (12.) In some cases of inflammation with exudation on the mucous membrane the *exudation* has become lardaceous (VIRCHOW). (13.) The *fibrine* of a hæmatocele (FRIEDREICH).

The extensive range of organs in which this remarkable lesion has now been demonstrated clearly shows that it cannot be regarded as one merely of local importance. Its occurrence seems rather to point to some general pathological state of which the lesion is the expression. In the first instance it is found more particularly affecting the functional capillaries

of the most important organs of the body—*e.g.*, the kidney, the liver, the spleen, the intestines, as well as the minute arteries of nutrition of those organs, and of the pia mater, bone, and lymphatic glands. The results of such a disease must therefore be sooner or later destructive—(1.) To the function of the invaded organ; (2.) To its nutrition; and we can only arrive at a correct pathology of this lesion by a close observation of the circumstances, condition, relations, and symptoms under which it appears. These must be studied especially in relation to the functional or physiological anatomy of the organs implicated. As yet the lesion has been recognised with certainty only in the dead-house. There it has been found associated with certain diseased states; and all the cases agree in this particular, namely, that the constitution of the patients has been broken up by ill-health (*cachexia*) of some considerable duration before death. So it has been amongst the soldiers dissected at Fort Pitt and at Netley; and the following statement is a summary of morbid conditions with which lardaceous disease has been associated, or upon which it has been engrafted, and in the order which has furnished the greatest number of cases of lardaceous disease:—

(1.) *Diseases of the bones*, especially *caries* and *necrosis* in scrofulous subjects. *Rickets* also leads to the lardaceous liver and spleen, as observed by Glisson, Portal, Rokitsansky, Lambe, Loeschner, Frerichs, and Jenner. (2.) *Syphilis*, especially in its ulcerative forms, the *cachexia* having been prolonged. Syphilitic children have been the subject of it when newly born. (3.) *The malarious cachexia*, especially *intermittent fever*. (4.) *Mercurial cachexia* and *marasmus*. (5.) Pulmonary and intestinal forms of *tubercle*. (6.) *Albuminuria* and *anasarca*. (7.) *Diseases of large arteries*.

It has been recently urged by Dr. Dickenson, that the lesion is always the result of extensive purulent discharges of long duration. He believes that its mode of origin has to do with the removal of alkalies from the system, which long-continued suppurations tend to do, leaving a relative increase of fibrine. Five-sixths of the cases recorded by him were connected with suppuration. Hence he proposes the term *depurative infiltration* as significant of its pathology. Pus is an albuminous fluid rich in alkaline matter, containing about one per cent. of alkaline and earthy salts, in the proportion of ten of alkaline to one of earthy salts. Next to suppuration, *albuminuria*, when connected with *nephritis*, is the most frequent antecedent of lardaceous disease; the long-continued discharge of albumen carrying alkali with it.

As to the origin of the lesion, Frerichs has propounded two questions, namely,—(1.) Is the lesion due to deposits from the blood of the albuminoid matter in some primordial form, and which is generated in the blood in consequence of a local disease, such as *caries* of the bones or other suppurative processes? (2.) Is the albuminoid matter developed locally in the affected tissue by the transformation of the tissue into albuminous matter?

Arguments are put forward by Virchow and Frerichs to show that the lesion may be due to a deposit from the blood; and Dr. W. H. Dickenson comes to the conclusion that the deposit essentially consists of an exudation of a peculiar material differing from the proper constituents of the body. He considers the substance essentially fibrinous, and so deposited

in consequence of the absence of alkali necessary to hold it in solution. Hence he names it "dealkalinised fibrine," and not a form of albumen. He believes it to be fibrine, rather than albumen, on account of the strong tendency it shows to undergo contraction after its deposition; but this is against the weight of chemical evidence as already given. Dr. Dickenson further argues that it becomes converted into *fibroid* tissue as a coagulum in the arachnoid, or as vegetations on the valves of the heart. In certain cases, also, it is identical in appearance and re-action with the *hyaline* casts of the kidney tubes, believed to be fibrinous. Fibrine can be also converted into the lardaceous substance by removing the alkali it naturally contains, or only neutralising it. Conversely, if potash or soda be added to the morbid matter, it ceases to give the characteristic re-action with iodine. It will not decompose the colour of sulphate of indigo, as healthy tissues containing alkali will do. Analyses of lardaceous livers show a diminution by one-fourth of alkaline salts, and the earthy salts exist in larger quantity than in health.

There is also undoubted evidence to show that in some parts it is of the nature of a degeneration. For—(1.) In cases where the lesion follows affections of the bones, the lymphatic glands adjoining the diseased bones are implicated before the kidneys, liver, or mucous membrane of the intestines. (2.) General causes of ill-health (cachexia), pointing to impoverished blood, are in operation, and organs situated in different parts of the body are simultaneously affected. (3.) The fibrine of the blood itself has been observed to undergo the degeneration; for Friedreich found a substance which gave the amyloid re-action with iodine in the old fibrinous layer of the sac of a hæmatocele.

In this remarkable disease an acquaintance with a new fact in pathology must be recognised—*i. e.*, since 1854—associating itself with grave constitutional disease, and distinguished from every other morbid condition hitherto known, by the physical, chemical, and physiological characters just described.

The Clinical History of Lardaceous Disease is remarkably deficient. The effect of the lesion is to interfere with the special function of organs and nutrition of parts; and the injurious effects are the more marked as the lesion extends through many important organs. For example, hepatic cells cease to take part in the formation of sugar or the secretion of bile. Blood-vessels lose their power of transmitting fluid through their walls, and become impervious as to their canals. Hence those who suffer from lardaceous disease have an appearance of *general ill-health*, denoted by paleness of the surface, by symptoms of anæmia, hydræmia, or by leukæmic affections of the blood; and the more so as the constitution is enfeebled by such morbid processes as suppurative ulceration of bones, syphilis, tuberculosis, albuminuria, or malaria. The sequence in which the different organs become affected is uncertain. In most cases of caries and necrosis the kidneys seem to be first attacked after the lymphatic glands. In cases of intermittent fever, it is usually the spleen which is first affected; and generally it seems rare to find several or all the organs affected to the same extent.

Signs or Symptoms associated with Lardaceous Disease discoverable during Life.—On these points data are wanting upon which to found any

statement. The pathological change is of so recent discovery, that well-recorded cases, terminating in death, with verification of the symptoms by *post-mortem* inspections, are very few indeed. There is no subject, therefore, more full of interest, or one more likely to repay close observation and well-directed pathological inquiry, than the diagnosis of lardaceous disease. Cases in hospital ought to be carefully noted (especially such ambiguous cases as those where marasmus, anæmia, or dropsy are *primary* symptoms, and which are not to be accounted for even after the blood has been examined microscopically during life), and the condition of the liver, heart, spleen, and lymph-glands carefully inquired into without evincing signs of disease. In a remarkable case recorded by Friedreich and Kekulé, and quoted in the *Medico-Chirurgical Review* for October, 1860, *diarrhœa* and *vomiting* were of frequent occurrence, with a systolic murmur of the heart, and high-coloured and albuminous urine, having a specific gravity of 1·019. The patient, a female, after suffering from tertian ague for twelve months, became dropsical and emaciated. The intestines throughout, the stomach, the colon, the jejunum, and especially the capillary vessels of its villi, were affected, as well as the vessels of the kidneys. The urine should be watched as to albumen or deposits, and its amount in relation to body-weight should be recorded. When albumen appears, it goes on gradually increasing; and hyaline casts increase with the increase of albumen. Professor Stewart, of Edinburgh, records twenty cases and *nine dissections* in cases of Bright's disease, where he considered lardaceous disease to have been present (*Edinburgh Medical Journal* for February, 1861). He records that large quantities of urine were passed in the early stage of supposed waxy degeneration, and of a specific gravity from 1·005 to 1·015. In all the cases there was a striking general correspondence in the other symptoms; and Dr. Stewart thinks that from this similarity of symptoms, and from other considerations, he is warranted in believing that lardaceous disease existed in the *eleven* cases that did not die. The history of all Dr. Stewart's cases is markedly different from that of the fatty kidney which Dr. Bright figured in his first plate, and illustrated in his first case. Almost all of the cases were associated with long continued wasting disease; and it has been long known that the form of renal affection accompanying phthisis, syphilis, and other wasting maladies, is this lardaceous disease. Of the twenty cases related by Dr. Stewart, six were associated with phthisis, six with syphilis, two with caries, two with intemperance, one with cancer, one with chronic rheumatism, and two with no particular disease.

The lesion is much more common than is generally supposed. It has been observed very frequently amongst the soldiers who have been dissected at the Military Hospital for Invalids, formerly at Fort Pitt, and now at Netley. The microscope and iodine test can alone determine its presence; and without microscopic examination the absence of the degeneration cannot be determined. For a detailed account of lardaceous disease in the various organs, see the descriptions given under LOCAL DISEASES.

CYST.

LATIN EQ., *Cystis*; FRENCH EQ., *Kyste*; GERMAN EQ., *Cyste*;
ITALIAN EQ., *Ciste*.

Definition.—*A sac, bag, or sharply defined cavity (to the exclusion of capsules or sheaths forming round foreign bodies, extravasations, or capsules of parasites) filled with fluid, or with some substance which may be regarded as entirely, or for the most part, its product, whether as a retained secretion or as an endogenous growth or extravasation.*

Pathology.—Many theories have been put forward to explain the formation of cysts; but no single hypothesis has yet been sufficient to account for their formation in all situations where they have been found. It has been extensively taught that the structure of a cyst consists of an excessive augmentation of volume of the alveoli of the areolar tissue, composed of condensed and modified filamentous tissue. Bichat urged many objections to this view, and held that cysts, being in many respects analogous to serous sacs, they ought to have the same origin. He held that cyst-growths were aberrant forms resulting from

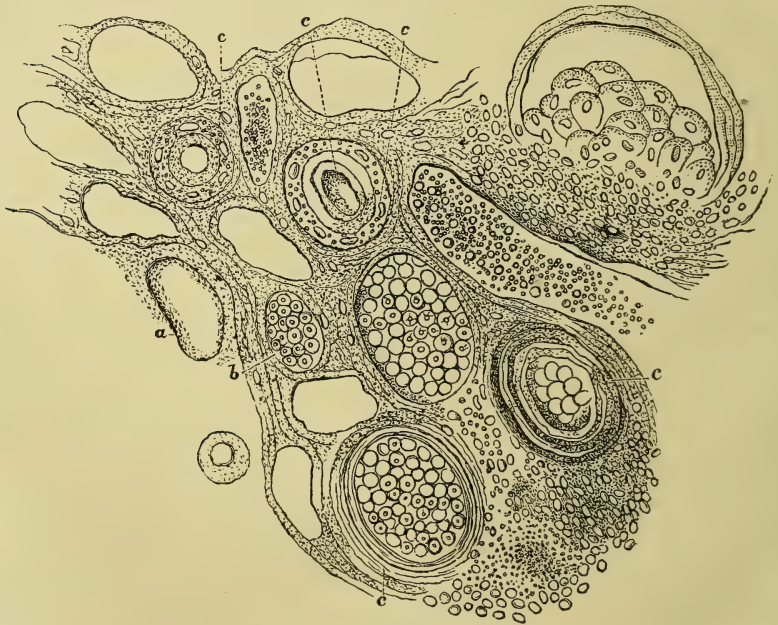


Fig. 16.

unnatural growth of germs in and amongst areolar tissue, and that the contents increase with the enlargement of the cyst by growth. Rokitsansky subsequently followed very much the idea of Bichat, regarding the cyst, from its organisation and secretory function, as a definite hollow structure,

whose essential rudiment is a definite functional elementary germ. But there are no doubt some cyst-formations due to the dilatation and growth of natural ducts, sacculi, or follicles (cysts of retention), as well as others which form by the enlargement and fusion of areolar spaces in connective tissue.

Thus there are three modes in which cysts may be formed, namely:—

(1.) Cysts which are a substantive new growth, having a distinct elementary groundwork, derived from cells or the nuclei of cells, pursuing a morbid course of growth from their origin, and reaching an enormous development.

(2.) Cysts which are formed by obstruction, dilatation, and growth of natural ducts or sacculi. Examples of such cyst-formations are seen in sebaceous or epidermal cysts, formed from enlarged hair follicles, and the cysts formed by dilated mucous tubes; also certain cysts, containing milk, from enlarged parts of lactiferous tubes; ovarian cysts from overgrown Graëfian vesicles; and, lastly, cysts formed from dilated blood-vessels shut off from the main stream.

(3.) Cysts formed by enlargement and fusion of the areolar spaces in connective tissue. The tissue of the wall of such cysts becomes condensed, and the inner surface secretes fluid like a serous sac.

Although the observations of Rokitsansky and Simon point to the growth of cysts in the kidney from the original primary cell-elements, yet the observations of Drs. Gairdner and George Johnson equally explain their formation by local obstruction of uriniferous tubules, and their dilatation into cysts above or between the points of obstruction (cysts of retention). But whatever may be the source of their formation, we have yet to learn, as Sir James Paget specially notes, why they tend continually to grow.

Figs. 16 and 17 are Rokitsansky's representations of the minute structure of cysts of the kidney. They represent "nests" of delicate vesicles, from a size just visible by $\frac{1}{3}$ to $\frac{1}{4}$ lens to the size of a millet seed, imbedded in a red grey whitish substance;—proliferous cyst-formations from the cortical substance of the kidney, as a sequel to Bright's disease. The two figures illustrate well Rokitsansky's history of proliferous cyst-development, and at the same time what he understands by the often-occurring expression, "alveolar type or arrangement."

In Fig. 16 we have the cyst in all its phases; *a* is a simple cyst, arising out of the expansion of the elementary germ, first into the nucleus, from this into the cell,

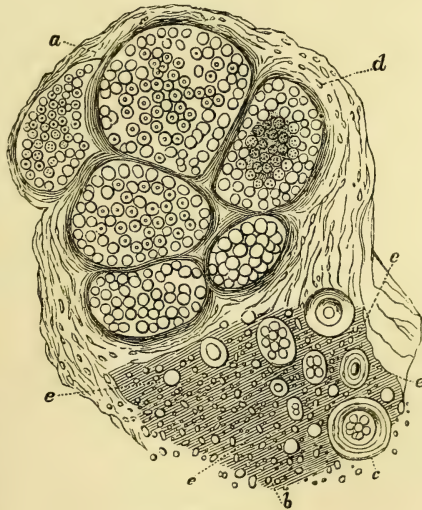


Fig. 17.

and progressively into the cyst. But it has remained barren, and contains only a diaphanous viscid serum within a simple cyst-membrane; *b* represents a parent cyst, the *early* history of which accords with that of the barren cyst; within it, however, new granules have formed, and gradually become developed into vesicles or cysts containing other nuclei, until the parent cyst has become replete with them, and, from being spherical, they are rendered polyhedral by mutual compression. In an adjoining parent cyst, many of the filial cysts have remained barren; others contain nuclei in the act of splitting; *c, c, c, c,* represent another form of development of the parent cyst. Here, again, the parent cyst has gone through the same phases, from the elementary granule upwards. But as the cell dilates into the cyst, a granule forms centrally to the latter, and expands into a filial cyst, centrally to which a third granule opens out in the same manner; and so on. These intra-cystic cysts in their dilatation ultimately close upon the parent cyst, forming secondary, tertiary, and ulterior layers, to which an external fibrous layer is generally added out of the surrounding blastema. Or this fibrous coat occurs in the *alveolar* shape. Fig. 16 affords several examples of this. It is, however, better seen in Fig. 17, where *a* is the fibrous sheath in progress of development out of *d*, the elongated and caudate nuclei coursing around the parent cyst or aggregation of parent cysts. They eventually break up into the requisite fibres; *e* represents the point-molecule, within an amorphous blastema, out of which the nuclei (*b*) form. They are at first spherical, afterwards elongated, and ultimately broken into fibrillation. This constitutes the "alveolar type or arrangement."

Rokitansky teaches that the *nucleus* grows to be the cyst, whether it be *simple* or *barren*; and that the outside layers of the cyst-walls, if they are complex and thick, are made up of endogenous growths, of nuclei, of cells, or of any other structures.

Thus a classification of cysts has been conveniently arranged into;—(A.) Simple or barren cysts, which contain fluid or organised matter; and, (B.) compound or proliferous cysts, which contain variously organised bodies (PAGET.)

(A.) The simple or barren cysts may occur singly or many together (when they are called "multiple cysts"), and contain a fluid like that of a serous sac (mammary cysts, choroid plexus cysts, synovial cysts). Some are full of blood, or colloid stuff (glue-like), or other peculiar or abnormal fluid. Others (transitional between barren and proliferous cysts) contain specific secretions, such as milk, semen, mucus, saliva; and they are thus named, according to contents, as *lactiferous*, *seminal*, *mucous*, *salivary*, *colloid*, *sanguineous*, *synovial*, *serous*. The simple or barren cysts contain one or other of the following materials:—(a.) *Gaseous cysts* (examples of *pneumatoses*, see p. 114, *ante*) are mentioned by Sir James Paget, on the authority of Hunter, Jenner, and Cavendish, and the preparation preserved in the Museum of the College of Surgeons, No. 153-4; but beyond the description of Plate xxxvii. in HUNTER'S Works, Vol. IV., p. 98, nothing is known concerning such gaseous cysts. (b.) *Serous cysts*, or *hygromata*, are the most frequently seen. They include nearly all which have thin liquid or honey-like contents, of a yellow or brown colour. They are most frequently found near the secreting or vascular

glands; and there is scarcely a part of the body in which they may not be found. So common are they in and amongst gland-structures, that they are believed to arise from the same germinal elements of membranes that furnish the perpetual growth of glandular or secreting elements. Thus, in such sites they are held as examples of perverted epithelial or gland-cells. But they are no doubt independent of such origin, as in bones, connective tissues, muscles, nerves, and fibrous tumors, where their origin is quite independent of gland-cells. So complicated are the contents of some cysts, as in bone, that perfect ciliated epithelium has been observed in them (WEDL).

Serous cysts occur chiefly in the neck, the mammary gland, and the gums. In the neck they are sometimes described as "hydroceles of the neck." They may be single, or multiple to the extent of hundreds, having many cavities, either separate or communicating. A case of this description proved fatal at Netley Hospital in November, 1870. The patient was a young soldier, eighteen years of age, and of five years' service. The left side of the neck was enlarged from many cysts, which had been opened into during life, and which, at death, were in a state of active suppuration, so that the original connection of the cysts was not traceable. On the right side of the neck a smooth tumor, about the size of a turkey's egg, occupied the lateral region, extending beneath the *platysma myoides*, and lying between the fibres of the deep muscles of the neck, from an inch and a half below the ear to an inch and a half above the clavicle. Commencing small cysts were seen posterior to the tissue of the left tonsil, and the pressure of the diseased parts, especially from the left side, caused extensive cedema of the pharyngeal and laryngeal mucous membrane. The cysts had no connection either with the thyroid or lymphatic glands of the neck. Another mass of cystic growths occupied the entire pelvis, and was apparently divided into four large lobes. These growths pushed the bladder over to the right side, pressing upon it anteriorly, and from the left. There was also another development of large cysts in the soft parts about the left hip and upper part of thigh. Some of the cysts had been opened in the thigh by a trocar, and had commenced to suppurate. Those cysts which were entire contained clear serous fluid, highly albuminous, of a straw colour, and having a specific gravity of 1.017. The fluid was frequently examined during life, several pints having been removed from time to time, but neither during life nor after death were any evidences of parasitic development (such as echinococcus) discoverable. The cysts were obviously developed in and amongst the areolar tissue of the neck, pelvis, and thigh; but the origin of the cysts is extremely obscure. It is most probable they had their origin in the corpuscular elements of the connective tissue or juice canal system, which permeates that tissue. The subject of this cystic disease had been considered to be suffering from scrofula, and had been 129 days in hospital at Gibraltar. The exciting cause of the swellings was attributed to sleeping in a wet tent, when encamped with his regiment at Windmill-Hill. Thence he was invalided to Netley, where the cystic nature of the tumors in the neck and hip was recognised. On admission, there was enormous swelling of the left neck from the ear to the clavicle, and from the region of the left parotid gland to the *ligamentum nuchæ*. Several small openings existed, from which a

thin watery discharge exuded. He complained of pains in the left hip, simulating rheumatism, accompanied with a moderate general swelling of the joint. Respiration sounds were normal. He was a spare anæmic lad, badly nourished, and of unhealthy aspect. For two months after admission to Netley, the swellings in the neck progressively increased in size, and the patient continued to lose flesh, and became more and more cachectic. A puncture was made with a small trocar in two places, by Staff Surgeon-Major Mackinnon, C.B., and serous fluid, to the extent of six ounces, escaped. The fluid, however, accumulated again very rapidly; and ten days later, about half a pint of blood-serum was removed and carefully examined for parasite developments, but with a negative result. At this time swelling about the hip-joint was observed. It was of a uniformly rounded contour; and the circumference of the upper part of the left thigh was five inches in excess of the corresponding part of the opposite limb (right side, $15\frac{1}{2}$ inches; left side, $20\frac{1}{2}$). There was much tenderness on pressure over the joint; but pressure on the condyles of the femur, in an upward direction, did not cause pain in the hip-joint, neither was there lengthening or shortening of the limb. Five days later, sixteen ounces of fluid were evacuated from the facial tumor, and eleven ounces on the following day. After another five days, Mr. Mackinnon made an incision over a tumor at the lower part of the neck, and exposed a dense glistening membrane—the wall of another cyst—from which serous fluid escaped as from the others. Five days afterwards, another tumor was observed on the right side of the neck, rounded, smooth, and fluctuating, and this gradually continued to increase till death. It was left unopened. The openings by trocar into the cysts in the left side of the neck, led to a depth of several inches; and two of the cysts communicated; two others opened into were isolated. Much pain was subsequently experienced in the region of the left hip, and in eighteen days the circumference of the thigh had increased three inches. An opening was now made by trocar into one of the swellings over the hip, and a pint of fluid evacuated; five days later, twenty ounces more escaped. The fluid was of the consistence of very thin arrow-root, and with a tendency to stringiness between the fingers, consisting almost entirely of an albuminous solution, coagulating into a solid white mass on boiling. Soon after, a large tumor, firm and round, containing fluid, was detected in the pubic region, giving a sensation to the hand similar to a contracted uterus. It was tender on pressure, and much pain was experienced on micturition. From the excessive daily discharges, combined with the acute pain in the hip, and the onset of laryngeal irritation from œdema and pressure on larynx and trachea this case eventually terminated fatally.

In young children such serous cysts are often congenital (*hygroma colli cysticum congenitum*). Some are connected with the thyroid gland, others are transformations of vascular tumors, such as erectile vascular growths or nævi (PAGER).

Cysts in or near the gums, with contents of a thick honey-like consistence, and generally sparkling with crystals of cholesterine, are usually found lying behind the reflection of the mucous membrane from the gum to the cheek. From their tough thick walls in that situation, they are apt to simulate disease of the antrum.

Cysts in the mammary gland are often due to dilated ducts, portions of which assume a cystic form. They may contain milk. Mr. Birkett records numerous cases of this kind, from one of which he evacuated ten pints of milk. Some contain the remains of milk, such as fat and epithelial scales; or they may be filled with transparent watery fluid, uncoagulable. More commonly they contain serous fluid, pure or tinged with blood. They may also originate (like those of the kidney) independently of the gland-tubes (BIRKETT, PAGET).

Sir James Paget has laid down the general rule that the cysts which contain the simplest fluids, and which have the simplest walls, are apt to grow to the largest size.

(c.) *Synovial cysts* acknowledge three methods of formation:—

(1.) From widening of spaces in areolar tissue, forming, by condensation, bursæ as the result of pressure.

(2.) Cystic transformation of cells inclosed in the fringe-like processes of the synovial membrane of the sheaths of tendons, such as those near the wrist-joint, forming *ganglions*, as they are called. They resemble the cysts of the choroid plexus, which grow from the villi at the margins of the plexus (ROKITANSKY).

(3.) The mouths of subsynovial follicles, which normally open into the cavity of the joint, become obstructed, and the follicles undergo such dilatation as converts them into subsynovial cysts (GOSSELIN, quoted by PAGET). Such synovial cysts vary as to contents, being sometimes serous, gelatinous, or honey-like.

(d.) *Mucous cysts* are formed in connection with simple mucous membranes, or ducts of mucous glands, such as those about the *cervix uteri*, giving rise to *Nabothian cysts*; or connected with Cowper's glands in the male, forming *Cowperian cysts*. Similar cysts form in connection with cutaneous follicles. Examined microscopically, such cysts contain epithelial scales, free fat, tables of cholesterine, crystals of triple phosphate, and small hairs in various proportions.

Many abscesses projecting into the vagina have their origin in glands near the orifice (BARTHOLINO), which may become cystic (PAGET).

Mucous cysts are recorded also in the antrum; in the mucous membrane of the stomach and other parts of the alimentary canal; in the uterus; in the posterior wall of the trachea, forming cystic tumors lying between the trachea and œsophagus (VIRCHOW); in the back of the epiglottis, and covering the upper orifice of the larynx (DURHAM).

(e.) *Ranula* is an analogous affection of the duct of the sublingual gland.

(f.) *Sanguineous cysts* contain blood, and are probably the result of hæmorrhage into the cavities of serous cysts, like the transformation of *pericarditis* into *hæmorrhagic pericarditis*, or of a *hydrocele* into a *hæmatocoele*. Such blood-contents are generally coagulated or thick. Others have their origin in vascular *nævi* (extravasation cysts).

(g.) *Cysts containing oil or fat* are rare, except as the residue of fatty degeneration of other matters.

(h.) *Colloid cysts* embrace cysts containing all those morbid materials described as “pellucid,” “jelly-like,” “flickering,” “half-solid,” “glue-like.” Such material is common in the cysts of bronchoceles and in kidney cysts.

The density of such contents varies from that of serum to that of a firm jelly, and in colour it may be of any hue.

(B.) The second kind of cysts are the proliferous or compound cysts, and are so named from the occurrence of secondary growths in the interior of the original cysts—cysts growing within cysts, or upon their walls, as in complex ovarian cysts. These secondary growths may hang pendulous from the walls; and immense proliferous power exists in ovarian cysts from Graëfian vesicles. On this subject the student must consult Mr. SPENCER WELLS' treatise *On Diseases of the Ovaries*.

The mammary and thyroid glands are also often the seat of similar proliferous cysts; and there are proliferous cysts described by Sir JAMES PAGET as recurring and cancerous; while there are others developing skin structures (sebaceous), and others developing teeth (dentigerous).

For very interesting details regarding these, the student must consult Sir JAMES PAGET'S *Lectures on Surgical Pathology*, a work which has mainly furnished the materials for this account of cysts.

PARASITIC DISEASE, AND PARASITISM.

LATIN EQ., *Morbus Parasiticus*; FRENCH EQ., *Maladie Parasitaire*; GERMAN EQ., *Parasitische Krankheit*; ITALIAN EQ., *Malattia Parasitica*.

Definition.—Forms of disease in which a great variety of lesions and of functional disturbance are brought about by the presence of ANIMAL, PLANT, or BACTERIA life, finding a subsistence within or upon some tissue, organ, or surface of the human body, or of other animals or plants.

Pathology.—The parasitic origin of disease, or the association of parasites with disease, challenges consideration from several points of view, and in a great variety of relations; and mainly embraces a consideration of—(1.) The natural history of the individual parasites, not only as independent, full-grown, mature living things, but also as immature forms of life. These are two important distinctions to be always observed in the identification of them, namely—the mature and the immature forms. (2.) The specific lesions, or forms of structural or functional diseases, constantly found associated with parasites—the influence which parasites exercise on the life of the animals or plants in or upon which they live.

Again, in following out the inquiry under these several heads, the pathological interest of the subject is extremely varied and extensive, following several distinct directions of inquiry. For example, there at once arise the very important questions for investigation:—(1.) How far are parasites the actual originators or cause of disease or injury? (2.) How far do any of them act as morbid poisons, or as the vehicles or carriers of morbid poisons? (3.) How far are any of them constant or characteristic accompaniments of certain specific diseases?

The natural history of the organic forms to which the name of parasites has been given, embraces a consideration of them under the three following heads or classes:—

I. Parasites of the animal kingdom:—*Entozoa* and *Ectozoa*—examples

of which are the *Round-worms*, such as *ascarides* and *filaria*; the *Tape-worms*, *hydatids*, *cysticerci*, *distoma*, *lice*, *sarcoptes*, and the like.

II. Parasites of the vegetable kingdom:—*Entophyta* and *Epiphyta*—examples of which are *thrush*, *sarcinæ*, *tinea*, *favosa*, and the like.

III. Parasites of extremely minute organic forms, alike claimed by botanists and by historians of animal life—*Bacteria*. We have but very recently found out that this third form of parasitism is constantly invading our bodies, and attacking the living protoplasm by which the work of life is carried on in our tissues—they are constantly present to us—constantly mixing up their life with ours (SANDERSON). But the same may be said of all parasites. They are constantly present with us; and although the subject may be extremely uninviting, it is nevertheless of great interest and importance, and we cannot wisely remain in ignorance of the nature of parasites, on account of the direct and immediate bearing of their origin and existence on practical questions in Pathology and Hygiène. Those last mentioned—namely, the bacteria group of parasites—at present occupy a very large place in the medical literature of this country, of Germany, and of France. In this country Lister was the first to indicate their scientific importance as bearing on the treatment and pathology of wounds and external injuries; and since then, especially in Germany, the belief has risen up that certain specific diseases (of the General Diseases, Class A)—especially scarlet fever, enteric fever, small-pox, cholera—may be due to the growth and activity within the blood and tissues of the human body of these low forms of organic life. But as yet it is a mere belief—a theory, in fact, of disease.

It has also been shown that there is a varied series of putrefactive processes, each of which has its special and appropriate bacterium as its cause, and in the absence of which the putrescible substance remains perfectly sweet.

Thus in some putrefactions one kind of smell and correspondingly foetid chemical substance is produced, and in other putrefactions other such foetid substances of another distinct kind are produced. In some other cases the special kind of bacterium produces a vivid colouring matter—red, green, or blue. Hence smell-producing, colour-producing, and disease-producing bacteria, are varieties now capable of recognition. The so-called disease-producing bacteria are said to have this effect—that their life and multiplication in the blood and other fluids of the tissues produce poisonous (chemical) conditions in these fluids to the subversion of life or of health. Bacteria having thus been detected in the blood of certain diseases in great quantity, a justification has been given to the theory which attributes the *origin* or *fatal termination* of those diseases to them. The terribly fatal disease known as *malignant pustule* in man is accompanied by, if it is not also probably caused by, enormous numbers of a minute bacterium in the blood. Also *septicæmia*, or *pyæmia* (which is a putrefaction of the blood following certain putrefactive processes in wounds, and fatal dissection wounds), is equally connected with the presence of swarms of bacteria in the blood.

From this short summary of the comprehensive range of the pathology of parasitism, it will readily be seen that the inquiry takes in a very wide

field. Every variety of life has its characteristic and peculiar parasites. Plants, as well as animals, have their peculiar parasites and parasitic diseases. The mistletoe is a familiar example of a *vegetable parasite*; and the oak apple, or gall-nut, is a familiar example of an *animal parasite* affecting a plant.

It is in many instances capable of experimental proof, that some of these parasitic diseases (vegetable as well as animal) may be transmitted or communicated indifferently from animals to man, and from man to animals. The *tape-worms*, the *encysted*, *vesicular*, and *round worms*, are examples of parasites intercommunicable among animals; and *Tinea*, from the "*Dartre tonsurante*" of the horse, ox, or cat, having been communicated from these animals to man, are instances of *vegetable* parasites intercommunicable among animals. It may also be that the so-called *blights* of plants, or the causes of them, are also communicable to animals and to man. We know that various species of "ergot" give rise to a specific form of disease—Ergotism.

The records of history furnish numerous examples of periods of *blight* in the vegetable kingdom, associated with epizootics among the lower animals, and with epidemics affecting the human family. (See Sir WILLIAM WILDE'S *History of Ireland*, compiled in connection with the census taken in 1857.) The relative connection of these events has scarcely yet attracted the attention of pathologists, in human or comparative anatomy. Here, indeed, is a wide field for investigation—a territory almost yet unexplored. The medical service of Her Majesty's British and Indian armies gives golden chances for observation, if the chances are seized at the moment, and the observations connected with facts already known. To the more salient of these facts the attention of the student is here directed.

Since the beginning of the present century, when Rudolphi published his systematic work on the entozoa (1808), almost every year has contributed new and important facts, which render the subject of *Parasitic diseases* one of increasing interest to the pathologist and the physician. The subject abounds with most puzzling riddles in natural history and pathology, especially concerning the reproduction, the development, and the propagation of parasites. So long ago as 1691 the independent nature of such structures as the "*hydatid cyst*" was established (TYSON, *Phil. Trans.*, CXCIIL, p. 506); and it was stated by Pallas, in 1766, that all the cystic worms were forms of tape-worms; but it was not then known how their generation and propagation were effected. For a very long time the received doctrines regarding the generation and development of living beings were tacitly set aside in behalf of such "existences." They were believed to arise spontaneously, just as bacteria are believed now by some to arise spontaneously. Inquiry was thus set at rest, curiosity seemed satisfied, or investigations followed a fruitless direction—as when observations were made on such cysts, in the hope of discovering in them some evidence of the existence of organs of generation, or evidence of some process of generation analogous to what prevails in other animals. Ova were looked for, and organs of generation were looked for, where neither ova nor organs of generation existed. The calcareous particles visible in the tissues of those animals were at one time mistaken for eggs,

and described as such, in the membrane of the *Cysticercus* (1841). At last, in 1842, a great insight was obtained regarding the nature of the generation and development of these and other parasites, by the publication of facts which showed that amongst a certain class of minute *Cercarie* (worms of a microscopic size found in stagnant water), the generation of them was carried on through a series of broods produced from one parent, each brood differing from the parent and from each other. The discovery of this fact was due to Steenstrup. He described the phenomena under the name of "alternation of generation" amongst these *Cercarie* which ultimately live within the body of different mollusca (*Planorbis* and *Lymnæus*).

These observations gave quite a new direction and impetus to investigation; and Steenstrup himself foretold that the hydatid cysts would be proved to be undeveloped tape-worms, each cyst capable of producing a tape-worm after its kind. This view was at once taken up, and has now been independently worked out by many well-known observers.

But Kuchenmeister and Von Siebold were the first to *prove by experiment* that the hydatid or vesicular worms were the young or larval states of tape-worms; and they demonstrated what must now be regarded as elementary facts in the history of parasites and parasite diseases, namely:—

- (1.) That each parasite had an independent life of its own.
- (2.) That most animals have each their own peculiar parasites; that even parasitic animals are themselves infested with parasites.

" So naturalists observe a flea
Has other fleas on him to prey,
And these have other fleas to bite 'em,
And so proceed *ad infinitum*."

- (3.) That some parasites pass or migrate from the body of one animal into that of another (including man), or from one part of the same animal to another cavity or viscus in it. Such migrations are required for the introduction of the entozoa or their ova into the animals they inhabit, and where they undergo those series of changes about to be described, by which they reach maturity.

- (4.) That thus, through food (vegetable or animal) or drink, or both, or by bathing in impure water, entozoa pass into the human body, finding their way into the most delicate tissues, as minute ova or embryos, or as already fecundated females, like the guinea-worm.

- (5.) That they undergo progressive changes of development towards maturity in each of the new localities where they find subsistence and protection.

These five statements are elementary facts in parasitic science; and the student of Medicine cannot now rest satisfied with the mere knowledge of the general appearance of these so-called "worms" as they are found in man and animals. It behoves the physician, and especially the sanitary officer, to ascertain their origin, their source, and their mode of entrance into the body they inhabit. The easy but unsatisfactory hypothesis of "spontaneous generation" can no longer be entertained. On the contrary, it is now clearly established that all the parasitic entozoa with which we

are acquainted are produced (more or less directly) from fecundated ova. The general and minute anatomy of these "worms" must be studied, as well as their modes of generation, reproduction, and phases of progressive development; the metamorphoses of their individual forms; and their transmigrations from one animal into another. We must become acquainted with their existence even in and upon plants, as well as in other animals besides man, especially in such animals or plants as constitute the food of man—fish, flesh, fowl, mollusca, and crustacea,—and especially also all fresh-water plants, or plants which grow on moist ground, water-cresses, lettuces, celery, &c.

But domestic animals which are not generally eaten, but which, being the companions of man, come, like him, to be infected with parasites, and so tend to promote the propagation of parasitic disease in man and other animals. These domestic animals, especially cats and dogs, therefore, require attention, as to their feeding and habits.

A knowledge of details relative to the generation and reproduction of parasites is absolutely necessary in order to appreciate the nature of parasitic diseases. Indeed, without such knowledge no advance is likely to be made in the prevention of the parasitic diseases. It is this kind of knowledge which has recently led to most important practical results in the history of animal parasites; which most of all seems capable of extending our knowledge of parasitic diseases, especially in relation to human pathology, to the rational treatment of them, and especially their prevention.

Parasites of animal organisation exist in man and animals in every grade of development; and the first lesson for the student to learn is,—*how to distinguish entozoa which are SEXUALLY COMPLETE, from those parasitical productions which are DESTITUTE OF SEXUAL ORGANS, which are IMMATURE LARVÆ or NON-SEXUAL PARASITES, but which have long been regarded as distinct animals.*

The following is a classified list of Human Parasites, with some additions to that given by the Royal College of Physicians of London, in the Appendix to their Nomenclature, p. 232 :—

HUMAN PARASITES.

SUBDIVISIONS.

1. ENTOZOA.
2. ECTOZOA.
3. ENTOPHYTA AND EPIPHYTA.

ENTOZOA.

CLASSES.

- A. CÆLELMINTHA. *English synonym*, Hollow worms. *Definition*: Worms with an abdominal cavity.
- B. STERELMINTHA. *English synonym*, Solid worms.
- C. ACCIDENTAL PARASITES. *Definition*: Internal parasites, having the habits, but not referable to the class, of entozoa.

CLASS A, CŒLELMINTHA.

1. *Ascaris lumbricoides*. (Linnæus.) *Habitat*: Intestines.
2. *Ascaris mystax*. (Rudolphi.) *Habitat*: Intestines.
3. *Trichocephalus dispar*. (Rudolphi.) *Habitat*: Intestines.
4. *Trichina spiralis*. (Owen.) *Habitat*: Muscles.
5. *Filaria medinensis*. (Gmelin.) *Synonym*, *Dracunculus medinensis*. *English synonym*, Guinea-worm. *Habitat*: Skin and subcutaneous tissues.
6. *Filaria oculi*. (Nordmann.) *Synonym*, *Filaria lentis*. (Diesing.) *Habitat*: Eye.
7. *Strongylus bronchialis*. (Cobbold.) *Habitat*: Bronchial tubes.
8. *Eustrongylus gigas*. (Diesing.) *Habitat*: Kidney and intestines.
9. *Sclerostoma duodenale*. (Cobbold.) *Synonym*, *Anchylostomum duodenale*. *Habitat*: Duodenum.
10. *Oxyuris vermicularis*. (Bremser.) *English synonym*, Thread-worm. *Habitat*: Rectum.
11. *Filaria sanguinis hominis*. (Lewis.) A parasite in the human blood.
12. *Filaria* in dog's blood.

CLASS B, STERELMINTHA.

13. *Bothriocephalus latus*. (Bremser.) *T. lata*. (Linnæus.) *Habitat*: Intestines. The broad tape-worm, endemic to man in some localities only. Its embryo is ciliated and developed in water (KNOCH).
14. *Bothriocephalus cordatus*. (Leuckart.) *Habitat*: Intestines. Recently found in North Greenland.
15. *Tænia solium*. (Linnæus.) *Habitat*: Intestines.
16. *Cysticercus* of the *Tænia solium*. *Synonym*, *Cysticercus telæ cellulosa*. (Rudolphi.) The larva or scolex of the *T. solium*.
17. *Tænia mediocanellata*. (Küchenmeister.) *Habitat*: Intestines.
18. *Tænia acanthotrias*. (Weinland.) *Habitat*: Intestines.
19. *Tænia flavopuncta*. (Weinland.) *Habitat*: Intestines.
20. *Tænia nana*. (Siebold.) *Habitat*: Intestines.
21. *Tænia lophosoma*. (Cobbold.) *Habitat*: Intestines.
22. *Tænia elliptica*. (Batsch.) *Habitat*: Intestines.
23. *Cysticercus* of the *Tænia marginata*. *Synonym*, *Cysticercus tenuicollis*.
24. *Echinococcus hominis*, or Hydatid of the *Tænia echinococcus*. (Siebold.)
25. *Fasciola hepatica*. (Linnæus.) *Habitat*: Liver.
- 25* *Distoma sienensis*. (M'Connell.)
26. *Distoma crassum*. (Busk.) *Habitat*: Duodenum.
27. *Distoma lanceolatum*. (Mehlis.) *Habitat*: Hepatic duct; intestines.
28. *Distoma ophthalmobium*. (Diesing.) *Habitat*: Eye.
29. *Distoma heterophyes*. (Siebold.) *Habitat*: Small intestines.
30. *Bilharzia hæmatobia*. (Cobbold.) *Habitat*: Portal and venous blood.
31. *Tetrastoma renale*. (Della Chiage.) *Habitat*: Tubes of the kidney.
32. *Hexathyridium venarum*. (Treutler.) *Habitat*: Venous blood.
33. *Hexathyridium pinguicola*. (Treutler.) *Habitat*: Ovary.

CLASS C, ACCIDENTAL PARASITES.

34. *Pentastoma denticulatum*. (Siebold.) *Habitat*: Liver; small intestines.
35. *Pentastoma constrictum*. *Habitat*: Liver and lung; Negroes on West Coast of Africa.
36. *Œstrus hominis*. (Say.) *English synonym*, Larva of the gad-fly. *Habitat*: Intestines.
37. *Anthomyia canicularis*. (A. Farre.) *Habitat*: Intestines; and exciting causes of boils by their larva.

ECTOZOA.

38. *Phthirus inguinalis*. (Leach.) *English synonym*, Crab-louse.
39. *Pediculus capitis*. (Nitzsch.)
40. *Pediculus palpebrarum*. (Le Jeune in Guillemeau.)

41. *Pediculus vestimenti*. (Nitzsch.) *English synonym*, Body-louse.
42. *Pediculus tabescentium*. (Burmeister.)
43. *Sarcoptes scabiei*. (Latreille.) *Synonym*, *Acarus*. *English synonym*, Itch-insect.
44. *Demodex folliculorum*. (Owen.)
45. *Pulex penetrans*. (Gmelin.) *English synonym*, Chigoe. *Habitat*: Skin and cellular tissue.

ENTOPHYTA AND EPIPHYTA.

46. *Leptothrix buccalis*. (Wedl. Robin.) *English synonym*, Alga of the mouth.
47. *Oidium albicans*. (Link.) *English synonym*, Thrush fungus. *Habitat*: Mouth in cases of thrush, and certain mucous and cutaneous surfaces.
48. *Sarcina ventriculi*. (Goodsir.) *Habitat*: Stomach.
49. *Torula cerevisiæ*. (Turpin.) *Synonym*, *Cryptococcus cerevisiæ*. (Kützing.) *English synonym*, Yeast-plant. *Habitat*: Stomach, bladder, &c.
50. *Chionyphe Carteri*. *Definition*: A cotton fungus occurring in the disease called Mycetoma. *Habitat*: Deep tissues, and bones of the hands and feet.
51. *Achorion Schönleini*. (Remak.) *Habitat*: *Tinea favosa*.
52. *Puccinia favi*. (Ardsten.) *Habitat*: *Tinea favosa*.
53. *Achorion Lebertii*. (Robin.) *Synonym*, *Trichophyton tonsurans*. (Malmsten.) *Habitat*: *Tinea tonsurans*.
54. *Microsporon Audouinii*. (Gruby.) *Habitat*: *Tinea decalvans*.
55. *Trichophyton sporuloides*. (Von Walther.) *Habitat*: *Tinea Polonica*.
56. *Microsporon furfur*. (Eichstädt.) *Habitat*: *Tinea versicolor*.
57. *Microsporon mentagrophytes*. (Gruby.) *Habitat*: Follicles of hair in Sycosis or Mentagra.

The foregoing list might be extended by the addition of various parasitic vegetations, which have been reported under the names of Algæ, Fungi, Mycodermis, Lep-tomiti, &c., but the characters or the existence of which, as causes of disease, are still the subject of inquiry.

BACTERIA.

Bacteria have been under observation for more than twenty years; and they have acquired an importance (perhaps undue) in connection with PASTEUR's theory of fermentation, as being the objective points supposed to demonstrate spontaneous generation; and also in connection with the origin and propagation of specific diseases. The genera were first distinguished by Ehrenberg into—*a*, *Bacterium*; *b*, *Vibrio*; *c*, *Spirochæta*; *d*, *Spirillum*. They are represented by "chlorophyll-free cells" of spherical, oblong, or cylindrical forms, sometimes twisted or bent. They multiply exclusively by transverse division; and occur either isolated or in "cell families." Their existence in fluids renders the fluids milky or opalescent; but in fluids of equal refractive powers with themselves, such as lymph or serum, their existence is not to be distinguished without a microscope. They are not destroyed by potash, ammonia, nor acids. They never branch. The division may separate at once (*unicellular bacteria*), or may remain attached as strings or threads (*filamentous bacteria*). By swelling up of their cell membranes they form a jelly-like mass or colony (*Zooglaea*). The filamentous and screw forms never form jelly-masses. *Bacteria* frequently form an oily scum or stratum near the surface of a liquid, attracted by oxygen (the *mucor* of Pasteur); also as a tough pellicle, in which the bacteria are closely packed in rows (the *myco-*

derma of Pasteur). They may also form a pulverulent precipitate, when they have exhausted the nutriment in a fluid; and, as in the parallel case of yeast, they are then in a "resting phase." They also present a *motile* and a *motionless* condition. The *motile* condition is said to be connected with the presence of oxygen. In certain filamentous bacteria (*Bacteridia*) movement has never been seen.

There are four tribes or groups which ought to be distinguished, namely:—GROUP I. *Sphærobacteria*, or *globular bacteria*, in which *micrococcus*, or *monas*, is a genus, having the following species:—(a.) *Zymogenous*, or *fermentative micrococci*—the ferments of putrescence, consisting of *M. crepusculum*, *M. candidus*, *M. ureæ* (ferment of ammoniacal putrescence). (b.) *Chromogenous*, or *colour-producing micrococci*—the ferments of colour: *Micrococcus prodigiosus* (in blood-stained bread), *M. luteus*; *M. aurantiaceus*; *M. chlorinus*; *M. cyaneus*; *M. violaceus*. (c.) *Pathogenous*, or *disease-producing micrococci*—the ferments or germs of certain specific diseases—ferments of contagion, namely:—*M. vacciniæ*; *M. diphthericus*; *M. septicus*.

This latter class is of course most important, but excessively difficult of study, and is at present surrounded by much speculative mist. The difficulties cannot be overrated. In examining into and cultivating the products of disease, fresh and bacteria-free substances and blood taken direct from the body, with great precaution, are used. In almost every example of healthy blood many little corpuscles exist, with active movements impossible to distinguish from *micrococcus bacteria*.

GROUP II. *Microbacteria* are rod-like, and have but one genus, namely:—*Bacterium*, with the species, (a) *Bacterium termo*, the common bacterium of putrefaction, the saprogenous ferment; (b.) *Bacterium lineola*, a larger species common in brooks and open ponds; (c.) *B. Xanthinum*, (d.) *B. cyanum*, (e.) *B. Ærugineum*, the ferment of blue-green or verdigris-like pus.

GROUP III. *Desmobacteria*, or *filamentous bacteria*, include—(1.) *Bacillus*, in which the filament is straight; (2.) *Vibrio*, in which the filament is undulated. Of *Bacillus* there are three species:—(a.) *B. subtilis*, or butyric ferment; (b.) *B. ulna*, like the former, but larger and coarser; (c.) *B. anthracis*, the pathogenous ferment in malignant pustule, splenic fever, *sang*, charbon. Of *Vibrio* there are two species, one larger than the other, namely:—*V. rugula*, and *V. serpens*. Certain reagents dissolve *Vibrios*, namely strong sulphuric and hydrochloric acids (GRIMM). Weak solutions act slowly; *acetic acid* dissolves them very slowly; *chloride of ammonium* kills them, and then dissolves them; *creosote* clears them, and brings a vacuole into view; *iodine* colours them brown; *carmine*, red; *alcohol* and *ether* slowly dissolve them. All tests show them to be formed of a substance similar to "protoplasm." They are indifferent to the electric current. Conjugation or union may be seen between single-jointed vibriones, so that they become fused into a two-jointed; and the subsequent union of others forms a many-jointed rod. This has been seen in the bacteria of *splenic apoplexy* and in *Spirillum undula*. The single joint is the individual.

GROUP IV. *Spirobacteria*, or *screw-bacteria*, include two genera—*Spirochæta* and *Spirillum*. Of *Spirochæta* there is one species, namely—*Sp. plicatilis*. Of *Spirillum* there are three species:—(a.) *Spiril. tenue*; (b.) *Spiril. undula*; (c.) *Spiril. volutans*—the giant among bacteria; believed by Grimm to be only the union of two individuals of *S. undula*.

Cohn's figures must furnish the basis for future nomenclature of these paradoxical organisms, and higher magnifying powers will, no doubt, increase our knowledge. Those we know require at least magnifying powers of 600 diameters.

Bacteria are considered to belong to the vegetable kingdom, exhibiting direct and near affinities with Algæ; "and it seems extremely probable that just as yeast may be a degraded form of some higher fungus, *Bacteria* may be degraded allies of the *Oscillatoria* which have adopted a purely saprophytal mode of existence" (Sir JOSEPH HOOKER). They have nothing to do with fungi, and are at once distinguished from torulæ—the latter and fungi-species are air-carried; bacteria require a surface of water to transport them. Bacteria, further, resemble green plants in taking up nitrogen from ammonia compounds, which animals are unable to do; and urea or nitric acid may replace ammonia as sources of nitrogen. But they require carbo-hydrates or their derivatives to supply carbon, which they are unable to take from carbonic acid. In this respect they differ from green plants. Thus *succinic*, *acetic*, and *lactic acid*, *tartaric acid*, *sugar*, *glycerine*, *cellulose* may become for them sources of carbon.

Putrescence never occurs where the access of bacteria is prevented. It is believed to be a chemical process excited by the growth of *B. termo*; and it ceases when those present have been destroyed, or die when their nutrient material becomes exhausted. Their precipitation then takes place as a sediment to the bottom of the fluid. But the relation of *Bacteria* to the process of putrefication of albuminoids is one of speculation as yet, and may be one of four:—(1.) They may assimilate albuminoid substances as animals do, and break them up into waste products in their own substance. (2.) The *Bacteria* may give or shed out a peculiar substance (comparable to *diastase*), which acts so as to break up albuminoid substances. (3.) *Bacteria* may act as oxygen carriers, and so break up albuminoid substances as oxidising ferments. (4.) They may act as deoxidising or reducing ferments, taking oxygen from albuminoid substances, and so pulling down their complex fabric.

As with all "*protoplasm*," so with bacteria—repair and waste only go on in the presence of oxygen, and with the excretion of carbonic acid. But the oxygen has to be got, in many circumstances, elsewhere than from the atmosphere; and Pasteur has shown that from the breaking up of organic molecules, the organisms of the butyric fermentation (*Bacillus*) multiply in the absence of atmospheric air, and are checked by its presence.

The peculiar *smells* of putrescence are produced independently of oxidising or deoxidising action on albuminoid substances. The peculiar *smells* indicate peculiar (specific?) products of saprogenous (putrid or rancid generating) bacteria. These peculiar smelling products are generated when the bacteria are altogether nourished by *tartarate of ammonium* and *ash-salts*. So also the colour-producing (chromogenous) bacteria are abundantly produced in culture experiments of the same kind. The internal phenomena of nutrition in bacteria are, therefore, the same whether albuminoid or ammoniacal food is supplied; and the supposition is very probable that *Bacteria* always take their *nitrogen* in an *ammoniacal* form;

and the essential feature of putrescence is the splitting up or reduction of the albuminoid molecule into ammoniacal ammonia, salt, and other fluid and gaseous products.

But it is here very difficult, if not impossible, to separate and identify the special or specific products of bacterian life (the excreta of *Bacteria*) from the by-products of the splitting of the albuminoid molecule which precedes their assimilation of the nitrogen from the ammonia. The explanation of the power of Bacterim to split up albuminoid molecules is overwhelmed in a mist of speculation. (Dr. FERDINAND COHN, *Journ. Micros. Science*, 1873, p. 156.)

The distinction between mature and immature parasites has not been maintained in the classification given at pp. 148–150; but in the description about to be given, it is necessary that this distinction shall be kept in view, namely:—

(1.) The *sexually mature entozoa* inhabit either the alimentary canal of animals or the cavities of the lungs; or, to express it generally, they inhabit such parts of the body as are in immediate or free communication with the external air.

(2.) On the other hand, the *non-sexual or immature entozoa*, while parasitic, live inclosed in cysts; such cysts being situated either in the parenchyma of organs, like the liver or lung, or in close internal cavities—*e. g.*, the peritoneum, pleura, pericardium, or eye, within secreting tubes, blood-vessels, and the like. In such places these non-sexual parasites are all proved to be *incomplete* animals. They are the embryos, larvæ, or early forms of entozoa, which can only attain to sexual maturity by migration from the place of their earlier abode into the alimentary canal, or pulmonary surfaces, or other open cavities or canals of different animals. Or, leaving their encysted parasitic state in the condition of larvæ, they reach maturity in a free state (*i. e.*, liberated from their encysted condition), when they are developed in water, in earth, mud, or upon moist plants, or in other conditions favourable for them.

The cystic or vesicular entozoa (established by Rudolphi as a separate order of parasites) are to be distinguished from those which are not vesicular, but which are inclosed in cysts. Some of the early parasitic forms of *round worms*, as well as others, are thus inclosed in cysts (*Trichina spiralis*, Fig. 18, p. 160); but they are not cystic or vesicular; they are encysted. As examples of *cystic* parasites the *Cysticerci* of the *tænia* may be mentioned. All entozoa so encysted (whether they be cystic or not) are found to be immature; and in no instance has the encysted entozoön been known to attain sexual completeness, however well grown it may appear to be, so long as it remains inclosed in a cyst. Sometimes cysts only are found, which may be identified as pathologically altered conditions of cystic or encystic entozoa. When free, all these entozoa proceed by growth and development to acquire sexual organs, and, when they have arrived at maturity, they exercise the function of sexual reproduction. The number of fecundated ova which most of them produce is enormous. In a *tape-worm* or *ascaris*, there are many millions; but “the struggle for existence” consigns the greater number of them to death as the food of birds, or fish, or other animals; and when they thus

become food, of course their existence as parasites comes to an end. Diarrhoea is largely explained by the presence of parasites. In birds—especially grouse, Cochín-China, and other fowls—nematoid worms pervade the intestines (e. g., *Anguilla stercoralis* and *A. intestinalis*). The prolific character of these minute worms is such, that in severe cases an examination of the fæces showed that hundreds of thousands of these minute worms were evacuated in twenty-four hours, and that only five days were necessary for the full growth and maturation of them.

The process of fecundation and the development of the embryo have now been actually observed in a considerable number of the parasitic entozoa; and it is to be remembered, as a general fact, that the development of the ova rarely takes place in the same being who entertains the unwelcome guest, or in the same part of an animal where the parent parasitic entozoön has passed its life and has exercised the generative function. There is either a migration from a parasitic to a free condition for a time (e. g., *Guinea-worm*, *Ascarides*, *Cercarie*); or from one animal into another animal, the free condition intervening (e. g., *Bothriocephalus*); or, lastly, the migration may take place from one part to another of the same animal who is the unfortunate host (e. g., *Trichina spiralis*; and cases of tape-worm giving rise to *Cysticercus*). Some entozoa, known only as incomplete or immature animals in the parasitic mode of life, attain to sexual maturity in the free state; others again, and perhaps the greater number, after living free for a time, become sexually complete in the parasitic condition (e. g., the *Ascarides* and the *Bothriocephalus*).

“The migrations or changes of habitation of the entozoa, or their ova or embryos, appear to take place in a variety of ways: *first*, by their being passed out of the body of the inhabited animal with the fæces or other excretions; *second*, by their being introduced into the bodies of inhabited animals with their food or drink; *third*, by their directly piercing the integument or other tissues; *fourth*, by their piercing the membranes and parenchyma, entering the blood-vessels, being distributed through them, and subsequently piercing their coats to attain other situations.”

The lesions and diseases caused by the existence of parasites rather tend to embitter existence than to cause death; and they are especially frequent amongst soldiers. With one exception—namely, in the case of the immature parasites—the disorders induced are, as a rule, not severe; indeed, it is a condition of parasitism that it should not actually destroy the life of the animal from which it derives its own subsistence. The “host” is a necessity for the “guest;” but the “guest” is not a necessity for the “host.”

It is the *immature parasites* which tend to destroy the life of their host, by the severe lesions they induce, and the destruction of the parts which they cause, when they pass from one place to another, or from one state to another onwards to maturity. Thousands of mature worms infest children, yet they do not appear ill. But such is not always the innocent history even of the mature worms,—undefined illnesses, violent

and sudden pains, febrile phenomena, like typhoid or rheumatic fever, chronic inflammations, wastings, convulsions, chorea, epilepsy, amaurosis, apoplexy, giddiness (staggers in sheep and horses), are the grave results which sometimes befall human or other animals who may become the unfortunate entertainers of such undesirable guests as *mature* or *immature* parasites. The accounts given of the *cerebro-spinal* symptoms of worms are remarkably defective. (Dr. HESLOP, *Dublin Quarterly Journal*, No. 35.) They are mainly as follows:—

“1. That in the great majority of cases of tape-worm, and, though with lesser frequency, in cases of other intestinal worms, more or less serious and peculiar nervous disturbances are apt to arise.

“2. That the most frequent of these are headache, giddiness, various troubles of the special senses, especially singing in the ears, flashes and dark spots before the eyes, imperfect amaurosis, and trembling of the limbs.

“3. That various anæsthetic, and, on the contrary, neuralgic phenomena, are very frequent, usually connected with general lassitude and sense of muscular feebleness.

“4. That, though less frequent than those previously cited, convulsive seizures, partaking of the nature of epilepsy or acute eclampsia, or sudden attacks of insensibility, mixed with syncope, and, in the female sex, severe forms of hysteria, are also often directly traceable to worms.

“5. That the last symptoms (No. 4) are more common in childhood and the earlier periods of life than afterwards, and are more frequently caused by the round and thread worms than by tape-worm.

“6. That chorea does not appear to be often excited by the irritation of worms.

“7. That a feeble state of the general health generally accompanies the presence of worms; often, in cases of *tænia*, proceeding to marked anæmia, so as even to lead to the suspicion of the possible existence of Bright's disease.

“8. That the irritative phenomena of the digestive tube, even when associated with various symptoms referred to the functions of that tract, do not warrant the diagnosis of the presence of *tænia*; and that their absence does not absolutely indicate the absence of the parasite.

“9. That the frequent appearance of the nervous symptoms above related, without a well-marked relation to any special lesion of the nervous system, especially if alternating with periods of perfect or nearly perfect freedom, should engender the suspicion that worms are present. If to these symptoms are added various ill-defined disturbances of the functions of assimilation, including occasionally colicky pains, without marked vomiting, pain after food, or decided emaciation, it is in the highest degree probable that worms are the source of the symptoms.

“10. That it is probable that many of the symptomatic phenomena of vermination are connected not with their direct irritation of the mucous membrane with which they are in relation, but with a general disorder of the system, partly resulting from the parasites, and partly the cause of their maintenance and development in the intestinal tract.”

The prevalence of parasitic disease in animals comes in the end to

be a national loss, affecting the health and wealth of nations, by rendering the flesh of many animals used as food innutritious, or altogether unfit for use.

Following nearly the arrangement in the Table at pp. 148-150 a detailed description of those parasites will now be given, and of the lesions associated with them, commencing with—

I.—ENTOZOA.

CLASS A.—CCELELMINTHA, OR HOLLOW WORMS.

Definition.—*Worms of an attenuated, cylindrical form, with an abdominal cavity, in which an intestinal canal is suspended free. They are possessed of a mouth and anus, and have the sexes distinct.*

Pathology.—In their mature state these worms inhabit the alimentary canal, the pulmonary tubes or areolar tissue of man and animals. In their immature state some are encysted in the human body, and others come to maturity in open waters. In the human subject they are represented by the *Ascarides*, the *Oxyurides*, the *Trichinæ*, the *Sclerostoma*, the *Strongylus*, the *Speroptera*, and the *Filaria*.

A knowledge of the generation of these round worms is of the utmost importance for preventing their occurrence in man. Their generative organs are adapted for the reproduction of an enormous number of fertile ova. They are males and females after their kind; but the males, as a rule, are scarcer and smaller than the females.

Dr. Eschricht has made an elaborate calculation regarding the enormous fertility of the *A. lumbricoides*, the commonest parasite of man. The ova being arranged like flowers upon a stem in the ovary tubes, he has counted fifty in a circle, or in every transverse section. The thickness of each ovum he estimates at $\frac{1}{500}$ of a line ($= \frac{1}{500}$ of $\frac{1}{12}$ = $\frac{1}{6000}$ of an inch); so that in every line of length of the worm there would be 500 wreaths of 50 eggs each = 25,000 eggs. The length of each horn of the uterus is taken at sixteen feet, which gives 2304 lines; and for the two horns it will give 4608 lines. The eggs, however, gradually get as large as $\frac{1}{60}$ of a line, so that only sixty wreaths of eggs come to be on one line, or about 3000 ova; and an average gives 14,000 ova in a line—i.e., sixty-four millions of ova in every mature female *Ascaris*.

What becomes of all these ova? Being discharged by millions, many of them, in large cities, are carried into streams of water. An extremely small proportion is ever likely again to find their way into the alimentary canal of the animal which was the dwelling-place of the parents of these ova. Thus they become food for numerous inhabitants of the water, and therefore stand in the same relation to many of these animals as the *cereal*ia in the vegetable kingdom to the higher animals on land. Indirectly they thus contribute to the sustenance of man and animals.

The source of the various *Ascarides* which inhabit man is not yet fully

known. But there are many circumstances which tend to show that the *A. lumbricoides* is most frequently introduced as a minute embryo with water or with fresh uncooked vegetable food.

Dr. Paterson, an eminent physician in Leith, observed that the children of certain families who drew water from a public well in a particular street there, were very subject to the *A. lumbricoides*; while towards the other end of the same street, the families were supplied by the pure water which supplies Edinburgh and its vicinity, and these families were free from the parasite. The water of the well came from a dirty pond or lake in the vicinity (called Lochend), and in its water numerous vermiform animalculæ existed, such as the *Anguillula fluviatilis*, perhaps the embryonic form of an *Ascaris*.

Another point to be remembered in all inquiries regarding the nature of parasites, is the intense tenacity of life, and revival from a state of apparent death, exhibited by some of them; and in no class of animals has the origin by spontaneous generation been more strenuously contended for than in regard to these entozoa. There is a minute worm of a nematoid kind, which is a parasite upon wheat grains (the *Vibrio tritici*); some of these being dried, and then remoistened after a lapse of four to seven days, resumed their living active state (BAUER, in *Phil. Trans.*, 1823, p. 1, quoted by OWEN). Dr. Blainville has given other similar instances of revival after desiccation; and some mature entozoa will even resist the effects of such destructive agents as extremes of heat and cold, to a degree beyond the endurance of any other minute animals. Owen relates that a nematoid worm has been seen to exhibit strong contortions—evident vital movements—after having been subjected above an hour to the temperature of boiling water with a codfish which it infested. Rudolphi mentions of some entozoa which infest herrings annually sent to Berlin hard frozen and packed in ice, that they do, when thawed, exhibit unequivocal signs of restored vitality (OWEN, *Lectures on the Invertebrata*, p. 80). *Ligulæ* are often found alive in undercooked codfish. Rudolphi found individuals of *Ascarides (spiculigera)* stiff and hard in the gullet and stomach of a bird (*cormorant*) kept in spirits of wine for eleven days, and which returned to life in warm water. Miram has seen individuals of the *Ascarides (acus)* from the pike become dry, and remain sticking to a board, where they would revive again by being placed in water; and in some instances they would move a part of the body which had imbibed the fluid, while the remaining part continued shrivelled up and adherent immovably to the board. I have seen the same results in the *Ascaris* which infests the peritoneal covering of the mackerel. But the tenacity of life on the part of the mature animal, is quite equalled by the powers of endurance possessed by the ova and young? They live and develop themselves under circumstances of the most improbable kind. Dr. Henry Nelson and Dr. Allen Thomson have observed the development of the ova of the *A. mystax* to proceed for several days, while the parent bodies containing them were immersed in oil of turpentine. I have once seen the same occurrence; and also the development of the embryo to continue in spirits of wine for about three weeks before signs of vitality had ceased.

Period of Incubation of the Ova.—The eggs are expelled with the fæces in

the case of the *A. lumbricoides*. They have been placed in water and taken care of for various lengths of time, and Richter records, that at the end of eleven months each ovum contains a living embryo. In August, 1853, Verloren and Richter put a fragment of a mature female *Ascaris* (*marginata*, of the dog) into water, so as to keep the ova merely moist. They were examined from time to time with the microscope. Segmentation having commenced, the development of the young was completed in fourteen days. They moved with great briskness within the eggshells, *but did not break through them*. In this imprisoned or encysted state they continued throughout *autumn and winter*; the movements of the embryos gradually diminishing, and at last entirely ceasing during the winter months, to recommence in the following spring, and to become again distinct in summer. *But they never broke through the shell*. Thus the condition of these *Ascarides*, from the encysted state of inclosure within the egg, is only changed under favourable circumstances—namely, when they are liberated, and carried on to further development; and it is now known that the embryo of nematoid worms may pass the winter in a torpid state, floating about in the open waters, or lying in moist places. The fully-formed embryo is cylindrical, its length $\frac{1}{100}$ of an inch; the mouth is not furnished with the three characteristic papillæ of the genus, and the tail terminates suddenly in a point. It is highly probable, from the evidence, that the embryos are directly transferred to the alimentary canal of man from river and pond water.

The *Ascaris lumbricoides*, or round worm, is perhaps the one of which we have the most ancient records, and it is the most common of human entozoa. It is much more common in children and adults than in old people. The body is long (six to sixteen inches), round, elastic, and attenuated towards both extremities, but more attenuated towards the anterior end. It is of a greyish-red colour, and sufficiently translucent to permit its viscera to be seen through its coverings. These *Ascarides* inhabit chiefly the small intestines, but may pass up into the gall-ducts, the stomach, the œsophagus, the nostrils, the mouth or frontal sinuses; and there are cases on record and specimens in museums where the worm has evidently penetrated the coats of the intestine, and got into the peritoneum or into the pleura. It sometimes makes its way by the bile-ducts into the liver, and leads to hepatic abscesses. An interesting example of this may be seen in a preparation in the museum of the Army Medical Department at Netley. In some cases they are so numerous as absolutely to obstruct the intestines; in others only a solitary worm, or a pair, may be found. Three distinct tubercles surrounding the mouth characterise the genus. The posterior end is obtuse, but is straighter and thicker in the female than in the male. It is abruptly acute and curved in the male. An anus is situated in both sexes close to the tail, and in form is like a transverse fissure. In the female the body presents a constriction at the junction of the anterior with the middle third; and here the *vulva* is situated. The parasite throughout is marked with transverse furrows and with fine striæ. Longitudinal-equidistant lines run from the head to the tail, and are independent of the exterior envelope.

The *Ascaris mystax*, which infests every domestic cat, is found also as

a human parasite (PICKELLS, BELLINGHAM, COBBOLD). It varies from an inch and a half to two or three inches long, its head end being spear-shaped, in consequence of two lateral processes, from which it has been named *A. alata*. The ova have the embryo developed within them before they escape from the parent, and in this respect they resemble the *Oxyuris vermicularis*, which has yet to be noticed.

The *Trichocephalus dispar* occurs in the cœcum and colon, and was first noticed by Morgagni. More than a hundred years ago (1760–61) a student of Gottingen was dissecting the valve of the colon of a girl five years of age. He accidentally opened the gut, and several of these entozoa came out. Wrisberg and other students considered the worm a new one; but the demonstrator of anatomy maintained that it was an *Ascaris* or an *Oxyuris*, and a dispute arose. At last the new parasite got a name as a new worm, and was called a *Trichinalis* or *hair-tail*. But it afterwards turned out that its *head* was hair-like, and not its *tail*, so it has been since called *Trichocephalus*. About this same time an epidemic raged in the French army stationed at Gottingen, and the disease was described under the name of the *Morbus mucosus*. This entozoön was frequently found in the bodies of the soldiers who died during the epidemic of this mucous flux.

It is said to be very common in persons attacked with typhus fever; and is found in those dying with excessive discharges from the bowels, as in cholera and diarrhœa. It is found in France, England, Egypt, Ethiopia, and rarely in Italy; abounding particularly in the *caput cœcum*. It is generally thought to be scarce in England—a persuasion which Dr. Cobbold thinks has probably arisen from “the negligence of pathologists, whose arduous duties connected with the superintendence of *post-mortem* examinations have, perhaps, left them little time for these inquiries.” On the other side of the Channel this parasite is so abundant in some localities, that not less than one-half the inhabitants of Paris are affected by it (DUVAINE).

These parasites are males and females, in separate sexes, varying from one and a half to two inches. The males are shorter than the females, and less thick posteriorly, with a long spiculum. The eggs are oval, with resisting shells $\frac{1}{110}$ of an inch in length. The anterior extremity, carrying the head, is the narrow hair end, and it is usually buried in the mucous membrane of the intestines, while the remainder of the body moves freely in the midst of the mucous secretion, generally coiled upon itself.

The *Trichina spiralis* was first seen by Tiedemann in 1822, and described by Mr. Owen in 1835, from a specimen taken to him by Sir James Paget. It has since been often recognised chiefly as a dissecting-room curiosity. The student tries in vain to clean the fibres of the muscle he is dissecting, which, however clearly displayed, still looks as if it were “sprinkled over with the eggs of some insect.”

Instances of the *T. spiralis* in the muscles of the human body are of much more frequent occurrence than has generally been supposed. Most probably, from their very minute size, they have hitherto been overlooked, and the symptoms of illness produced by them has been mistaken for *enteric fever* and *rheumatism*. Virchow had not seen a case before 1859, after

which he met with no less than six in one year. It is perhaps more common in man than even the *Cysticercus*, and Dr. Zenker, of Dresden, found *Trichinae* in four out of 136 dissections—i.e., one in thirty-four. Much interest has been excited of late in this subject; for, in place of the *T. spiralis* being quite a harmless parasite, as has been hitherto the belief, Zenker has shown that it is the source of a new and most alarming form of disease; that in place of remaining harmless and encysted in its capsule, only to cretify or degenerate, its migrations amongst the muscles may give rise to symptoms of the most serious kind before it becomes encapsuled, causing death in a strong and healthy person after a few weeks of painful suffering. It seems, indeed, to be the most dangerous of all parasites—not even excepting the *Echinococcus*; and it behoves the physician to know something about the nature, origin, and development of this entozoön, seeing that its pathological relations are now known to be of extreme importance and interest.

Hitherto the *T. spiralis* has been known as a minute round worm, inclosed in a more or less transparent capsule, lying between the sarcolemma of the primitive muscular fibres (Figs. 18, 19); but when the parasite is free, it finds its way within that sheath. It forms the type of a distinct genus of *nematoides*, having no genetic relations with the *Trichocephalus dispar*, as was supposed; but is reproduced viviparously. The non-encysted *Trichina* may exist in the flesh of animals without being visible to the naked eye. In the encysted state they are difficult of



Fig. 18.*

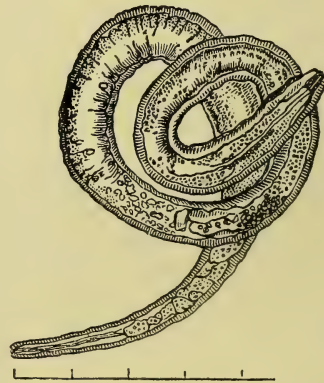
100th of an inch \times 300.

Fig. 19.†

detection without the aid of a lens, if cretification has not commenced in the cyst. The cysts are round or elongated, and appear, according to their shape, like small round dots, granules, vesiculæ, or streaks, greyish-white or opaque, and quite distinct from the red transparent muscle. When the cyst has become calcareous, its limy material may be dissolved

* Slightly magnified cyst of *Trichina spiralis* (after VIRCHOW).

† The *Trichina spiralis* removed from its cyst (after VIRCHOW).

away by acetic or hydrochloric acid with the evolution of gas, and the parasite is then seen coiled up within (Fig. 18).

The following is a detailed account of this parasite :—

“(1.) In its mature state the *Trichina spiralis* is an extremely minute nematoid helminth, the male in its fully developed and sexually mature condition measuring only $\frac{1}{18}$ of an inch, whilst the perfectly developed female reaches a length of about $\frac{1}{8}$ ”; body rounded and filiform, usually slightly bent upon itself, rather thicker behind than in front, especially in the males; head narrow, finely pointed, unarmed, with a simple central minute oral aperture; posterior extremity of the male furnished with a bilobed caudal appendage, the cloacal or anal aperture being situated between these divergent appendages; penis consisting of a single spicula, cleft above, so as to assume a V-shaped outline; female shorter than the male, bluntly rounded posteriorly, with the genital outlet placed far forward, at about the end of the first fifth of the long diameter of the body; eggs measuring $\frac{1}{1270}$ ” from pole to pole; mode of reproduction viviparous. (2.) The sexually mature trichina inhabits the intestinal canal of numerous warm-blooded animals, especially mammalia, and constantly in great numbers. (3.) At the second day after their introduction, the intestinal trichinae attain their full sexual maturity, lose their spiral figure and become stretched, whilst they grow rapidly, and their generative organs are developed. (4.) Most females contain from three to five hundred ova. In six days the female parasites will contain perfectly developed and free embryos in the interior, and these, on attaining full size, pass out at the vaginal opening. The eggs of the female trichina are developed within the uterus of the mother, and contain minute filaria-like embryos, which are born without their eggshells. (5.) The new-born young soon afterwards commence their wandering. They penetrate the walls of the intestines, and pass directly through the abdominal cavity into the muscles of their bearers, where, if the conditions are otherwise favourable, they are developed into the form hitherto known. (6.) The direction in which they proceed is in the course of the intermuscular connective tissue. (7.) The majority of the wandering embryos remain in those sheathed muscular groups which are nearest to the cavity of the body (abdomen and thorax), especially in those which are smaller, and most supplied with connective tissue. (8.) The embryos penetrate into the interior of the separate muscular bundles, and here, after fourteen days, acquire the size and organisation of the well-known *Trichina spiralis*. (9.) Soon after the intrusion of the parasite, the infested muscular fibre loses its original structure. The fibrillae collapse into a finely granular substance, whilst the muscular corpuscles change into oval nucleated cells. (10.) The infested muscular bundle retains its original sheathing up to the time of the complete development of the young trichinae, but afterwards its sarcolemma thickens and begins to shrivel at the extremities. (11.) The spot inhabited by the rolled-up parasites is converted into a spindle-shaped widening, and within this space, under the thickened sarcolemma, the formation of the well-known lemon-shaped or globular cysts commences by a peripheric hardening and calcification. One cyst may contain from one to three trichinae. (12.) The migration and development of the embryos also take place after the transportation of impregnated trichinae into the intestines of a new host. (13.) The further development of the muscle trichinae into sexually mature animals is altogether independent of the formation of the cal-

careous shell, and occurs as soon as the former have reached their completion. The male and female individuals are already recognisable as sexually distinct in their larval state." (*Entozoa: an Introduction to the Study of Helminthology, with reference more particularly to the Internal Parasites of Man.* By T. Spencer Cobbold, M.D., F.R.S. London, 1864.)

The symptoms of disease induced by this parasite are at first of a febrile nature, having a close resemblance to some forms of specific fevers. Dr. Philip Frank, lately Assistant-Surgeon on the Staff of Her Majesty's Army Medical Department, was the first to send an account of this remarkable disease from Germany to this country. He described a case of its occurrence in the *Medical Times and Gazette* of May 26, 1860. The history of the case is as follows:—In January, 1860, a servant girl about twenty years of age died in the Dresden Hospital from the effects of the *T. spiralis*; and the muscles of her body furnished materials for numerous observations and experiments, which have thrown much light on the origin and development of this parasite. The illness of the patient commenced about Christmas, 1859. The symptoms may be arranged into two sets:—*First*, Extreme lassitude, depression, sleeplessness, loss of appetite, and eventually febrile phenomena, which were well expressed, so much so, that the case was set down as one of typhoid fever. But grave doubts prevailed; for, *second*, a new train of symptoms developed themselves—namely, the whole muscular system became the seat of excruciating pain, especially in the extremities. Contractions of the knee and elbow joints supervened, and associated with such extreme pain as to render extension of the limbs impossible. Œdema of the legs followed; and the case terminated fatally by pneumonia, about the twenty-eighth or thirtieth day after the first symptoms of illness.

A *post-mortem* examination of the body showed the muscles moderately developed, of a pale reddish-grey colour, and dotted over with specks, which turned out to be groups of non-encapsuled *Trichinæ* lying freely upon and within the sheaths of the muscular fibres. They were alive—some coiled up and others lying straight; and they appeared to be in all stages of development, diffused throughout all the striated muscles of the body, not even excepting the heart itself. They abounded in such vast numbers that as many as twenty *Trichinæ* were seen in the field of view through a low magnifying power, the muscular tissue being everywhere in a degree of very marked degeneration. In the jejunum were found sexually mature *Trichinæ*. Death was due to the development of the *T. spiralis*, whose existence fully explained the anomalous symptoms which attended the case.

On looking into the history of the girl, it was found out that she had been a servant in a farm-house, and had been taken ill very soon after the killing of two pigs and an ox—animals which it is customary to kill about Christmas. Pigs are known to be infested with the *T. spiralis*—so are oxen; and Professor Zenker went to the master's house, and found some ham left of the identical pig that had been there killed, and also some sausages. The flesh of the pig was examined microscopically, and every specimen examined showed that the pig's flesh was infested with *Trichinæ* in the encysted state. At same time Professor Zenker

learned that soon after the girl had been taken ill, the housekeeper also became unwell, with similar symptoms, but in a less severe degree; and all the servants about the farm became more or less ill about the same time. The house of the butcher who had killed the pig was then visited by Professor Zenker, who was informed by the wife of the butcher that he had been very ill since that event. He had been three weeks in bed, suffering from *rheumatic pains in the limbs*, and had been as if paralysed over his body—unable to move his arms, legs, or neck. He had never suffered anything of the kind before, but had always been a healthy and strong man. He thought he had caught cold the day he killed the pig; but when it is known to be a habit of German butchers to taste the meat they kill, in the raw condition, the history of these cases, to Professor Zenker, became a history of *trichinatus disease*—the development and growth to maturity of the *T. spiralis* in the muscles of those who lived at the farm-house, as well as of the butcher who had killed the pig, and who no doubt had eaten some of its flesh.

Numerous experiments were made with the flesh of the girl who thus died of this remarkable disease. Portions were sent by Zenker to Professor Virchow at Berlin. He fed a rabbit with some of the flesh, and this rabbit died about a month after the feeding, with symptoms of general muscular paralysis, and myriads of young *Trichinæ* were seen in its muscles. Other rabbits were fed with the flesh of the first rabbit, and they too died with similar phenomena. Virchow found the villi of the intestines of the rabbits loaded with the ova or *prosperrmiae* of the entozoa; and he found mature *Trichinæ* of *both sexes* moving freely in the mucus of the intestine. The males were filled with sperm corpuscles, while the females were densely stocked with ova and their germs, and with young ones in the eggs coiled up like little snakes. Another observer had before made similar experiments. Herbst, in 1852, fed three young dogs with the flesh of a badger whose muscles were saturated with *Trichinæ*. The dogs in their turn became *trichinatus*; being killed after a few months, the parasites were seen in their flesh. Pigeons also were fed with moles' flesh known to be *trichinatus*; and free *Trichinæ* were found in the flesh of the neck, the wings, and the thighs of the pigeons in eighteen days. But Herbst did not examine into the relation between the capsuled and the free *Trichinæ*, as Virchow and Zenker have done.

In the summer of 1860, Dr. Turner (then Demonstrator of Anatomy in Edinburgh, and now Professor) took the opportunity of verifying these experiments of the German professors. He fed kittens with portions of the human flesh containing the worms, which were observed to move, though somewhat languidly, on rupturing the cysts. To one cat, on the 7th, 13th, and 16th of July, he gave portions of the flesh, and in the intervals fed it on bread, milk, and fish. He killed the cat on the 24th of the month. Nothing could be seen with the naked eye in the fluid of the small intestines; but on placing a drop below the microscope, thread-like worms were seen actively moving about in it, or coiling themselves up in a spiral form. Every drop of fluid taken contained one or more. Each of these thread-like worms was about

$\frac{1}{40}$ of an inch long, and $\frac{1}{1000}$ of an inch broad, with a pointed and a rounded end, and about two-thirds smaller than the mature flesh-worms met with in the muscles of the cat. These had migrated from the intestines, and after working their way between the fibres of the muscles, had become encapsulated—the capsules being perfectly transparent. Herbst and Virchow have found the flesh-worms both in the mesenteric glands and in the mesentery, and therefore, presumably, *in transitu* between the intestines and the muscles. All the phenomena described occurred within the space of a single month; and even as early as three weeks after feeding, Virchow found the young brood equal in size to those administered at the commencement. The genesis, development, and migrations of these flesh-worms are thus proven to be astonishingly rapid. Dr. Thudichum has also very recently verified these experiments; and at the conversazione of the British Medical Association, held at Downing College, Cambridge, on 4th August, 1864, he exhibited the parasite, *living*, in various stages of development, which he obtained from the muscle of a rabbit infested with them, and also from some pork chops (*Brit. Med. Journal*, August 13, 1864).

The *Trichina spiralis* is now well-known not to be limited to the muscles of man. It occurs in eels, cats, dogs, badgers, hedgehogs, pigeons, moles, and swine. It has been found in the horse, ox, sheep; and it has been reproduced in rabbits, rats, mice, and Guinea-pigs fed with trichinous flesh. Thus the *débris* of an animal eaten by *carnivora* may become fatal to *rodents*; and a carcass infested with trichinæ and decomposing near a marsh or rivulet may communicate the *parasites* to man or animals drinking such water (DEVAINE).

Thus the *T. spiralis* has been shown to be a bisexual parasite, producing its young alive in the intestines of the animal whose muscles it may infest. In the mucus of the intestines the mature *Trichinæ* find a suitable place for growth and for breeding. The progeny find their way to the muscles, where they eventually become encysted; and their favourite haunts seem to be the small muscles of the larynx (ZENKER). According to Virchow's conclusive testimony, all these phenomena occur within the space of a single month; and, in his experiments, even as early as three weeks after ingestion, the young were found to equal in size those that he administered at the commencement. But even in the seventh and eighth week, living *Trichinæ* have been found in the intestines, filled with eggs and embryos, rendering it not improbable that the intestinal *Trichinæ* bear young repeatedly, which pass into the muscles at different times; hence also the exacerbations of the disease. The genesis and migrations of *Trichinæ* are therefore astonishingly rapid, and probably without parallel in this class of parasites (COBBOLD). So long as it remains in the capsule, it is immature and non-sexual, and is so far harmless.

Since the discovery by Leuckart of the round worm, of which the *T. spiralis* is the immature condition, since the case recorded by Zenker, and since the more complete knowledge that has been acquired by experiments of the wonderful migrations of the young *Trichinæ*, attention has been especially directed to the possibility of the *trichinatus* disease in man being much more common than was anticipated, and it has been found to be so.

That individuals enjoy good health, although the muscles are infested with the encapsuled *Trichinæ*, is now well known, from the number of cases that have been seen in dissecting-rooms. It has also been recognised in the muscles of men killed by accident, when engaged in severe manual labour (CURLING, *London Med. Gazette*, Jan., 1838; also TURNER in *Edin. Med. and Surg. Journal*, 1860, p. 209). The distinguished teacher of clinical surgery at Berlin, Professor Langenbeck, related to the Medical Society there, in 1863, the case of a man from whom he had recently removed an epithelial cancer situated in the neck. During the operation, the *platysma myoides* exhibited a singular appearance, which, on careful inspection, was found to arise from the presence in the muscle of innumerable dead *Trichinæ*, contained in calcified capsules. On inquiry, the following facts were elicited:—In the year 1845 there was a “church visitation” (which seems to imply some jovial meeting), in which eight persons took part, and of these seven afterwards sat down to a breakfast consisting of ham, sausages, cheese, roast veal, and white wine. In the course of three or four days every one of the seven persons was seized with diarrhoea, pains in the neck, oedema of the face and extremities. Of the seven, four died, and the three who survived (among whom was the man operated upon eighteen years afterwards by Professor Langenbeck) remained ill for long afterwards. The suspicion arose that poisoning, through the agency of white wine, had taken place; and an investigation was made, but without any result. The innkeeper, however, at whose house the breakfast was given, being still under suspicion, was obliged to give up his business and emigrate. The importance of such a case in its forensic aspects cannot be overrated; and it becomes an important subject of inquiry whether some of our cases of death from suspected but unproved poisoning may not be due to *trichina disease*, which is now known to be much more prevalent than has hitherto been supposed, both in this country and in Germany.

Another occurrence, almost tragical, took place about the middle of October, 1863, when there was a festive celebration at Heltstädt, a small country town in Prussia, near the Hartz Mountains, numbering from 5,000 to 6,000 inhabitants. One hundred and three persons sat down to an apparently excellent dinner, mostly men in the prime of life. Within a month more than twenty persons had died, and more than eighty persons were then suffering from the fearful malady, while those who were apparently unscathed were in hourly fear of an outbreak of the encapsuled flesh-worms. The dinner had been ordered at a hotel, and it was arranged that the introduction to the third course should consist of “Röstewurst.” The sausage-meat was therefore ordered at the butcher’s the necessary number of days beforehand, in order to allow of its being properly smoked. The butcher, on his part, went to a neighbouring proprietor of pigs, and bought one of two pigs from the steward of the pig-farm. The steward unfortunately sold a pig which his master intended should not be sold, because it was not considered to be in good condition; and so, for this time, the butcher got “the wrong sow by the ear.” The ill-conditioned pig was the one that was killed and worked up into sausages. These were duly smoked and delivered at the hotel; and after being toasted before the fire (so as to be warmed through

merely), they were served to the guests at the dinner-table. On the day after, several persons who had eaten the dinner were attacked with great irritation of the bowels, loss of appetite, great prostration, and fever. The number of persons attacked rapidly increased; so much so, that great alarm was felt in so small a town lest an epidemic of typhoid fever was about to set in. But one of the physicians at last conjectured that some poison must be at the bottom of the outbreak, and an active inquiry into all the circumstances of the dinner was instituted; and when the muscles of the calves of the legs of some of the sufferers began to be affected, the descriptions of Zenker's case (already described, pp. 162, 163, *ante*) was at once remembered. The remnants of sausages, and of pork employed in the manufacture of them, were examined with the microscope, and found to be literally swarming with encapsuled flesh-worms. From the muscles of several of the suffering victims small pieces were excised, and under the microscope they were seen to be charged with *Trichinæ* in all stages of development. It could therefore no longer be doubted that as many of the 103 persons as had dined together and partaken of the "*Rösteuwurst*" were affected with trichinous disease by eating the trichinous pork, the flesh-worms of which had not been killed by the smoking and toasting. On the contrary, the subdued heat of toasting would rather foster their vitality. This catastrophe awakened sympathy and fear throughout the whole of Germany. Most of the leading physicians were consulted in the interests of the sufferers; and some visited the neighbourhood where most of the affected patients were. None could bring relief or cure. Case after case died a slow and lingering death, by exhaustion from nervous irritation, fever, loss of muscular power, inflammation of the lungs, or of organs essential to life.

The symptoms of trichinatus infection begin a few days after eating the meat in which *Trichinæ* exist, with loss of appetite, general discomfort after eating, irritation of the stomach, vomiting, and diarrhoea. These symptoms last from four to eight days, till the progeny are born. Severe symptoms may set in, and continue till the parasites are encapsuled, if not previously fatal. These symptoms are,—continued diarrhoea and fever; œdema of the eyelids; also pain, or at least painful sensation of weakness in the limbs; œdema of the joints, sometimes of the whole body; difficulty in moving the tongue; profuse clammy perspiration; and those patients who do not become convalescent die either unconscious, with symptoms of typhoid fever, or, in a few cases, remain conscious to the end, complaining of inability to breathe freely. The sufferer generally lies on his back, with his legs drawn up, unable to move or speak. The only important symptom of typhoid fever said to be *absent* in the disease is the enlargement of the spleen; and it is very probable that some of the so-called epidemics of typhoid fever in former days were caused by the propagation of *Trichinæ* in the human body. But the epistaxis, the pains and gurgling on pressure in the right iliac region, the rose-coloured eruption, characteristic of typhoid fever, cannot exist in trichinous disease; while the early irritation of the stomach and bowels, with œdema of the face and severe muscular pains, especially on motion, with breathlessness increasing to dyspnoea, or almost asphyxia, ought to render

the parasitic disease easy of diagnosis from enteric fever. Pneumonia or peritonitis may be suddenly fatal in mild cases.

Since the disease has become better known, a great many cases have been observed in Germany, and several cases in the United States.

"The symptoms of the disorder in the pig are said to be loss of appetite, a hoarse voice, and aversion to movement, particularly to running; and when this is attempted there is dragging of the extremities (COBBOLD). Still it is positively asserted that the animal may be infested, and yet show no signs. Cobbold mentions an instance where a pig appeared remarkably healthy, and yet the butcher who ate his flesh died of the disease. Delpech says, in his Report to the French Government:—"It is rare that any symptoms are spontaneously developed in the infected animal which would lead to any suspicion of the disorder; it has the appearance of perfect health. The butchered meat, too, looks well.* Dr. H. Jardine states (*loc. cit.*) that, in his vicinity, the opinion prevails that the *Trichina spiralis* exists in the flesh of animals affected with hog-cholera, the symptoms of this disorder being diarrhœa, swelling of the neck, stiffness of the limbs, debility, and cough; but the opinion has not been yet verified by microscopic examination.

"The Chicago Academy of Sciences appointed, in the spring of 1866, a committee of physicians, 'to examine into the facts concerning the supposed existence of trichinæ in pork raised in America. The results of their observations were, that having procured and examined portions of muscles taken from 1,394 hogs in the different packing-houses and butcher-stalls of Chicago, they found trichinæ in the muscles of twenty-eight hogs; from which they conclude, that in the hogs brought to that city, 1 in 50 is affected with trichiniasis in a greater or less degree; which would indicate that trichiniasis in pork is even more common in the North-Western States of America than in Germany.† One of the tables of the Chicago committee shows the great variation in the number of helminthes infesting the several muscles examined. An approximation only to the number existing in a cubic inch of a given muscle could be obtained. The method adopted was to count the trichinæ existing in several different portions of a muscle, each a cubic $\frac{1}{10}$ of an inch in size, and to multiply the average number to a cubic inch. Of twenty-eight specimens examined with this view, only three of them contained over 10,000 to the cubic inch,—18,000, 16,000, and 15,000 respectively. The remaining twenty-five were infested to a much less degree—from 48 to 6,000 in the cubic inch. It was calculated that a person eating an ordinary meal of the pork from which the specimen containing 18,000 to the cubic inch was taken, would soon become infested with not less than 1,000,000 of young trichinæ.‡

"With regard to the muscles of the hog which are the most common site

* See the Report officially made to the French Minister of Commerce by MM. Delpech and Raynal, *Bulletin des Académie de Médecine*, May, 1866.

† *Chicago Medical Examiner*, April, 1866.

‡ As many as 2,000,000 trichinæ have been estimated in the muscles of a man who died of the disorder; and Prof. Dalton counted in a piece of muscle (in one of Dr. Schnetter's cases), $\frac{1}{12}$ of an inch square, and $\frac{1}{16}$ of an inch thick, where they were in average abundance, twelve trichinæ, which would give, in round numbers, over 85,000 to the cubic inch; and in the portion of muscle taken from the living subject, in Dr. Voss's case, they numbered a little over 7,000 to the cubic inch (DALTON, *l. c.*) In one of Dr. Wilson's cases, which proved fatal, 104 trichinæ were counted in a piece of the rectus femoris muscle measuring $\frac{1}{12}$ of an inch square and $\frac{1}{12}$ of an inch thick, which would give nearly 180,000 to the cubic inch (CLYMER).

of trichinæ, the observations of the Chicago committee do not agree with those of European observers. In Germany, the inspectors of pork are instructed to examine microscopically nine different sets of muscles,—namely those of the diaphragm, tenderloin, shoulder, front and back of neck, extensors of the fore-arm, flexors of the leg, and the muscles of the larynx. In the trichinous-infested muscles examined by the Chicago committee, more than one-half were spinal muscles, which are not named in the German list. In conducting an examination of the trichinatus pork, the tendinous extremities of muscles should be selected, as here they are usually most numerous. The cysts are visible to the naked eye as whitish, round, or ovoid specks, sprinkling the surface of the muscle. If a very small piece of muscle is cut off with scissors, and then torn in shreds with a needle, freeing the cysts from the flesh, and these are touched with a drop of hydrochloric acid, the lime is dissolved, and the white colouring disappears; or a piece of the suspected flesh may be put into a watch-glass with liquor potassæ (1 part to 8 of water), when it becomes changed to a mucus-like, clear mass, and the capsules will be seen as sharply defined minute white specks (LEUCKART). But it is always better, if possible, to use the microscope, and trichinæ not yet encysted can only be recognised by the microscope. A thin layer of the suspected flesh should be cut out with a sharp knife, and spread over a glass plate, moistened with a drop of water, covered with a thin piece of glass, and examined by a magnifying power of 50. Their intimate structure cannot be recognised with a less power than 200" (ALTHAUS).*

The vitality of the *Trichinæ* is not destroyed below the temperature of boiling water, in the meat or in other substances, such as sausages, in which they may be located; and it is fully proved that if subjected for a sufficient time to insure that every particle has been acted upon by that degree of heat, they are incapable of further development, and are, in fact, destroyed. Salting, smoking, and toasting trichinous meat, as is usually done, does not appear to be sufficient to destroy the worms in all parts of the meat.

Picric acid (*Acidum picro nitricum*) has failed to destroy the parasite when given internally to a pig, even in quantity sufficient to cause death. In its trichinous flesh the worms were found alive (W. MULLER, of Homberg). Benzine, too, has similarly failed. *Carbolic acid* has been recommended. Any of these acids may kill or render the parasite inert if it is still in the stomach; but if its progeny have bred, and commenced to penetrate the tissues, nothing seems able to remedy the evil till the parasite becomes encapsuled. A few doses of calomel and castor-oil repeated occasionally, even although diarrhœa may be present, is advised to remove intestinal *Trichinæ*; and wet-sheet packing to relieve the sleeplessness and the copious sweats (RUPRECHT).

Numerous cases of fever and epidemics of inscrutable peculiarity are now claimed by medical writers, with much show of reason, as outbreaks of the flesh-worm disease.

For the diagnosis of *Trichinæ* in the muscles of man, Kuchenmeister has proposed to *harpoon* the muscles; but this seems a very severe operation. Welcker believes that the best place to look for them is under the tongue,

* *On Poisoning by Diseased Pork; being an Essay on Trichinosis, or Flesh-worm Disease; its Prevention and Cure.* By Julius Althaus, M.D., London, 1866.

close to the frœnum: in cats they can be easily seen in this situation. Whether it is so in man is not yet known (VIRCHOW's *Archiv.*, 1861, p. 453, quoted by Dr. Parkes, l. c.)

The *Filaria medinensis*, commonly called Guinea-worm, or *Dracunculus*, lives amongst the connective tissue of man and of some animals, resembling a long piece of uniformly thick white whip-cord. It is only known as a female, containing in its germinal sac an enormous quantity of young *Filaria*. In this country few are familiar with its appearance, or with the lesions it produces; and we therefore look for our knowledge regarding the main points in the natural history of this parasite to be furnished to us by observers in Africa or Asia, who, we hope, may fill up the gaps which still exist.

The Guinea-worm is essentially a tropical parasite. It is endemic in the hot intertropical regions of Asia and Africa, extending from Egypt, about 23° or 24° north latitude, to Sumatra and adjacent islands, as far as 10° or 12° south. But it is only in some districts within these tropical limits that the parasite abounds. For example, it is endemic in Arabia Petrea, the borders of the Persian Gulf and of the Caspian Sea, the banks of the Ganges, Upper Egypt, Abyssinia, and Guinea. Its occurrence in Guinea (although it has its common name from this place) is extremely capricious. In some districts every native who comes off to the ships seems to be affected by it; in other places in Guinea it is very rarely seen. It is unknown in America, unless the person in whom it exists has been in the places where the *Dracunculus* is endemic. The only exception is the Island of Curaçao. It is sometimes so extensively disseminated that it has been said to prevail after the manner of an epidemic.

Although this parasite rarely causes death, still it is often the cause of great distress and loss of strength to regiments quartered in those places where it is endemic.

In India, the average number of days which those affected with the Guinea-worm remain in hospital increases progressively with advancing age. During the *first period* of life (18 and under 20 years of age), the average number of days under treatment—during which period each person ineffective—has been 14·8; during the *second period* (20 and under 25 years), 16·188 days; during the *third period* (25 and under 30 years), 18·001 days; during the *fourth period* (30 and under 35 years), 22·718 days; during the *fifth period* (35 and under 40 years), 24·290 days; during the *sixth period* (40 and under 45 years), 31·620 days (EWART).

When a fatal result takes place it is generally from hectic (LORIMER) and exhaustion, consequent on the copious discharges which sometimes follow the presence of the parasite, or from abscesses forming and bursting into the abdominal cavity (EWART). Death has followed from tetanus (Drs. MINAS and M' KENZIE, *Trans. of Hyderabad Med. and Phil. Society*). Great destruction of tissues may result from sloughing; and deep-seated inflammation may attend its existence, with the formation of abscesses and deep-seated sinuses. The death of one person is recorded by Dr. Minas at Sirsa, in whom the whole body and skin was a network of Guinea-worms.

The number of worms observed in any one individual is very various.

In the majority of cases only one is present, or known to be making its exit at one time. But there are remarkable exceptions to this rule. Most of those affected have had two worms extracted; but many have had four, five, and six; and as many as fifteen have been at one time exposed to view, and many of them extracted. Dr. A. Farre mentions that as many as *fifty* worms have been met with in one person. Such cases, however, are confessedly rare even in India, where *fifteen* worms is about the greatest number observed.

Seat or Locality of the Parasite.—The lower extremities are by far the most frequently affected; or rather, the parasite most frequently tends to make its exit from some part of the lower extremities—98·95 per cent. of the parasites do so. Two cases are recorded by Lorimer, remarkable in this respect, that one gave vent to seven and the other to thirteen parasites. In the case where *seven* parasites were extracted, *two* were from the left foot, *three* from the left leg, *one* from the right leg, and *one* from the left forearm. In the case where *thirteen* parasites were extracted, *four* were taken from the left foot, *two* from the right foot, *two* from the left leg, *one* from the right leg, *one* from the right thigh, and *three* from the right forearm.

The *Dracunculus* has also been known to make its appearance in the socket of the eye, in the mouth, in the cheeks, and below the tongue (SCOTT). Dubois records its exit from the nose, the ears, and the eyelids. Dr. Kennedy records cases in which the parasite made itself apparent in the back and muscles of the loins. One preparation exists in the Museum of the Army Medical Department, in which a great number are preserved which were removed from beneath the scalp. Instances are recorded in which the worm has been found in the internal viscera. All such cases are regarded as extremely rare. It is of importance to notice, however, that both Dr. Scott and Dr. Van Someran agree in stating that the men who carry water in India, in leathern bags on their back, are infested by the *Dracunculus* on all that part of the skin that has often been wetted; while Drs. Chisholm and Scott state that the legs of persons who walk among grass (especially during the rainy season, and particularly gardeners and agriculturists, and those who are obliged to wet themselves frequently) are liable to *Dracunculi*. Some animals are said to be affected by the parasite. Forbes says that horses and dogs are so affected, and relates that a "tatoo" (a small Indian horse) was exhibited at Dharwar, having a *Dracunculus* protruding from its right hind fetlock. The parasite was of the usual size, and made its appearance as a boil; and no difference could be perceived in any respect in it from the *Dracunculus* which infests man. Clot Bey remarks that dogs are also sufferers; but on this head information is greatly to be desired. Assistant-Surgeon Adam Taylor, of the 1st Goorkha Light Infantry, wrote me from Bootan, in 1866, that during the rains of August, 1862, he being in medical charge at Hissar, a sandy district about seventy miles from the desert of India, he saw a favourite bull-terrier with swollen feet; and from a fluctuating tumor in one of the hind feet he liberated four ounces of serum, and eighteen inches of a Guinea-worm; four inches more the next day; and the remainder, about fifteen inches, was wheedled out by a native barber the following day. The worm was exactly the same as those found in man; and he has

repeatedly seen *Dracunculus* in horses, and heard of their existence in camels. He believes the *habitat* of the parasite is sandy soils, and not water.

Migratory Powers exhibited by the Guinea-Worm before Extraction.—As a rule the patient is unconscious of the presence of the *Dracunculus* till it is nearly mature and ready to make its exit. Dr. Smyttan relates the cases of two officers, in one of whom the *Dracunculus* could be felt, and traced with the fingers like a cord under the skin at the top of the shoulder. By and by it made its way to the elbow, where it was not less distinctly felt, and in a few weeks it gradually worked its way to the wrist, whence it was extracted. In the other case the Guinea-worm was observed under the skin inside the biceps, and about the middle of the upper arm. It then passed round the elbow joint, and down to the middle of the forearm, then back to the region of the inner condyle of the humerus, whence it was removed. It was three months engaged in this migration. Dr. Paton records similar cases (*Edin. Med. and Surg. Journal*, 1806, Vol. II., p. 151); and Dr. Morehead says of his men, that when they had felt the Guinea-worm in the thigh, in the first instance, it had subsequently been ejected from the foot. He has distinctly noticed the corded feeling of the worm below the skin, and when he examined the same part the next day it was entirely gone. Dr. L. W. Stewart, of the Madras Medical Service, relates a very distressing instance of this kind which happened to an officer, from whose scrotum a Guinea-worm fifteen inches long had already been extracted. Ten days afterwards he experienced an unpleasant sensation in the posterior aspect of the left thigh. Day by day the sensation shifted lower down, till it reached the popliteal space. A few days later the sensation was experienced in the calf. Hitherto nothing had been visible; but at the end of sixteen days from the first sensation in the thigh, the convolutions of a Guinea-worm could be distinctly traced at the outer side of the ankle-joint. Dr. Stewart now wished to cut down and extract the parasite, but the evening was too dark, and he delayed till the following morning. By the morning visit, however, the parasite had again taken up a new position in the deeper muscles of the foot. Not a trace of the worm could be recognised near the ankle-joint. Many abscesses now formed, and severe inflammation of the foot resulted, which confined the patient for three months before he was free of this wandering parasite. Dr. Ewart has seen the worm change its position from the upper part of the lateral aspect of the thorax to the groin in the course of twenty-four hours; but he has never seen it travel from below upwards (*Indian Annals*, Vol. VI., p. 490, July, 1859).

Structure of the Dracunculus.—It is often a matter of extreme difficulty to extract the whole worm without breaking it, and on account of its remarkable elasticity (for it may be extended to twice its apparently natural length), good measurements of any large number of worms are not easily obtained (BUSK). Of forty Indian specimens, Ewart gives the average length at 25·25 inches, the shortest being $12\frac{3}{4}$ inches, the longest 40 inches. Clot Bey records their length at from 6 inches to 4 feet in Egypt. Carter gives their dimensions in India at about 28 inches long, $\frac{1}{9}$ of an inch in diameter. He has dissected five.

Busk gives the dimensions at from 4 to 6 feet, and $\frac{1}{12}$ of an inch in diameter; and he has made out that it grows in the human areolar tissue at the rate of about an inch a week. Dr. H. C. Bastian, Professor of Pathology, University College, has recently read an account of the anatomy of this parasite at the Linnæan Society; and from the records of these excellent observers we have now a very complete account of the anatomy of the Guinea-worm. The anterior end of the worm (Fig. 20)

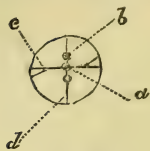


Fig. 20.*

may be recognised by a "punctum" in its centre, $\frac{1}{2000}$ of an inch in diameter; surrounded by rugæ in circles, the external of which was $\frac{1}{271}$ of an inch in diameter. Above and below are two papillæ opposite each other, with a transparent area in the centre of each. These are rather oval, $\frac{1}{475}$ of an inch in diameter, with a transparent area of $\frac{1}{1900}$ of an inch. Besides these, two lateral tubercles exist, much smaller, more indistinct, and farther from the punctum than the upper and lower papillæ. They are $\frac{1}{1900}$ of an inch in diameter. It is difficult to obtain a good view of the head; for as it is the first part to protrude through the skin, it is usually rubbed off or destroyed by the treatment adopted for extraction.

Great varieties in form are presented by the tail or posterior end of the worm (Fig. 21). The remains of the attenuated extremity of the young *Filaria*, being more or less persistent in the form of a hook or

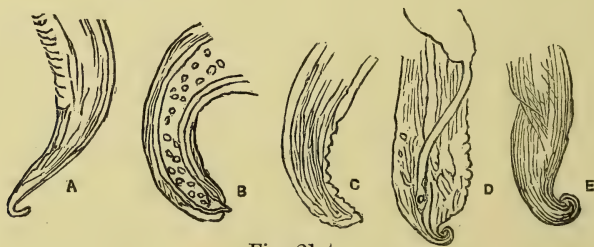


Fig. 21.†

spikelet, was believed at one time to be the penis of a male; and such specimens as showed such spikelets have been mistaken for male Guinea-worms. All these forms have been found in specimens containing living young ones (proligerous). All are females that have yet been found, and no males are known to exist in the human body. The strength of the tissue of the *Dracunculus* is such that a loop of the parasite will suspend a weight of $11\frac{3}{4}$ ounces (SCOTT), and it is elastic to a remarkable degree. On opening the body, two longitudinal muscular bands are seen on the dorsal, and two on the ventral aspect, running from end to end, while circular or transverse rugæ mark the worm over its whole extent;

* Diagram of the head or anterior end of the Guinea-worm; showing, a, Punctiform mouth, $\frac{1}{2000}$ of an inch in diameter; b, upper large papillæ; c, one of small lateral papillæ; d, one of four crucial white lines meeting at the mouth, and occupying inter-muscular spaces (H. C. BASTIAN).

† Various forms of the caudal end of the Guinea-worm:—A, B, C. After BUSK—all of them proligerous; D. After CARTER; E. After GREENHOW.

and these are approximated or parted as the worm is contracted or extended. The body of the worm (Fig. 22) contains an alimentary canal, which commences at the "punctum," and terminates in the concavity of the tail end. It is of a yellow colour, nearly uniform in size throughout its extent, and in its course through the body winds several times round the genital tube (BASTIAN). No outlet has yet been detected. It is distinct from the tube containing the young (FORBES.)

The genital organs consist of a large uterine sac or tube, occupying nearly the whole length of the worm, and terminating abruptly at either extremity in a much smaller tube (probably ovarian), about three-quarters of an inch in length. No vagina or vulva can be discovered (BASTIAN). The whole extent of this uterine sac or capsule is crowded with innumerable young, and, with the exception of a transparent half-inch or so of the worm, the whole extent of the parent seems to be a *uterus*, a *matrix*, or a *proligerous capsule*, carrying a countless offspring, to which no parturient female of any animal can be compared for productiveness; and from the fact that no inlet has ever been discovered to the genital organs, and from various other circumstances, Dr. Bastian has endeavoured to show that this innumerable progeny has been produced by a process of *parthenogenesis* similar to that which occurs in the *Aphis*. "Insects in general come from an egg; then turn to a caterpillar, which does nothing but eat; then to a crysalis, which does nothing but sleep; then to a perfect butterfly, which does nothing but increase its kind. But the *aphis* proceeds altogether on another system. The young ones are born exactly like the old ones, but less. They stick their beaks through the rind, and begin to draw up sap when only a day old, and go on quietly sucking away for seven or eight days; and then, without love, courtship, or matrimony of any kind, each individual begins bringing forth young ones, and continues to do so for months, at the rate of from twelve to eighteen daily." (*Entom Mag.*, No. III., 217.)

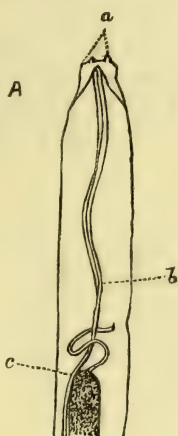


Fig. 22.*

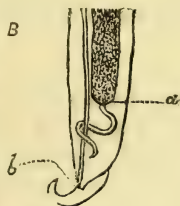


Fig. 23.†

If a living worm recently extracted be well lit up by an Argand lamp,

* *A*.—Anterior extremity of worm, slit open and magnified, showing, *a*, Upper and lower cephalic papillae in profile; *b*, junction of cesophagus with intestine, and constriction of peritoneal sheath; *c*, anterior termination of uterus, with short ovarian tube. *B*.—Posterior extremity of worm, slit open and magnified in same way, showing its hook-like termination; and, *a*, Posterior termination of uterus with ovarian tube; *b*, termination of intestine (BASTIAN).

† Anterior extremity. The ovisac *a*, is protruded, dilated, and contains young; *b*, a funnel-shaped sheath surrounding the protruding ovisac (GREENHOW).

the hair-like filaments may be seen in motion with a good simple lens; and if a section be made across the parasite after it has been hardened in glue, the young may be demonstrated *in situ* (Fig. 24).

When the animal is mature, and presenting its head through the skin, it protrudes the extremity of the proligerous capsule through one of the small papillæ or puncta, carrying forward a prolongation of something in the form of a loose corrugated sheath (Fig. 23). It gradually assumes the form of a dilated vesicle filled with limpid fluid—the contents of the proligerous capsule—containing flocculent granular matter and young Guinea-worms. If kept moist, the full-grown parent will live many hours; and in this state the young will live till the parent begins to decompose; and when the head end of the worm during its extraction may have been dried up for several days outside the wound, the remaining part with the young still remains alive, and will survive after having undergone a considerable degree of drying up. They are exceedingly numerous, and constitute the bulk of the contents of the parent's body. Each young one may be said to consist of a body and a tail, hair-like and finely pointed. The body constitutes $\frac{3}{5}$ and the tail $\frac{2}{5}$ of the whole length. The anterior extremity has a blunt end, with a rounded oval orifice communicating with a cavity occupying about one-half of the whole length of the body, and terminating cœcally.

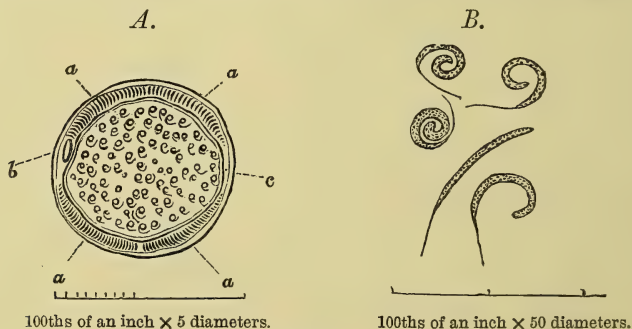


Fig. 24.

Symptoms of the Guinea-Worm in the Human Body.—As a parasite in the human body it may be studied during two periods of existence; but from the beginning to the end of its cycle of development its history embraces at least *three*, if not *four* phases:—

(1.) During the first period of its life in the human body the parasite is latent, residing in the connective tissue at variable depths from the surface. During this period it does not exert any irritating influence on the surrounding tissue, as has been shown by dissections (BUSK).

(2.) The second period of existence comprehends that of ripening or maturation of the worm and its progeny, when the worm makes itself felt, and begins its exit through the skin. This period is marked by characteristic symptoms. These are a pricking, itching heat, which is felt at the part where the worm lives, seldom amounting to pain till after the lapse of three or four weeks. A small vesicle forms over the part,

and this immediately precedes the appearance of the anterior end of the worm. Dr. Scott was himself a sufferer, and writes feelingly on this point (see *Med.-Chir. Review*, 1823). This itching may happen before any vesicle forms; and when the vesicle forms, it rapidly enlarges—so rapidly that in a few hours it attains the size of a good large filbert (LORIMER). If it is opened early, it is seen to contain a clear and limpid fluid (the fibrinous serum of irritation?); but if untouched for a day or two, its contents become turbid, and sometimes bloody, from the rupture of the proligerous sac, and the discharge of the young *Filaria* amongst the serum. These greatly add to the irritation; so much so, that when the cuticle is removed, an angry-looking ulcer is exposed, in the centre of which the parasite may be seen presenting itself, *with a thin transparent tendril about an inch in length hanging from its point*. After the appearance of this vesicle or blister, it is sometimes weeks before the worm protrudes itself. The contents of the blister, when turbid, are a discharge from the tube of the animal; and the best way to procure the young Guinea-worm saline for microscopic examination is to lay open this vesicle before the delicate membrane of the proligerous capsule bursts. After the escape of the serum from the vesicle, the delicate transparent membranous tube, or *cul de sac*, is sometimes protruded from the extremity of the worm; and if cold water is *gently poured in a constant stream* upon this protrusion, the dilatation and protrusion increase, till an innumerable quantity of young is ejected from the ruptured orifice of the dilated tube. Forbes says that he has often repeated this experiment; and in one instance the transparent tube was again retracted within the limb, after three emissions of young Guinea-worms. On the following day the tube was found again protruding as before; and the same result (namely, emission of young) followed the gentle application of the stream of water. The animal will emit its young daily in this way for some time; and when it ceases to emit them, it is then time to begin the extraction of the parasite (FORBES).

The Period of the Year when Dracunculus is most prevalent seems to vary considerably in different parts of India, and the probable causes of these differences are of great interest in regard to the origin and spread of this parasite. At Madras and its vicinity Guinea-worm annually appears with greater prevalence during the hot season (LORIMER), comprehending February, March, April, May, and June. At Dharwar and its vicinity the admissions to hospital for Guinea-worm generally commence in April and May. At this time water is scarce, every tank is dried up, wells yield a scanty supply, and the natives are obliged to remain at the bottom of the wells by turns, till the required supply is obtained; and when the monsoon sets in (rainy season), the admissions gradually increase through June, July, August, and September. The increase of the disease amongst soldiers or residents advances with length of residence, generally during the rainy season. In the Bombay and Matoongha districts the admissions to hospital begin in May or June (irrigation of fields by the natives being common at this time); but it chiefly prevails during the rainy months of June, July, August, and September, and is rare after October (SMYTTAN). Its annual endemic prevalence in the Carnatic villages is in December, January, and February, during which time more than half the inhabitants are affected. Dr. Morehead's experience at

Kirkee and vicinity gave March, April, May, June, and July as the months of gradual increase and prevalence; and September, October, November, December, and January as those of comparative exemption. In the Bheel districts Guinea-worm begins to increase in frequency in February; it is *four* times as frequent in March, and *six* times as common in April, as in February. It reaches the monthly prevalence in May. It prevails to a great extent in June, and continues to be common throughout the monsoon months of July and August. During September, October, November, December, and January it is least of all prevalent. The half of the year comprising the hot and rainy season is, therefore, the period when *Dracunculus* abounds, abruptly commencing with the initiation of the former, and terminating more abruptly still with the exhaustion of the monsoon in September (EWART). Thus all the records agree in assigning to this parasite—(1.) An annual periodic recurrence; (2.) Periods (annual) of progressive increase and subsidence; (3.) A probably fixed latent period of residence in the connective tissue—a period of incubation—of not less than twelve months (LORIMER, MITCHELL); or of twelve to eighteen months (BUSK).

The Guinea-worm never makes itself manifest in the human body before the second season of residence in the places where it is endemic, a complete season being requisite to mature the worm. There are some remarkable cases which fix the period of incubation in a very decided way. For example, it is related of a gentleman well known to be extensively acquainted with natural history, that when he was travelling in the Northern Circars, the tents were pitched near a tank of bad repute. He was accompanied by five friends, who, against his advice, bathed in the tank. Each of these five persons subsequently became affected with the Guinea-worm (MITCHELL, Supplement to the *Madras Times*, of December 18, 1861, and January 13, 1862). In the Indian annals many accurate accounts are given which fix the period of incubation at about twelve months.

Geological Features of Locality and Soil where the Dracunculus is Endemic.—Evidence of a circumstantial kind tends to connect the parasite with something geologically characteristic in the *soil, mud, moisture, or water* of the places where the parasite is endemic; yet information is still very imperfect on these points. All the districts where *Dracunculus* prevails are composed of the secondary trap rock—*i. e.*, of igneous formation, as in the villages of the Deccan and Northern Concan, where the parasite is indigenous. In the country between the Western Ghauts and the sea-coast, where the parasite is rare, the soil is a conglomerate ironshot clay, of a red colour. Chisholm's investigations on this point led him to the conclusion that the districts where Guinea-worms abound are of volcanic origin, with an argillaceous soil, holding much moisture, impregnated with salts or percolated by sea-water. Dr. Carter's evidence as to soil is, that the parasite abounds where the soil is a decomposing trap, of a clayey consistence, and of a yellow colour. Every regiment which has occupied the lines at Secunderabad, "near the large tank called the 'Hausen Saughur,'" has suffered from the *Dracunculus* (LORIMER). The cause of the disease exists *in or near* the lines at that place; and the soil is marshy which borders on the tank. The experience of the 19th, the 4th, 5th, 1st, and

35th Regiments of Native Infantry all fix the locality of the Guinea-worm germs to be "*in or near these lines.*" For example, the 19th Regiment arrived at Vepery on the 20th May, 1838. It had been free from *Dracunculus* for five years before: twelve months after its arrival twenty-eight cases of Guinea-worm appeared, and several cases amongst the followers and children. The 45th Regiment occupied the same lines previous to the arrival of the 19th Regiment; and the disease appeared amongst them at the same season of the year, and after twelve months' residence. The Guinea-worm had not been amongst them for many years before. At Perampore (in the 1st Regiment, N. I.) it manifested itself, after twelve months' residence, in March, April, and May. For many years previously Guinea-worm had been unknown in the regiment. Those who suffer most in cantonments are those who use water of the filthiest kinds.

In some form or another the Guinea-worm has an existence in moist earth, sand, and mud; and it is probable that the hair-like worms found by gardeners in India coiled up together may be the young *Filaria* of the Guinea-worm in sexual congress; whose progeny in process of *parthenogenesis* as *Zoöspirms*, or as filiform female worms (like the Tank worm of CARTER), make their way into the body. It is known that the *Gordius aquaticus*, when young, enters the bodies of large water beetles, and at a certain stage of life it leaves its abode in the beetle and goes into the water, where it becomes a variety of *Tank* worm. It appears that there are white and brown *Tank* worms—nay, that there are no fewer than seventeen species of minute *Filaria* (CARTER, MITCHELL); and some say that all *Tank* worms are white at first, but become black after a time in the water (GUNTHER). Observations are greatly wanted on these points. According to observations collected by Pallas and quoted by Vogel, it appears that even in Europe thread worms like the *G. aquaticus*, common in stagnant water and moist earth, can in certain cases infest the human subject (*De Infestis Viventibus intra Viventia*, p. 11).

The most obscure and incomprehensible parts of the history of this parasite are—(1.) The phase of its existence, and that of its young after it leaves the body of man; and, (2.) The future life of the young, and their sexual differentiation. Although the parasite may be removed in several ways by surgical interference (either by cutting down upon it, or, after it begins to show itself, to commence winding it on a stick, gently pulling a portion of it out every day), there is a natural termination to all diseases; and it is still a fair subject of inquiry as to what becomes of the *Dracunculus* if left to itself, and its expulsion unaided by art. How would it be expelled, and what becomes of the progeny? How does it come that they are ever placed in circumstances where they lead an independent existence, becoming sexual and multiplying their kind? In reply to these questions it is to be observed that there are undoubted examples of the *spontaneous evolution* or *expulsion* of the Guinea-worm. Scott once observed about five inches of the worm to start suddenly out, firm, elastic, and spirally twisted like a cork-screw, showing evidence of resistance to a progressive force from behind. So firm was the parasite that it supported itself for a little time perpendicularly to the limb. It

is only when the animal dies that great mischief happens to the part where the parasite is. Then and there it acts as a foreign body; but alive it does not cause disturbance (JOHN HUNTER, *On the Blood*, 4to, 1794, p. 208). The part first protruded is the head; and its future progress, though slow and invisible, becomes in time very obvious (SCOTT). As an example of its spontaneous evolution or expulsion, Dr. Forbes relates that on one occasion eight Sepoys were admitted with Guinea-worm, and all of them had a characteristic vesicle on the ankle. These vesicles were opened on the fourth or fifth day. The loose skin was cut away with scissors, and a stream of cold water was poured daily on the part. Under these circumstances the young were daily ejected from the proligerous tube of the parent parasite, and continued to be so for fifteen to twenty days. After this time a watery fluid only was emitted, without any young, but sometimes containing particles of a white flaky appearance, which continued two or three days longer. The Guinea-worm then became flaccid, and was discharged spontaneously, without pain or swelling. The only exception was in one case, where the worm was constricted by the pressure of a band of areolar tissue, which led to retention of the young and sloughing. Dr. Kennedy relates an anecdote which has an interesting bearing upon the spontaneous evolution and the probable future of the Guinea-worm after expulsion. "In 1791, when marching up the Ghauts with a Sepoy battalion, an African soldier stepped out of the ranks, and requested permission to go to a rapid running stream of water near by, in order to relieve himself of a worm in his ankle, after his own fashion. The man unbound a bandage from his foot, loosened the worm (of which a part was extracted) from the cloth round which it was secured, and plunged his naked foot into the current of the stream. The constant but gentle force of the running water was sufficient to stimulate the worm to come forth, and it was extracted almost immediately." Another custom, recorded by Dr. Lorimer, illustrates the spontaneous evolution, and points, at the same time, to the probable future of the Guinea-worm. He says, "Many people belonging to the bazaars in the vicinity of the lines, affected with the parasite, came for the express purpose of extracting the worm to the same tank where the men of the regiment bathe. The people so infested swim about in the water with the worm hanging loose, drawing the limb quickly backwards and forwards through the water, and from side to side, till expulsion is effected." Galvanism renders it more easy of extraction. The natives do not believe that they get the parasite from bathing in the water; on the contrary, they believe they rid themselves of it by bathing.

In these and similar cases the remains of the parent, being carried away in the stream, decay, and so give freedom to her immense brood of young. The water seems a natural or congenial stimulus to the parent Guinea-worm, and sooner than anything else induces her to leave her position in the human body, and so to extricate herself, perhaps by stimulation of the muscular structures. This water method of extraction was also recommended by Dr. Helenus Scott, of Madras, (*Edin. Med. and Surg. Journal*, Vol. XVIII.)

Vitality of the Parasite in Water.—It has been stated that young

Dracunculi die in four, five, or six days, if placed in pure water from well or tank (and that is the case with many animals), simply for want of food. Water *not pure* is, no doubt, the proper element for them (MITCHELL). Those artificially kept in impalpable red clay, partially covered with water, and exposed to the sun, were found alive after fifteen, eighteen, and twenty-one days, burrowing into the fine soft and ochry mud.

Forbes experimented on two pups five or six months old. He poured down their throats water containing the young Guinea-worm *Filaria*. After three minutes the first pup became uneasy, sick, and vomited; the watery part of which was found to contain the animal still alive. Four hours after this the pup was killed, when abundance of *Filaria* were seen in the mucous of the stomach and duodenum; but none showed signs of life. The other pup was killed twenty-four hours afterwards, but none were alive, although abundant in the mucus. Lorimer tried upon himself and others if the parasite could be propagated by inoculation of the young *Filaria* emitted from the parent's orifice. Five besides himself were inoculated. He remarks that *he is sorry to say* they did not hatch in any, although in his own case he put them in their favourite place—namely, the *foot and ankle*. Such experiments were not likely to succeed, because the parasites were introduced under unnatural circumstances. Inflammation and pus are inimical to the life of the worm. Besides, it is most probable that they go through another stage of existence, and become sexual, before they enter the body of man or animals; for it is *only females*, and these *impregnated ones*, which are found in man. Thus Guinea-worm is propagated by a female and impregnated *zoöspERM*, and not directly from either the young of the full-grown female Guinea-worm or from tank worms (EWART, *Indian Annals*, Vol. VI., July, 1859).

Examination of Water, Mud, and Tanks.—In the months of August and September, 1837, Dr. Forbes examined several of the tanks in the vicinity of Dharwar, and found the mud on their banks, and in half dried beds, abundantly supplied with animalcules (*Filaria*), some of them very much resembling those produced by the Guinea-worm when infesting the human limb. Their vermicular motion in the water is exactly the same; their general appearance is the same; and they are active and equally numerous. The point of a penknife inserted into the mud will raise up abundance for examination. They are most numerous where the water assumes a variegated appearance, with a pellicle floating on its ochry surface; and the fine, soft, impalpable mud just above water-mark contains most. The best time to find them is about three or four o'clock in the afternoon. Two kinds may generally be detected in the soft mud: one kind is seven or eight times the size of the young *Filaria* Guinea-worm, the other exactly resembles them. The larger one may be the more mature form of the progeny after becoming sexual. The smaller one may be the first-generation born of that sexual progeny, whose females, being fecundated, enter the body of man in this young and minute condition.

The habit of the tank worm is to bury itself under any organic *débris* that may be in the water in which it is found; and if it be disturbed, it will immediately seek a hiding-place, nor rest until again covered. This implies that its proper habitat is the bottom of tanks, wells, or other reservoirs, among the decayed and decaying organic matter. It may be

assumed that the water-carriers referred to by Dr. Morehead were Army Bheesties, who as such probably had access to good wells (Dr. Morehead having found that Guinea-worm was not more common among them than among other people); and as the tank worm, habitually resident in the mud at the bottom, would only be disturbed when the water became very low, and would go back again to its retreat, if possible, the fact of water-carriers being as little affected with Guinea-worm in the upper part of the body as other people does not carry so much weight as at first it would seem to do, and as it would in reality if the tank worm was in the habit of swimming at the surface like many other aquatic animals. It has not been said that the worm finds its way into the body by any of the natural cavities of the body, such as the alimentary canal. On the contrary, it is supposed that the water may be drunk with impunity, as known by experience, and from the experiments of Forbes, already noticed.

The young *Filaria* can work its way into a proper receptacle by its pointed extremity, "which is a long cone, ending in a point so inconceivably fine that the point of a cambric needle is a large marline-spike in comparison with it." But notwithstanding its exceeding tenuity, it appears tolerably rigid; and as the proper receptacle referred to is one of the sudoriparous ducts, a ready-made aperture exists for a distance quite long enough to contain so small a creature; and it is by no means inconceivable to one who has seen its active exertions, that it should be able thus to hide itself in a foot or leg kept for some time in the water. The well-known native custom of going into a tank to take water favours the entrance of the *Filaria*. In these tanks water-carriers may often be seen standing for five or ten minutes at a stretch, chatting and washing themselves. They of course stir up the bottom mud, and, if the tank worm be there, and is the origin of the Guinea-worm, they certainly afford it every opportunity to effect a lodgment. One circumstance which makes this the probable mode of entry is, that natives are much more subject to attack than Europeans. Thus the evidence is very strong which refers the entrance of the parasite to bathing, walking, or lying on moist places where the tank worms abound. Greenhow states that the Sepoys of the Maiwara Battalion bathe in and drink the water of a well sunk in the limestone rock, which generally contains about twenty-eight feet of water, clear and sweet; while the prisoners of the jail at Beaur use similar water from another well; but they never bathe, which the Sepoys do every day. The result is, that *Dracunculus* is much more prevalent among the Sepoys, compared with the prisoners, in the proportion of three to two. Again, amongst "Puchallies" the numbers affected are four times as great as among the men of the regiments. The former frequent the tanks more than the men of the regiments.

Generation and Propagation of the Guinea-Worm.—The following periods may be recognised in its natural history:—

(1.) It is probably got by bathing in tanks or places where the young and impregnated females abound. (2.) A period of maturation in the human body takes place. (3.) A time favourable for extraction comes, when the animal seems to seek delivery from its imprisonment, to fulfil a new law of its existence. The adult female animals thus perish annually. It is necessary they should die, that the young may live; and, indeed, the

Guinea-worm of the human body is not adapted to live. It has no functional arrangements for continuing life after expulsion from the body.

Men being exposed to the cause about the same time, the period for extraction will arrive about the same time in all, but with just sufficient variation (as to time) to suggest the idea of contagion (SCOTT, *Med. and Surg. Journal*, Vol. XVII., p. 99). But the idea of contagion or infection from one man to another (as BRUCE, M'GRIGOR, and PATON wished to establish) is quite untenable. The evidence is all the other way. In Paton's cases on board Her Majesty's ship "Cirencester," from 30th May, 1805, to 9th August of the same year, the origin of the disease is quite traceable to the preceding July and August, when the ship lay in Bombay harbour (*Med. and Surg. Journal*, 1806, Vol. II., p. 151). Sir J. M'Grigor's cases in the 88th Regiment, and the absence of Guinea-worm among the artillery on shipboard, related in his medical sketches, were not fully investigated. We have no account of the water supply previous to embarkation. Afterwards he wrote a paper, or rather an account of the sickness in the regiment from all diseases, in the *Edin. Med. Journal*, Vol. I., p. 270, and from this it appears that the regiment had been quartered in the Fort of Bombay, which is partly surrounded by a wet ditch; and several months after leaving this place most of the cases of Guinea-worm occurred. Bombay is well known to be extremely infested with Guinea-worm. There is as much foundation for believing *Dracunculus* to be contagious as that a thorn in the foot is contagious (MOSELEY). The parasite is known to occur in persons who have neither eaten nor drank in the countries where it is endemic, but who have exposed themselves to its moisture and its mud (RUDOLPHI). The moisture contained in native canoes is sufficient to have carried to a ship off the coast the germs of the Guinea-worm, which find their way into the seamen of the ship, who are in the habit of going into these canoes with bare feet.

Problems for Solution.—More than forty years ago Dr. Scott suggested that a patient and careful investigation of soils and waters ought to be made wherever *Dracunculus* is known to be endemic, and especially the soil round brackish wells and the beds of tanks. Morehead, in 1833, recommended that the following points be attended to, namely:—(1.) Geological structure of the ground and nature of the site generally; (2.) Nature of soil, wells, and well-water; (3.) Nature of rocks through which wells are sunk; (4.) Abundance or scarcity of water; (5.) Seasons of increase or decrease of the disease; (6.) Opinions of natives.

I desire very much to obtain specimens of Guinea-worm taken from the dead body long before the parasite arrives at maturity; such specimens may be got in the dissecting rooms, or at *post-mortem* examinations in India.

The occurrence of Guinea-worm is sometimes defined by a distance of a few miles. So it is with many Algæ and minute water animals and plants as to habitat.*

* My friend, Dr. H. C. Bastian, Esq., Professor of Pathology in University College, London, has furnished a most interesting account of the anatomy of the Guinea-worm to the Linnean Society, and has been kind enough to furnish me with drawings of his observations. He writes to me as follows:—"Since I saw you last I have discovered several species of Carter's 'tank worms' in soft mud, &c. (at Falmouth); that is, small *Nematoids*, agreeing in almost every respect with those found by him in

Filaria oculi (*F. lentis*).—Length, $\frac{2}{10}$ to $\frac{6}{10}$; width, $\frac{1}{500}$ of an inch. The body is thick posteriorly, filiform, and ending in a pointed tail, transparent, and partly coiled up in a spiral form. The *alimentary canal* is surrounded by the folds of the oviduct. This *Filaria* is very imperfectly known, and the female only has been seen. It was detected by Nordmann in the *liquor Morgagni* of the capsule of a crystalline lens of a man whose lens had been extracted for cataract by the Baron Von Gräfe. In this instance the capsule of the lens had been extracted entire; and upon a careful examination half an hour after extraction, there were observed in the fluid two minute and delicate *Filarie* coiled up in the form of a ring. One of them presented a rupture in the middle of its body (probably made by the extracting needle), from which rupture the intestinal canal was protruding. The other was entire, and measured about $\frac{6}{100}$ of an inch in length. It presented a simple mouth, without any apparent papillæ, such as are seen to characterise the large *Filaria* which infests the eye of the horse; and through the transparent integument could be seen a straight intestinal canal, surrounded by convolutions of the oviducts, and terminating at an incurved anal extremity (OWEN, p. 64).

A *Filaria oculi*, vel *lachrymalis*, has been described as not uncommon among the negroes on the Angola coast, where it is called *loa*; also at Guadaloupe, Cayenne, and Martinique. Its length is $1\frac{4}{10}$ to $1\frac{9}{10}$ of a line. It is a filiform, slender worm, pointed at one end, obtuse at the other, tolerably firm, and of a white-yellow colour. This parasite has been considered a *Strongylus* by some, by others a young Guinea-worm, and by others as an *Oxyuris vermicularis*.

The **Strongylus bronchialis** was first discovered by Treutler, in 1791, infesting the enlarged bronchial glands of an emaciated man. The parasite is cylindrical, slightly narrowed anteriorly, filiform, but somewhat compressed at the sides, semi-transparent posteriorly, and of a blackish-brown colour. It measures from half an inch to three-quarters of an inch in length.

The **Eustrongylus gigas** is fortunately rare in man, though common in a great variety of animals, such as weasels. It inhabits the kidney, destroying the substance of the organ, the walls of which become the seat of calcareous deposits.

The **Sclerostoma duodenale** is known to be common throughout Northern Italy; and according to Pruner, Bilharz, and Griesinger, it is so remarkably abundant in Egypt, that about one-fourth of the people are constantly suffering from a severe anæmic chlorosis, occasioned solely by the presence of this parasite in the small intestines. "Its length is about one-third to half an inch, its width about one-twentieth of its length. Its head has a

Bombay. The more I see of these, the more thoroughly am I convinced of the undoubted relationship existing between them and the Guinea-worm, coinciding as they do in their anatomy even to minute details, and in many respects where there is a salient distinction between the anatomy of the *Dracunculus* and that of the *Ascarides*. One which I sketched to-day had an exsertile, rigid, sharp-pointed œsophagus.

"The great difficulty in the theory is to account for the fact of the localisation of the disease, whilst these animals are probably so widely spread; and I suppose it is one particular species which is limited in its diffusion; but I suspect that many of those others will hereafter be discovered as parasites in animals or vegetables. The *Vibrio tritico* I have examined, and find it to be a worm essentially similar; and Dr. Cobbold tells me that he has found a long thread-like worm in the subcutaneous tissue of the back of a water-bird. The whole question wants working out."

round apex, and its extremity, which is bevelled at the expense of its posterior surface, is provided with hooklets that occupy converging papillæ. The mouth contracts, to open into a thick muscular pharynx, which, widening as it passes downward, ends, after occupying one-seventh of the body, in the intestine. The sexual differences of the male and female are very interesting. Its pathological significance is chiefly due to the hæmorrhage caused by these parasites, which are often present in thousands between the *valvulæ conniventes* of the duodenum, jejunum, and ileum, and not infrequently in the submucous areolar tissue. In short, the physician practising in Egypt must never forget that the chlorosis of this climate is often the result of repeated and small hæmorrhages from the intestine, caused by these parasites. Turpentine, as Griesinger points out, promises to be the best remedy both as a styptic and as a vermifuge," (*Brit. and For. Med. Chir. Review*, l. c.)

The Oxyuris vermicularis was known to Hippocrates, and is one of the most troublesome parasites of children, and occasionally of adults. It is a minute, white, thread-like worm, the male being about a line and a half in length, and the female five or six lines. They inhabit chiefly the rectum, where they are often found in clusters, rolled up in balls of considerable size, and from the rectum may creep into the vagina or urethral orifice. Sometimes they give rise to profuse and exhausting bloody discharges from the vagina. The eggs have embryos developed within them prior to their escape from the parent; and in this respect they differ from the *A. lumbricoides* and the *Trichocephalus* on the one hand, and from the viviparous *Dracunculus* on the other. In this character, however, they resemble the *A. mystax*. In all probability the young escape from the eggs soon after the latter are expelled, or migrate *per rectum*; and, like others of the *Nematelmia*, they gain access to the human body with our vegetable food or water whilst still in a sexually immature condition.

Filaria sanguinis hominis.—

This round worm was discovered in 1869, by Dr. Timothy R. Lewis, of the Army Medical Service, in the urine of patients in the General Hospital at Calcutta suffering from chyluria; and a report on cholera, in which this discovery was noted, was communicated to the Government of India in that year, and published in 1870 (*Sixth Report of the Sanitary Commissioner for Government of India for 1869*, p. 143), and in the same year (1870) a specimen of the worm was sent to Netley. In the following year (1871) the same filaria was discovered in the blood by Dr. Lewis, and several specimens were sent to



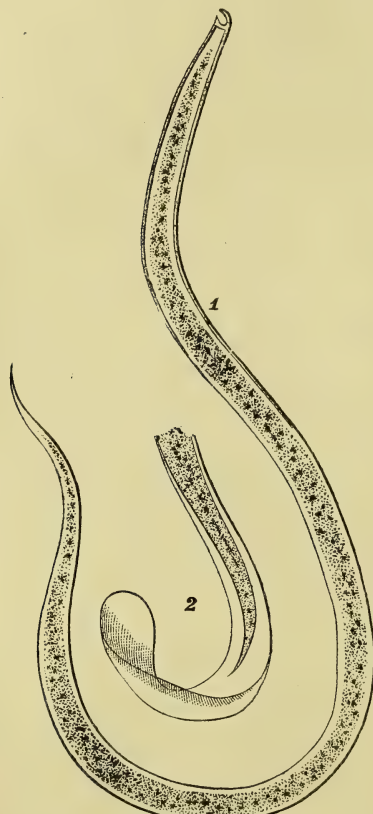
1000th of an inch \times 600.

Fig. 25.*

* Embryo of a round worm imbedded in gelatinised matter in "chylous urine" (after LEWIS).

the late Dr. Parkes for the museum at Netley. The condition of those in whom this parasite abounds is one in which the whole blood is infested with living active worms about $\frac{1}{75}$ of an inch in length, and with a transverse diameter of about $\frac{1}{3500}$ of an inch—a condition in which they are persistently so ubiquitous as to be obtained day after day by simply pricking any portion of the body—even to the tips of the fingers and toes of both hands and both feet of one and the same person with a finely pointed needle.

The *Filaria sanguinis hominis* is the name first given to this hæmatozoon. Its appearance is very characteristic. On first being removed from the body in a drop of blood, it moves about incessantly, coiling and uncoiling itself unceasingly, lashing the blood-corpuscles about in all directions, and insinuating itself between them. Each filaria is enveloped in an extremely delicate structureless and transparent tube, closed at both ends, within which it is capable of elongating or shortening itself. That it is so inclosed is a proof that in such a stage of existence it is immature, with its “home” in the blood. No ova have been discovered. It has no visible means of perforating the tissues, depending on the currents of the blood for its transference from place to place. The following table by Dr. Lewis shows the relative size of the *Trichina spiralis*, the embryo Guinea-worm, and this hæmatozoon:—



1000th of an inch \times 600.

Fig. 26.*

	Average Breadth.	Average Length.	Proportion of Breadth to Length.	Aspect of		Tail to Total Length.
				Head.	Tail.	
<i>Trichina spiralis</i> (human muscle),	$\frac{1}{700}$ in.	$\frac{1}{25}$ in.	1 to 28	Pointed	Blunt	
<i>Dracunculus</i> (embryo),	$\frac{1}{1000}$ in.	$\frac{1}{32}$ in.	1 to 31	Rounded	Acutely Pointed	1 : 3 $\frac{1}{2}$
Human Hæmatozoon,	$\frac{1}{3500}$ in.	$\frac{1}{75}$ in.	1 to 46	Rounded	Acutely Pointed	1 : 8

* 1. Embryo in “chylous urine” immediately after addition of acetic acid; 2. tail of embryo after prolonged action of acid, seen through the delicate tube (LEWIS).

Hæmatozoa of nematoid development, but not always of the same species, have been found also in the dog, wolf, horse, ass, sheep, and in various wild animals, and various names have been given to them. In the wolf, Bohe-Moreau discovered them about twenty-seven years ago, and these were identified as similar to the *Spiroptera sanguinolenta* of Rudolphi. One hæmatozoon from the dog is the *Filaria immitis*, and another, according to Dr. Cobbold, is the *Filaria papilosa hæmatica* (Dr. T. S. COBBOLD, *Lancet*, p. 463, 1873. See also some valuable contributions to this subject by PATRICK MANSON, M.D., in *Medical Times and Gazette* for 1877.)

The subject is one of great practical importance for the inhabitants of tropical and sub-tropical countries. This has been pointed out in an interesting paper by Sir Joseph Fayrer, read before the Pathological Society in February, 1879. Ever since Dr. Lewis discovered this parasite evidence has been accumulating to show that this hæmatozoon is more or less intimately connected with several severe and intractable, and, unfortunately, by no means rare diseases in those regions. It has been shown that disorders of the lymphatic system, especially in the tropics, are found frequently associated with, if not caused by, filaria. Nævoid and ordinary elephantiasis Arabum, chyluria, hæmaturia, lymph varix and abscess, hydrocele, affections of the cord and testis, diarrhœa, fever, cachexia, deterioration of general health, certain skin diseases, deafness, eye disease, have been ascribed to the existence of such filaria, although it is not contended that they are always so caused. A powerful impulse has also been given towards the further investigation of the relation of the *Filaria sanguinis hominis* to disease, by the discoveries of Dr. Manson in China, and of Dr. Bancroft in Australia. Dr. Manson has found that the mosquito is an active agent in the propagation of the filaria. The embryos are taken into the mosquito's stomach with the blood of persons infected by the hæmatozoon, the further development of which shortly begins in the stomach of the mosquito. Thence they are transferred to water, whence it is assumed that it again finds entrance into the body of man. The mature filaria has been found by Dr. Bancroft in a person suffering from lymphatic abscess of the arm, in 1876; and Dr. Lewis found two specimens in blood-clot from a young Bengalli lad who had been operated on for nævoid elephantiasis, in 1877. Specimens were sent by Dr. Bancroft to Dr. Cobbold, who gave an account of their anatomy in October, 1877. The following are the dimensions given of the mature female:—length, $3\frac{1}{2}$ inches; breadth, 1 line; embryo, $\frac{1}{125}$ to $\frac{1}{200}$ of an inch; breadth, $\frac{1}{3000}$ to $\frac{1}{25000}$ of a line (*Brit. Med. Journ.*, 22nd Feb., 1879).

Treatment of those infested by Round Worms.

The habitat of the *Ascarides* being for the most part a collection of mucus, the means used for their expulsion are such as may expel mucus. Four grains of *compound scammony powder*, with five grains of *aromatic powder*; or, two to three grains of *calomel* and ten grains of *jalap*, taken at bedtime, are useful. In weakly children, small doses of Epsom salts, with *quinine* in a bitter infusion, such as *gentian*, will ultimately effect the same

object, and with less distress to the patient. Many persons place great confidence in *calomel* as a medicine capable of destroying round worms; but it does not appear to act beneficially except as a purgative in expelling mucus. The day after the administration of the purgative, the patient ought to be kept on low diet, without solid food, only a little beef tea being taken; and on the second day—the day succeeding the purgative—from five to ten grains of the *etherial extract of santonin* may be given during the day; or from three to four grains of *santonin* itself.* About three doses are sufficient; one every second night, followed by a brisk cathartic the morning after each dose. It may be compounded as *troches*, containing one or two grains in each; or two to six grains may be dissolved in one ounce of castor-oil, and a teaspoonful given every hour till the oil operates. A *santonate of soda* is also recommended by Kuchenmeister in doses of two to four grains. *Santonin* seems to be a specific for the destruction of *Ascaris lubricoides*; but it may be necessary to state to the patient or his friends, that the sight sometimes becomes perverted as to colour after a few doses, and colourless objects may be seen to be blue or yellow.

The *Oxyurides*, or small vermicular *Ascaris*, being situated so near the rectum, enemata have at all times been much used to expel or destroy them; and injections of oil have been much commended, especially of castor-oil, olive oil, or sweet oil. But these animals will live from thirty-six to forty-eight hours in castor-oil. Indeed, very little benefit has been derived from any such local treatment. Warm water injections tranquillise the intestine, and give more temporary relief than anything else. The *Oxyurides* are killed by cold; and injections of cold water, with a little vinegar, are very efficacious. If the child is a vigorous child, large injections of very cold water may be administered, with vinegar or a few drops of *ether* or of *alcohol*. In obstinate cases, a weak solution of *corrosive sublimate*, in the proportion of one quarter of a grain to two ounces of enema. Injections of the following bitter substances have been found very useful in the treatment of the *Ascaris vermicularis*:—Three or four ounces of a strong *infusion of quassia* repeated three or four times, or of *steel*, and *quassia*, and *aloes*, or a solution of *common salt* in gruel, or a similar quantity of *lime-water*, has been found of service. At the same time it is well to administer internally some bitter medicines; for example, half an ounce (or any dose suitable to the age and strength of the child) of *compound decoction of aloes*, taken in the morning fasting, once or twice a week; and three ounces (or other suitable dose) of *infusion of quassia* may be taken every morning that the *aloes* is not taken.

Chloride of sodium, to the extent of an ounce in a pint of *quassia infusion*, has also been found a useful injection; so also has an enema composed of *aloes*, *carbonate of potash*, and *mucilage of starch*. But whatever local remedies are used, it is necessary to attend to the general health, which usually is at fault, and to persevere in the use of enemata

* *Santonin* is a crystalline neutral principle obtained from the *Artemisia santonica*, or *Semen contra*,—which is not a seed, but is the unexpanded flower-head of a species of *Artemisia* imported from Russia, and is the only so-called worm-seed which yields *Santonin* in quantity worth extracting. It is a tasteless and pleasant vermifuge for children (SQUIRE).

twice a week for several months. The digestion is generally slow and imperfect, the secretions from the mucous membrane of the alimentary canal being abnormal. For this condition, small doses of the extract of *nux vomica*, with *sulphate of iron*, in extract of *gentian* or *aloes*, or in *rhubarb* or *colocynth* pill mass, taken twice a day, will be found of great service.

From what has been already written, it will be seen how important it is, in the treatment of all parasitic diseases, to take every means of *utterly destroying, by burning or by chemically destructive agents, all débris or excreta which may be passed by patients suffering from these parasites*; and also how necessary it is *to look to the purity of all water supply used either for the purposes of food, drinking, or bathing; to the quality of pork or bacon, especially in connection with the Trichina spiralis*; and in the use of flour that it be free from contamination by the excreta of cats and mice, which might so introduce the eggs or larvæ of *Ascaris* or *Oxyuris* (STEIN).

CLASS B.—STERELMINTHA, OR SOLID WORMS.

Of the solid worms, twenty-one forms have been found in man, some of them mature parasites, but others immature. Of these eight are tape-worms with their immature hydatids. Two are *Bothriocephali*, and others are forms of *Distomata*.

***Bothriocephalus latus*.**—Although classed with tape-worms, the *Bothriocephali* differ essentially from *Tænia*. Two species have been found in man, namely,—(1.) *Bothriocephalus latus*; and, (2.) *Bothriocephalus cordatus*.

The *Bothriocephalus latus* is endemic chiefly in the north of Europe; and is found more especially in Russia, Sweden, Norway, Lapland, Finland, Poland, and Switzerland. The inhabitants of the French provinces adjoining Switzerland are infested with both species. Instances of *Bothriocephalus latus* are said to have occurred both in England and France; but, when carefully inquired into as to their history, it will be found that this parasite (almost as much as the Guinea-worm) maintains a very fixed geographical distribution. Of the six specimens in the College of Surgeons of England, one is from a native of Switzerland; one from a Russian, belonging to the Russian embassy in London; one from a person who had been travelling in Switzerland; a fourth happened in the practice of Sir William Gull, in the person of a little girl from Woolwich, where there is always a number of foreign ships and sailors, bringing with them native food and water; another was passed by a native of Russia, who, after a long residence in England, paid a temporary visit to his birthplace, and returned to England with this parasite as a pleasant memento of his native country. The liability to this form of parasitic disease appears to be greatest towards the sea-coasts and along river districts. Huss, of Sweden, describes it as extremely common on part of the Lapland frontiers, in Finland, and on the shores of the Gulf of Bothnia. On the extreme coast there is scarcely a family altogether

free from it—old and young, rich and poor, native and immigrants, alike suffer from this worm; and in one or two large towns on the mouths of rivers, at least two per cent. of the population experience its attacks. On passing inland its frequency diminishes, until, eight or ten leagues from the coast, rivers, or lakes, it almost ceases to be found. Dr. Huss attributes it to the use of salmon, (*Brit. and For. Med. Review*, l. c.)



Fig. 27.

The head of the *Bothriocephalus latus* is peculiar, and very different from that of the *T. solium*. It is of an elongated form (Fig. 27), compressed, with an anterior obtuse prominence, into which the mouth opens; an opaque tract extending from the mouth separates two lateral transparent parts, which are supposed to be depressions. There are no traces of joints till about three inches from the head; and throughout the entire body the segments have more length than breadth. The whole length of the mature parasite varies from six to twenty feet.

It is of a greyish-white or yellow colour; and the ova are very brown, giving the mature segments a very marked appearance. The neck is not always obvious, for the worm has the power of making it long and thin, or thick and short; and there are no joints or segments to be seen in it, but merely prominent ridges. The segments, when they



Fig. 28.



Fig. 29.

become first apparent, are nearly square; but afterwards they become much wider than they are long. There are two orifices on one of the flat surfaces of each segment; the anterior orifice is connected with a male organ of generation, the posterior is connected with the female. The proglottides are never passed singly, but always in chains of many links, and particularly in February, March, October, and November. The ova (Fig. 29) are always discoverable in the fæces, of an ovid form, with a perfectly translucent operculated capsule, through which the segmented yolk (Fig. 28) is distinctly visible; and at the period of discharge of the proglottides the ova show merely the stage of segmentation of the yolk. The *six-hooked* embryo, cased in a

mantle studded with vibratory cilia, develops itself after segmentation, protected by the capsule in fresh water, for several months after the expulsion of the proglottides. When so far matured, the lid of the capsule opens up, and the ciliated embryo escapes (Fig. 28), and becomes globular in shape, and moves actively about for a considerable period (a week). If during this period they do not succeed in obtaining access to the intestine of an animal adapted for their development, they lose the ciliated mantle, and perish. When these embryos are introduced by experiment into the intestines of mammals, the *scolices* and mature *Bothriocephali* were found. Experiments in which living embryos were introduced by implantation between the brain and dura mater, and into the eyes of dogs, also under the skin of frogs, and by injection into the blood-vessels of mammals, give a negative result; *quoad* the development into *cysticerci* or *scolices*. So, also, feeding experiments with the *scolices* of the *Bothriocephalus* found in various fish lead to negative results; just as the feeding of fish with the eggs themselves. It is therefore justifiable to assume that drinking water from lakes and rivers is the medium

through which the living embryos of the *Bothriocephalus latus* find their way into the intestines of men and of mammals, (Dr. J. KNOCH, *Petersburger Medicinische Zeitschrift*, 1861; COBBOLD, l. c.)

Bothriocephalus cordatus.—This species (Fig. 30, *a*) is comparatively new to science, and has recently been described by Leuckart, who received about twenty specimens from Godhaven, in North Greenland, one of which was from the human intestine. The parasite measures about a foot in length, and exists in dogs in considerable abundance. It differs from *Bothriocephalus latus* in the form of the head, which is heart-shaped (Fig. 30, *b* and *b'*), or obcordate, short, and broad, and set on to the body without the intervention of a long neck. The segments are distinct from the very commencement, near the head; and so rapidly do they increase in width, that the anterior end of the body becomes lancet-shaped. About fifty joints are immature; and in the largest example Leuckart counted a total of 660 joints. It displays a greater number of the calcareous corpuscles, and a greater number of lateral uterine processes (LEUCKART; COBBOLD, "Remarks on the Human Entozoa," in *Proceedings of Zoological Society*, Nov., 1862.)

Tænia solium.—In their mature condition the tape-worms are all more or less jointed entozoa, of a riband-like form, marked with bands, or girdled. Each mature joint or segment is of hermaphrodite conformation, containing at once male and female reproductive organs, which produce fecundated ova. In their immature condition the embryo penetrates the tissues, and becomes encysted. In this stage of development they are known as the "cystic entozoa," because they terminate in a bag or cyst.

Eight varieties of true tape-worms have been found in man, but only two of them are of frequent occurrence—namely, the *T. solium* and the *T. mediocanellata*. The former is the one endemic in this country; the latter is the more common tape-worm on the Continent, in South Africa, and India.

These tape-worms have been known for a very long period; but they have not always been distinguished from each other. Indeed, the distinguishing characters are but recently known. They have often been confounded together under the name of "solitary worm," because it was believed they lived singly. This, however, is a mistake.

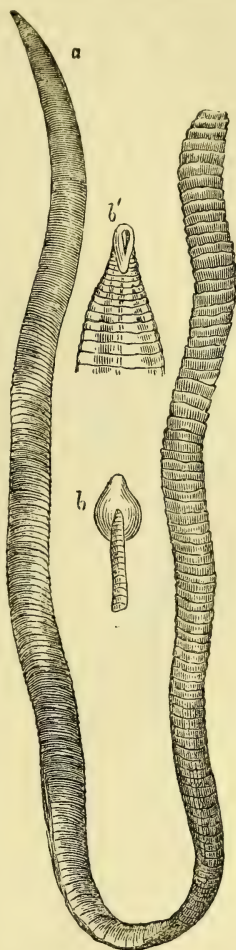


Fig. 30.*

* *a.* *Bothriocephalus cordatus*, natural size; *b.* Head, back view, magnified five diameters; *b'.* Upper part of body and head, magnified two diameters.

The *T. solium* and *T. mediocanellata* appear at first sight to be very similar to each other in general appearance. The latter is much the larger of the two, and Surgeon-Major Welsh (for five years my valued helpmate at the Army Medical School), in Vol. XV. of *Quarterly Journal of Microscopical Science*, in a very exhaustive account of the anatomy of the *T. mediocanellata*, has pointed out very clearly the distinctions between these two forms of tape-worms.

It is only in the alimentary canal—the small intestine—that *Tænia* become sexually mature, in natives and native animals of all countries. They are very common in natives of Abyssinia—so common that its absence is the exception to the rule. Their presence is there looked upon as a natural occurrence; and so general is this belief, that when a slave is sold into Abyssinia he provides himself with a plentiful supply of kousso—the local remedy for expelling the parasite.

The *Tænia solium* is a common tape-worm of this country. It is composed of segments of variable size, numbering from 800 to 1,000; and these being endowed with considerable contractile power, the length of tape-worms varies greatly, and so also does the width and thickness. Nine to thirty-five feet may be quoted as average measurements of length, and there are grounds for believing that a length exceeding thirty feet may be attained by a *T. solium* in the human intestine in about three or four months. The body narrows from the posterior to the anterior extremity, till towards the head it becomes extremely attenuated. The parenchyma is soft and white, with microscopic calcareous particles, which have been mistaken for ova (because they are round or oval), scattered through nearly every part.

The HEAD (Fig. 31) is very small, but it may be seen with the naked eye to be of a globose or triangular form, and generally with black pigment ingrained into its substance. On the most anterior part of the head, with the aid of a lens magnifying twenty-three or twenty-five diameters, four circular projections, equidistant from each other, may be seen. Each has a circular disc or cup, surrounded by a rim of dense tissue. The parasite is

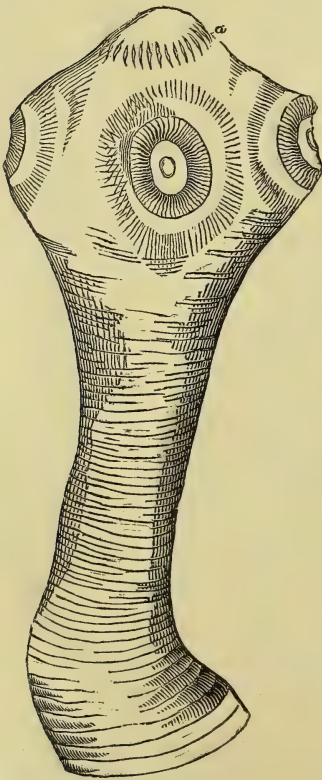


Fig. 31.*

able to elongate and retract these projections; so that, while opposite ones are put forward, the two others are kept back. Between the suckers, and anterior to them, is a convex protuberance or rudimentary proboscis,

* Head and neck of *Tænia solium*.—a. Circle of hooks.

which is impervious, and surrounded by a double row of hooks (Figs. 30*a*, and 31). These are siliceous, and number twelve to fifteen in each row. Their shape is peculiar, consisting of a straight stem or handle, a middle nob, and a distinct hook or claw, surrounded by a sheath or sac. Bremser believes that a *Tenia* loses these as it gets old; or it may shed them periodically by rows; and, being lost, may not be renewed, that so the parasite may be got rid of in the course of nature. The head terminates a long and slender neck, on which there are transverse markings, but no visible joints or articulations. Such joints distinguish the body; and these joints, segments, or zoönites are united end to end in a single linear series. The characters of these segments vary at different parts of the body. They are square or oblong; and in the mature part of the animal the length of them is equal to twice the width. The anterior border of each segment unites with the anterior or previous segment, and is thinner than the posterior border, and also narrower. The posterior border is thick, and projects or overlaps the border of the segment next in order, and is undulating or indented. The lateral margins incline to each other anteriorly. The two surfaces are flat or slightly elevated towards the centre. Each mature segment contains male and female organs of generation. The opening at the side of each is the sexual aperture, indicated by a prominent papilla. These openings are sometimes at one edge, and sometimes at the opposite edge. Two, three, or four consecutive segments may have them on the same edges, or on opposite; but there is no regular alteration. With a lens a cup-shaped depression may be seen, showing two mesial apertures. From one of these a *lemniscus* or rudimental penis projects, connected with a horizontal (deferent) canal (sometimes indicated by a dark

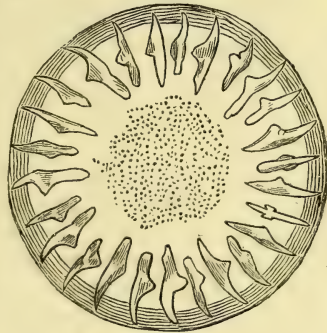


Fig. 32.*

pigmentary material) from a vesicular body in the middle of the posterior end of the segment (OWEN). Behind this male orifice is the opening to the female organs, by a canal leading to a lobulated organ, which is the ovary or germ-stock. These parts are more distinctly developed the farther the segments examined are from the head end of the worm. While the head continues to adhere, by its circles of hooklets and oscula, to the mucous membrane of the intestine, the last or caudal joints, when they have arrived at sexual maturity, are separated one by one, or in numbers together, and new joints are at the same time gradually formed behind the head. The total number of joints in a *tenia*, ten feet long, has been found to number upwards of 800; and the joints appear to be sexually mature about the four hundred and fiftieth segment from the head. Thus growth and development take place mainly towards the neck of the parasite, by a process of transverse fission; and thus a segmented individual or compound animal appears to grow. This segmentation of

* Circle of hooks more highly magnified (after LEUCKART).

individual links by transverse fission ceases when the organs of generation begin to develop in them; and when those are complete, the segment

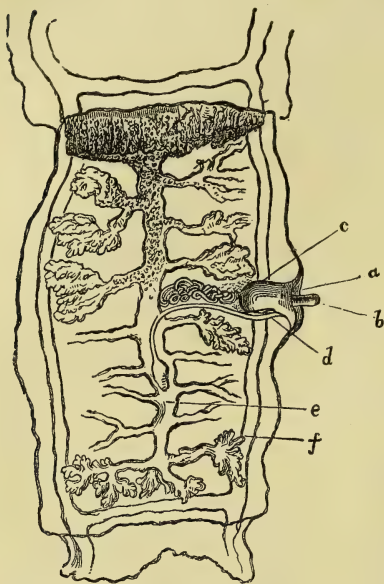


Fig. 33.*

or link having arrived at sexual maturity or completeness, is called a proglottis. Thus all the new segments come to be developed between the head and those which are advancing to sexual completeness; and if the characters of complete sexual development be taken as the distinctive mark of individuality, then each segment of a tape-worm may be looked upon as a distinct animal; and this separation by fission or segmentation may be considered as analogous to what takes place in the medusæ or polypes, in which the segments, zoönites, or proglottides may be regarded as making up a colony of animals. It is only in the alimentary canal of man and other animals that the tape-worms, or cestoid entozoa, attain to sexual maturity; and in all of them the ova are fecundated before being discharged, and may often, as in the *T. solium*, be perceived to have

undergone the first stage of their development before they are excluded from the oviduct of the mature segment. The expulsion of the ova occurs in some one of the following ways:—

(1.) The impregnated segments separate from each other, and passing out of the body singly or in numbers with the fæces, or without any fæcal evacuation, become decomposed, and so the eggs are set free. The activity of these separate segments is retained for a considerable time

after passing out of the body—a circumstance which led to their being at one time taken for a distinct species of worm, to which the name of *Vermes cucurbitini* (from resemblance to a pumpkin seed) was applied. The contracted appearances of a segment during its movements out of the body, are represented by the forms shown in the accompanying wood-cut (Fig. 34).



Fig. 34.†

* Proglottis of *Tania solium* magnified.—a. Genital pore, with its preputial cover or sheath-skin; b. Lemniscus or penis; c. The oviduct; d. The seed-vessel; e. The uterus; f. The water vascular system of vessels (after ROKITANSKY).

† Proglottides of a *Tania (mediocanellata)* in various stages of contraction, thus showing active movements (after LEUCKART).

One may readily observe the activity displayed by these impregnated segments as they disport themselves on the recently extruded excrement of almost every constipated dog. The expelled joints may be seen to become violently contracted shortly after their expulsion, as if the stimulus of physical climate in their new situation provoked excessive contortions. The long single joints thus expelled become still more elongated by contractions of their transverse fibres, while the alternate contractions of these fibres with the longitudinal ones cause shortening of the joint to such an extent that its breadth exceeds its length. Such a sequence of contractions produces movements which simulate those of progression in a worm; and thus these segments may be seen to move some little distance from the spot on which they may have first fallen, discharging ova during their march from the interior of the segment. Thus they may move about for a time; but the growth of the myriads of embryos in the interior of the *proglottis* causes it sooner or later to burst, when the embryos become scattered over grass or ground, dispersed in drains, sewers, ditches, surface water, or waste places, while wind and insects help to diffuse them still farther. (2.) Eggs are also discharged through the genital pores of the mature segments; and if the segment be slightly squeezed, the ova may be pressed out. (3.) The mature joints of the adult tape-worm seem, in some instances, to undergo a disintegration within the intestine of the animal they live in. Thus, Kuchenmeister on one occasion found the wall of the large intestine of a dog occupied by a white sandy powder, the particles of which, on examination under the microscope, turned out to be innumerable ova of a *T. serrata*, which lived higher up the bowel, accompanied by its separated joints. Such contingencies, as those mentioned above under (2) and (3), are not unlikely to happen within the rectum, when, by constipation or otherwise, the matured joints are retained, giving rise to the most serious dangers which the matured tape-worm inflicts on the animal it inhabits, and one of the strongest indications for its speedy removal. It has been recently ascertained that in one or two instances the presence of a *Cysticercus telæ celluloseæ* (the embryo of the *T. solium*) has been found to co-exist with the previous prolonged existence of a *T. solium* in the intestinal canal of the human subject.

The mature segments are often expelled from the human rectum at the rate of six or eight a day, sometimes with the excreta, and sometimes *per se*; and they exhibit evidence of very active vitality for some time. Moisture is favourable for maintaining their existence, and for promoting the spread of the eggs over herbs, grass, ground-fruit, or vegetables, which may become the food of man or of cattle.

The structure of the ova (Fig. 35) is peculiar; and the provisions possessed by their coverings for preserving the embryo are important points for consideration in connection with their transmission through apparently impossible conditions into the bodies of animals, where they become further developed; and in connection with their powers of resistance to therapeutic agents (which have been called anthelmintics or vermifuges) administered for their removal.

It is only in their earlier stages of development that they are

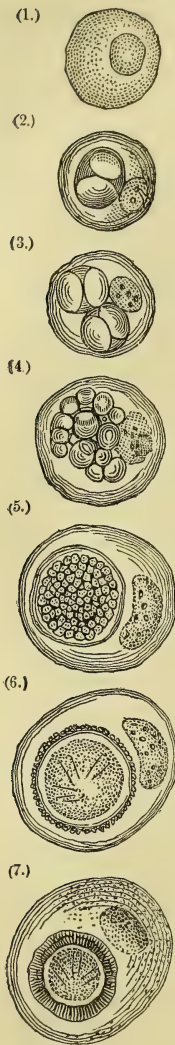


Fig. 35.*

really the analogues of ordinary ova. In the blind extremities of the oviducts of the mature joint of the tape-worm, the shells of the ova appear to be composed of a calcareous transparent substance; and by the time the ova reach the central segments of the tube, their hitherto transparent calcareous shell becomes not only much thickened, but is converted into a dark-yellow or brown mass, in the interior of which the embryo is formed. The egg at first is of the simplest structure, and very minute, about $\frac{1}{700}$ part of an inch in size. The admixture of these organic elements with the calcareous shell imparts to it that extraordinary power of resistance to chemical, and even mechanical, violence, which it certainly possesses. Dilute acids and alkalies have little immediate effect on this leathery husk; and even after hours of immersion in them, scarcely more than a slight swelling and transparency are produced upon the shell. It is, therefore, no matter of surprise that after months of exposure to warmth and moisture, or to cold and dry air, the pulpy, putrid, or dried-up mature segments of *tape-worms* should yield ova which show no sign of degeneration or decay. A more or less speedy death of the expelled segments is followed by their putrefaction, hastened, it may be, by warmth and moisture. The eggs in their interior are then set free, to be carried by winds, waters, or other agents, wherever accident may determine. Thus they may lie to rot upon the soil, or they may be consumed as food by various animals which feed on such minute particles of food; while the minority of the eggs, after many and long wanderings of this passive nature, may at length be engulped unconsciously by some unfortunate animal, or by man feeding on vegetable food, or taken by drinking water.

Within the alimentary canal of the animal which is thus so unfortunate as to eat the egg, a small embryo of most simple form is set free from the ovum by the rupture of the calcareous husk which incloses it. Such rupture is absolutely necessary to liberate the embryo, and may be effected by mechanical violence, such as friction, or crushing by the teeth in mastication of the food, rather than by solution or digestion in the stomach. Animal heat does not seem to be alone sufficient, nor is mere moisture sufficient, to liberate the embryo; which when set free consists of little more than a highly contractile vesicle about the same size as the yolk of the ovum, measuring only about the $\frac{1}{1250}$ of an inch.

* Development of the ovum of *Tænia solium*—(1.) Previous to segmentation; (2, 3, 4, 5.) Segmentation in the impregnated ovum; (6.) Appearance of the early embryo, with its three pairs of siliceous spikelets; (7.) Mature condition of the ovum containing the embryo inclosed within its leathery case (after LEUCKART).

It is peculiarly armed for progression through the most delicate tissues. On one side of it are placed three pairs of spikelets; one pair points forwards, and the two other pairs are so placed that a pair is towards opposite sides of the embryo, or at right angles to the anterior pair. These spikelets are shown in the last two drawings of Fig. 35. The embryo when free is named a "*proscölex*," and commences life on its own account by efforts at active migration. It makes a passage through membranes, walls of vessels, and textures of solid viscera, by the vigorous exercise of the spikelets, so as to reach localities where it becomes encysted, to pass another phase of existence. The first portion of its path is pierced by bringing close together the several pairs of spikelets so as to form a kind of wedge-shaped stiletto. The lateral pairs of these spikelets are then brought backwards to a rectangular position, and so they thrust the embryo forwards in the direction in which the anterior pair of spikelets pointed. Similarly repeated actions eventually accomplish progression to a resting-place; and the action may be aptly compared to the movements of the arms and attitude of the head of a swimmer. But this active migration is not the sole means by which the embryo *Tænia* is enabled to traverse the animal body. The embryo may penetrate a mesenteric vein, when it will at once be swept onwards by the current of the blood to the portal vein, and passing into the minute ramifications of the portal system, may find a resting-place in the liver. Leuckart has found the embryos of tape-worms in the blood in such large numbers, that he inclines to regard the currents of the blood in the vessels as the ordinary and more usual channels for the migration of the embryos. It also explains the wide diffusion of *tape-worm* embryos as *cysticerci* or *echinococci* in various stages of development throughout different viscera of the body, where they become encysted, and especially their very frequent site in the liver, peritoneum, and mesentery. Thus far completed and encysted, the embryo is called a "*scolex*," and as such in *Cysticercus telæ cellulose* was first described by Rainey. The embryos of *Echinococci* and *Cænuri* give rise to numerous *scolices*, which complete their development into tape-worms in the alimentary canal of another animal, when that animal happens to eat the liver or brain containing the cysts of such *Echinococci* or *Cænuri*; but the embryos of such *Echinococci* or *Cænuri* tape-worms find their way into man or animals with drinking-water, or with raw, uncooked articles of vegetable diet from moist soils, such as salads, roots, fallen fruit, all of which may be doubtless so exposed as to receive the germs or ova containing the embryos, passed along with faecal excrement of dogs, pigs, and the like, and which, after being dried, are carried by wind or water in all directions.

The third stage of development consists in the formation of segments, which are first seen in the form of marks, like girdles, surrounding that portion of the entozoön next to its oscula and hooklets, terminating in a caudal vesicle. It is now an incomplete segmented *Tænia*, in scientific nomenclature called a *Strobila*; and the development to this stage may occur while the entozoön is still within the closed cyst which has formed round it, as may sometimes be seen in the livers of mice.

It is only in the alimentary canal of animals that the last and perfect stage of development is attained, by the tape-worm reaching sexual

maturity. The segments or links marked off by the bands, joints, or girdles, in the encysted *Strobila embryo*, become mature segments by the development of sexual organs within them. This only takes place after the *Strobila embryo* has passed into the alimentary canal of an animal which can afford it a place to live and spend the rest of its days as a fixture attached by its hooks to the mucous membrane. The human alimentary canal is an oft-chosen place of the *T. solium* and *T. mediocanellata*. Here these tape-worms form complete sexual segments or links, each being hermaphrodite, and tending to separate when completely mature. After living for some time in this prolific condition, and having produced often a very large number of joints and an enormous quantity of ova, the existence of this troublesome parasite is terminated by the separation of the animal from its attachment to the intestinal membrane. When this separation occurs spontaneously, it may be that the circle of hooklets being shed periodically, or being lost, they are not renewed, and so the prolonged life and romantic vicissitudes of a tape-worm may be brought to a natural termination. The whole length of the beast is then ignominiously expelled, while some reputed vermifuge, however innocent, may get the credit of its death. The apparent success of many such *parasiticides* is recorded and measured by yards of tape-worms, which, being ingeniously bottled by worm-doctors and charlatans, are duly advertised to have been

passed by John Smith or Sarah Brown, after they had been dosed with the "infallible" remedy.

***Tania mediocanellata*.**—This is a hookless, flat-headed tape-worm, the *cysticerci* and embryos of which are developed in the muscles and internal organs of cattle (LEUCKART); and man becomes infested with this tape-worm by eating imperfectly-cooked veal and beef in which the *cysticerci* abound. It was first discovered by Kuchenmeister in 1855, and shown by him to be different from the *T. solium*. Its head (Fig. 36) is large, obtuse, and truncated, and carries no hooks. Its sucking discs are much larger than those of the *T. solium*, as if to compensate for the want of the hooks. Its segments, when mature, separate easily. The ovaries are simple, giving

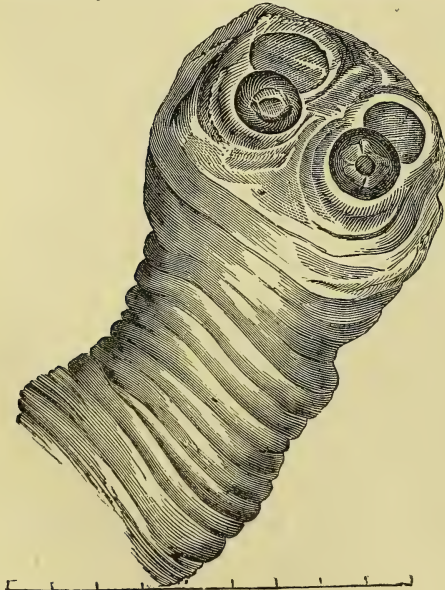


Fig. 36.*— $\frac{1}{100}$ of an inch \times 23 diameters.

* Head of the *Tania mediocanellata*, drawn from the camera lucida, by B. J. Jazdowski (then Assistant-Surgeon, A.M.D.), from one of three specimens, all of which were removed from the small intestines of a soldier who died at Fort Pitt in 1860, and who had been for many years a cook at the Cape of Good Hope. The specimens are in the Museum of the Army Medical School at Netley.

off sixty lateral parallel branches. The eggs are similar to those already described.

The *T. mediocanellata* has been found in several instances of invalid soldiers who died at Fort Pitt, and at the Royal Victoria Hospital at Netley. In one man three very large and long worms existed in the small intestine, each of them precisely similar in all respects. The soldier in whose intestine they were found died of *diabetes mellitus*, and he had been a cook for many years to a military mess at the Cape of Good Hope; another occurred in a soldier who had been also a long time at the Cape.

Tænia acanthotriax, the rostellum of which is furnished with three rows of hooks, fourteen in each row, like the *T. marginata*, is only known in the human muscles, as an embryo or *cysticercus*.

Tænia flavopunctata measures about eight to twelve inches long. The proglottides are short, and there is a yellowish spot, clearly visible to the naked eye, situated about the middle of each joint, which reminds one of the colour and situation of the genital organs in the *Bothriocephalus*. The reproductive orifices occur all along one side of the worm, and the eggs are unusually large. Only one instance of the occurrence of this parasite is on record, in 1842, from an infant nineteen months old. They were expelled without medicine, their presence not having been suspected (WEINLAND, COBBOLD).

Tænia nana, when fully grown, attains a length of eight or ten lines, and carries from 150 to 170 joints. Its hooks are essentially the same in form as those of other *Tænia*, only they are very minute, and have a peculiar form, owing to the close approximation of the claw and of the anterior root-process (LEUCKART), which gives them a "bifid" appearance. Its head is comparatively large and obtuse, with a long neck. It was first described by Bilharz in 1851, having been found in Egypt in the intestine of a young man.

The *Tænia elliptica*, whose *cysticercus*, or embryotic condition, is not yet known, is common to cats and dogs, and sometimes infests man (ESCHRICHT, LEUCKART). Weinland believes that the *cysticercus* will be found in flies, and that dogs obtain the larvæ by snapping at dipterous insects.

The tape-worms just noticed are all mature parasites; and, for that reason, they have been considered together. The following are—

The Immature Tape-Worms, Non-sexual, Cystic, or Vesicular Parasites.

These entozoa are variously spoken of by the older, and even by many recent writers, under the vague terms of *hydatids*, *cysts*, and *acephalocysts*. They inhabit the closed cavities of animals, or they are inclosed in cysts in the more solid parenchyma of organs. They are represented by the *scolices*, or second stage of the *tape-worm* embryo, and consist of a *Tænia*

head, provided or not with a similar circle of hooklets, according as the mature parasite is so provided; or there is an absence of hooklets, according to the variety of the *Tænia*. Four oscula are *always* present, and the head is united by a neck to a vesicular body of variable size. These are now known to be, as already described, varied forms of *Tæniæ embryos*, of which the following infest various parts of the human body, or of animals used by man as food, or of domestic and wild animals not so used:—

The *Cysticercus* of the *Tænia solium* (synonym, *Cysticercus telæ cellulose*) is the *larva* or *scolex* of the *Tænia solium*, and as seen in man, the pig, the ox, horse, calf, camel, sheep, and roe deer, it consists of a vesicle—conical, glistening, and white—containing fluid; to this a head is attached by a narrow pedicle or neck, which is transversely lined—the lines approaching to rugæ towards the vesicle. Its size varies in solid viscera from that of a small pea to a large marble; but in free cavities, such as in the ventricles of the brain, it attains a larger size. The smallest “measle” (*scolex*) measured has been $\frac{1}{25}$ of an inch in diameter, from the brain, liver, and intermuscular septa of a pig, thirty days after feeding with *proglottides*; and the complete development is usually accomplished within ten weeks, when the cyst measures $\frac{1}{4}$ to $\frac{1}{3}$ of an inch, or from the size of a pea to that of a kidney-bean. The cyst is more dense at the side or edge where the head and neck are growing, than at the great vesicular part, which is the vesicular or bladder-like end. The head and neck can be drawn as if into the vesicle, so that the form and appearance of the parasite may thus be very much altered. An external cyst incloses the *cysticercus* when it inhabits a solid viscus, such as the substance of the liver, or amongst the connective tissue of muscle; but in close cavities, such as the eye or the ventricles of the brain, there is no enveloping cyst, and the parasite floats free within the cavity. In these more free conditions it tends to grow more like the form of a tape-worm; and if it happens to be in the eye, it may soon destroy it, by attaching itself, or by fixing its hooks in some of its delicate textures. The cyst which envelops the parasite in solid organs is developed at the expense of the tissue in which the parasite imbeds itself. They have been thus seen in man, as well as in animals, in the heart, liver, choroid plexus, the brain, in the tissue between the sclerotic and the conjunctiva, in the anterior and posterior chambers of the eye (MACKENZIE), and in the retina (GRAEFE). The head resembles that of the *T. solium*, and carries thirty-two hooks in two rows. The neck varies greatly in length. The parasite is especially frequent in domestic swine, and in them it produces the appearance known as “measle” or “measly pork.”

The *Cysticercus* of the *Tænia mediocanellata* is to be found in the muscles and internal organs of cattle. For our knowledge of the larval state of the *T. mediocanellata* we are mainly indebted to Professor Leuckart, of Giessen. He artificially reared them in the flesh of calves, from the eggs of a *T. mediocanellata*; and recent experimental researches incontestably prove that the “measles” of cattle give rise to the *T. mediocanellata* in any man or animal which has fed on such “measle.” He fed two calves with the fresh eggs of the *T. mediocanellata*, by giving them the *proglottides* of this parasite. The first animal he experimented on died

from a violent attack of the measles disease; and on dissection the muscles were found filled with measles, or vesicles containing imperfectly-developed *scolices*. On the second occasion a smaller number of *proglottides* (in all about fifty) were administered, and the febrile symptoms again appeared with such virulence that Leuckart thought this animal would die. Fortunately, after the lapse of a fortnight from the commencement of symptoms, some abatement of the illness took place, which gradually continued until the animal was perfectly restored to health. Eight-and-forty days subsequent to the earliest feeding experiments (which were continued at intervals for eighteen days) Professor Leuckart extirpated the left *cleidomastoid* muscle of the calf, and whilst performing the operation he had the satisfaction of seeing the *cysticercus* vesicles lodged within the muscles. They were larger and more opalescent than those of the *Cysticercus tæniæ cellulosa*, but nevertheless permitted the recognition of the young worms through their semi-transparent coverings. The heads of the contained *cysticerci* exhibited all the distinctive peculiarities presented by the head of the adult *Strobila* (the *T. mediocanellata*). Dr. Cobbold has verified this experiment. Taking the results of such experiments in connection with previously ascertained facts, the most unequivocal evidence is brought together that man becomes infested with the *T. mediocanellata* by eating imperfectly cooked veal or beef in which the *cysticerci* abound.

The *Cysticercus* of the *Tænia marginata* (*Cysticercus tenuicollis*) is rarely found in man, but it has occasionally been found in the mesentery and in the liver. Eschricht and Schleissner have shown that these *cysticerci* are sometimes associated with the *echinococcus* in Iceland (COBBOLD, l. c.) It is only in this immature state that it is known to infest man as a *cysticercus*. The full-grown tape-worm is found in the dog and wolf, and is often confounded with the *T. serrata*, from which it differs in its comparatively bulky size and the peculiar form of its hooks. The *proglottides* nearly equal in size those of the *T. solium*. In its scolex or immature condition this parasite has a very wide distribution. In addition to its occasional presence in man, it has been found in various monkeys, in cattle, sheep, reindeer, and other ruminants; in horses, swine, and squirrels. Its *habitat* is for the most part the peritoneum (ROSE and others). They are *cysticerci* which occasionally attain an enormous size.

The *Echinococcus hominis* is the larva of the *T. echinococcus*; and the first accurate description of the immature form of this parasite was published by Bremser in 1821. These parasites have been, and are still often indifferently named "hydatids" or "echinococcus cysts;" but English writers have restricted the term "*hydatid*" to designate the enveloping cyst, and the term "*echinococcus*" to signify the contained entozoön. The *Echinococcus* is an extremely common parasite of the human body. It has been found in the kidneys, lungs and pleuræ, liver, brain, heart, spleen, ovaries, breasts, tissue of the throat, and the bones; and the characteristic cysts are not unfrequently discharged with the expectoration, or by stool, or by the urine. In the Icelandic endemic disease due to this parasite, there is scarcely a part of the body in which it has not been found. It appears that the people of that country have been for some time suffering to a great extent

under this very remarkable hydatid disease, which mainly affects the liver, peritoneum, and subcutaneous texture. Eschricht, writing to Von Siebold, says,—“The disease has extended itself to such an alarming degree, that about a sixth of the whole population of Iceland are affected with it, and that it is attracting considerable attention at Copenhagen.” It produces a long, protracted illness, terminating with a painful death, and means of cure have not yet been discovered. Von Siebold considers it “probable that this disease arises from the immense number of dogs kept in Iceland, for the purpose of herding sheep and cattle” (SCHLEISSNER, *Medical Topography of Iceland*; ALLEN THOMSON, l. c.; also LEARED, in *Medical Times*, 1863). In some cases only a single “hydatid tumor” is developed in an organ or part; but occasionally two, three, or more tumors may be found. These “hydatid tumors” consist externally of a firm fibrous capsule, or enveloping cyst, of a tint which varies with the organ in which it may be developed. In the liver they are white, or of a yellowish tinge. The capsule adheres intimately to the surrounding tissue, and is abundantly supplied with blood-vessels. Bands of connective tissue may be seen stretching outwards from the capsule, and incorporating it with the tissue in which it is imbedded, and from which it has been developed. Within this capsule, and completely filling it, are—(1.) A gelatinous, translucent grey bladder, composed of numerous concentric hyaline layers, giving a

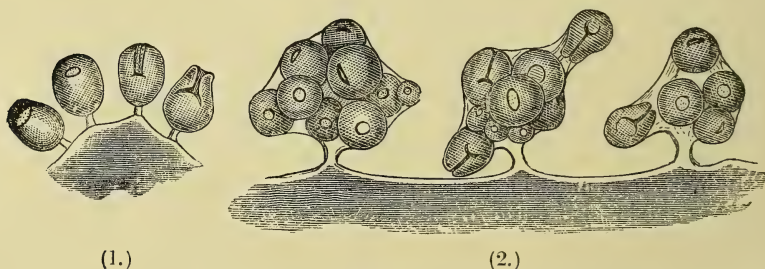


Fig. 37.*

laminated appearance to a section. It is finely granulated in some parts (degeneration?) and highly elastic. (2.) A very thin and delicate membrane is spread over the interior of this elastic hyaline bladder, which is the innermost layer of the “hydatid tumor.” This membrane or layer is the mother sac of the *Echinococcus* embryo (HUXLEY), and corresponds with the germinal membrane of Professor Goodsir. It is studded with innumerable transparent cells, varying in extremes of measurement from $\frac{1}{100,000}$ to $\frac{1}{3000}$ of an inch. It is the seat of the development of innumerable *Echinococci*; and to this membrane, in a fresh hydatid tumor, they are found connected by a delicate membrane, either singly or (more commonly) in clusters, the number of individuals on the cluster varying

* Groups of *Echinococci*, showing—(1.) The pedunculated connection between these parasites and the germinal membrane; (2.) Their occurrence in groups, enveloped by a very delicately thin membrane, continuous with the germinal membrane (after ERASMUS WILSON).

from ten to a hundred or more, as shown in the preceding woodcut (Fig. 37).

On close examination with the naked eye these groups present the appearance of a number of delicate white particles upon the inner surface of the germinal membrane; and when the aggregation into groups consists of many individual embryos, they may be seen through a transparent cyst. They are the *scolices* or *embryos* of the *T. echinococcus* in various stages of development. The "hydatid tumor" is filled and distended with a clear watery fluid, with numerous large and small vesicles, more or less clear and transparent, floating free, or so closely packed together that they cannot be removed without some degree of pressure. Some of them, particularly the smaller, adhere to the germinal membrane. They vary in size from that of a millet seed to a size as large as a goose's egg, and their number not unfrequently amounts to several hundreds (560, PEMBERTON), or even thousands (7,000 and 8,000, ALLEN, quoted by PLOUQUET and FRERICHS). The larger of these free vesicles sometimes contain smaller ones of a third generation, and occasionally they in their turn contain others of a fourth generation. The size of the "hydatid tumor" and the germinal membrane must increase and grow according to the number and size of the daughter-vesicles, and in proportion to the quantity of contained fluid, which is sometimes rendered slightly opaque by the quantity of embryo *Echinococci* floating free in it. From the rotundity and distension of these inclosed vesicles it is difficult to fix them for examination. When they are punctured, their fluid contents issue from the vesicle in a jet of considerable force, impelled by the contractile power of the elastic tissue; and if the incision be of a sufficient size, the vesicle will roll up, and turn itself inside out. The *Echinococcus* embryo varies in size from $\frac{1}{18}$ to $\frac{1}{20}$ of a line in length in the contracted state (Fig. 38, *a*), and from $\frac{1}{10}$ to $\frac{1}{8}$ of a line in its elongated form (Fig. 38, *b*). These variations in length are according as the head of the parasite is extruded or retracted within the vesicle. The contracted state in which the head is retracted within the vesicle is the form most commonly seen in the "hydatid tumor," after removal from the organ in which it was developed. In this state it is usually globular or oval, and slightly flattened at the opposite poles. In the elongated state, when the head is extruded and the hooks appear outside, the parasite is usually larger at the cephalic end, where there may be seen four suckorial prominences and the circle of hooklets. While the number of hooks fluctuates in all the forms of *Echinococci* that have been described, the alleged differences in the size and character of these hooklets have reference to the degree of development of the parasite (LEUKART, COBBOLD), and do not differ sufficiently to give ground for specific distinctions to be made amongst them. These hooks are arranged in a double festoon,

* Two *Echinococci* from a "hydatid tumor." The one (*a*) has the head retracted within the vesicle; the other (*b*) has the head extruded.

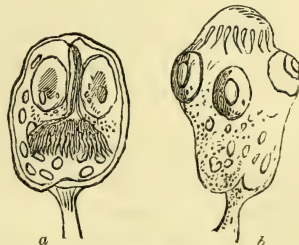


Fig. 38. =

round a membranous disc (Fig. 39, *A*), and vary from twenty-eight, thirty-four, forty-six, or fifty-two. They are arranged in two rows (Fig. 40, *B*), one row containing longer hooks than the other; the longer ones measuring about $\frac{1}{1050}$ of an inch. They each possess a gentle curve, so that there is a concave and a convex border, and a base (Fig. 40, *b*, *c*), which encroaches on the concave border for nearly half its length. The base is broader than any part of the hook, and has a bifid end. These hooklets move on the central bifid process as on a pivot (Fig. 40, *f*, *g*, *k*). Hook sacs may be distinguished with a sufficiently powerful lens. These hooklets are of so minute a size, and at the same time so extremely characteristic and important in diagnosis, that the annexed woodcuts (Figs. 39 and 40, after ERASMUS WILSON) may give an idea of their shape and arrangement. The head of the embryo echinococcus when extruded is separated from its body by a groove, and at its opposite pole is an umbilical depression, which gives insertion to the peduncle which fixed it to the germinal membrane (Fig. 38). As the growth of the "hydatid tumor" advances, the external enveloping capsule gradually loses its uniform thin and smooth character. It becomes rigid, fibrous, or even

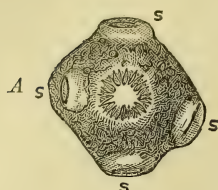


Fig. 39.*

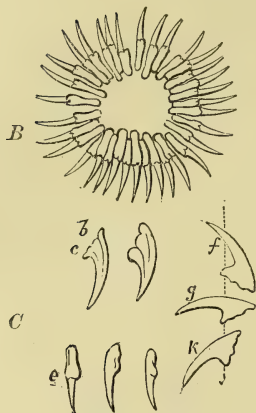


Fig. 40.†

cartilaginous, while its inner surface becomes rough and uneven, covered here and there with laminated deposits. Frerichs, of Berlin, has seen such capsules completely surrounded by a calcareous shell; and the thicker and more rigid the capsule becomes, the greater is the resistance opposed to the further growth of the *Echinococci*, which may even be thus arrested, so that the parasites die, and a spontaneous cure results (CRUVEILHIER, FRERICHS). Compound "hydatid tumors" have been found in which the cavity is multilocular. Outgrowths or buds form which give an alveolar character to the lesion (BUHL, VIRCHOW, FRERICHS); and such occurring in the liver (as in several specimens in the museum at Netley), show on section a most peculiar alveolar appearance, the alveoli varying in size from that of a pin-head to a pea, which are lined by the characteristic germinal membrane and gelatinous hyaline laminae, as demonstrated by microscopic examination.

It has been usual to consider that there are two distinct forms of

* (*A*.) An *Echinococcus* viewed transversely, the head being directed towards the observer; *s*, *s*, suckorial discs. The hooklets are seen to encircle a membranous disc.

† (*B*.) The circle of hooklets seen upon its under surface; thirty-four in number, seventeen long and seventeen short. (*C*.) *b*, *c*, Lateral views of the separate hooklets—*b*, The base; *c*, The central extremity, or bifid process of the base; *e*, Hooklets viewed upon the concave or inferior border; *f*, *g*, *k*, A diagram illustrating the movements and position of the hooklets. The dotted line represents the outer surface of the neck, and runs through the fixed point of the three hooks.

Echinococcus cysts severally referable to different tape-worms; but they are now regarded as one and the same. The cysts so prevalent in Iceland are known to occur indifferently in men and oxen, and are sometimes so prevalent that about one-eighth of all the cases of disease are referable to this cause; and generally several members of one family suffer (LEARED). The *Tænia echinococcus* is very often seen in Iceland. It is a very small one, consisting of not more than three or four segments, and not much longer than a line, carrying twenty-eight to thirty-six hooklets. It is found in large numbers in the intestines of dogs, but from its very minute size it is apt to be overlooked.

Acephalocysts require some notice. They were first described by Laennec as growths of membranous cysts. In the present state of science they are regarded as abortive *Cysticercus* or *Echinococcus* cysts—parasites of one or other of those kinds in which the development has been arrested. Remains of hooklets have been found in them; and the structure of the cyst may sometimes be seen to be precisely similar to that of the *Echinococcus* cyst. They have been found in all those places where the *Cysticerci* and *Echinococcus* cysts are known to abound—*e.g.*, liver, spleen, kidney, bladder, and in exostoses of bones.

Thus we have seen that each kind of *Tænia* has not only its own definite vesicular embryo, but each *Tænia* has a definite *Cysticercus* or *Echinococcus*; and, all of them are capable of being developed or reared into *Tæniæ* when transferred into the alimentary canal of a suitable animal; and, conversely, the development of *Cysticerci* and *Echinococci* occurs in man and other animals in consequence of segments of tape-worms, or the ripe ova they contain, being eaten with their food. The proof of these statements it is the principal scientific merit of Kuchenmeister to have established, by experiments which date so far back as 1851; and they have since been repeated and confirmed by many other observers (SIEBOLD, NELSON, ZENKER, LEUCKART, WEINLAND, KNOCH, and COBBOLD). The experiments of Kuchenmeister were made on a variety of animals, and in one instance on the human body. His observations, together with those of Von Siebold, Dr. Allen Thomson, and others, demonstrate the relation between the *Cystic* and the *Cestoid Entozoa*. The experiments to determine this relation mainly consist in observing the effects of feeding an animal with the ova or larvæ with which it is designed to affect it; and other animals with the cysts developed from these ova. The first experiment of this kind was performed by Kuchenmeister in 1851. He caused young dogs to eat with their food a number of the *Cysticercus pisiformis* of the rabbit and hare, and found that after some weeks these *Cysticerci* were converted, in the intestine of the dogs, into the *T. serrata*. The more important of the experiments devised and performed by Von Siebold confirmed these results. (See previous editions of this work for details.) But, perhaps, the most important and interesting series of experiments were those he made on man. Having the opportunity of repeating on a condemned criminal the experiments which he had previously performed on animals, Kuchenmeister contrived, at seven successive times, to give to a man, condemned to death (between one hundred and thirty hours and twelve hours previous to his execution, mingled with various articles of

food), a number of *Cysticerci* from the hog and some from the rabbit. "They appear to have been partly disguised by their resemblance to the grains of rice in warm rice soup; partly by their likeness to the small bits of paste in a kind of vermicelli soup; and partly foisted on the unhappy wretch by being substituted for the small lumps of fat in blood-puddings" (*Brit. and For. Med.-Chir. Review*, Jan., 1857, p. 119). After death, a number of young *Tæniæ*, in different stages of advancement, were found in the intestine; the greater number of them loose, but a few attached to the mucous membrane. The form of the hooklets, and other circumstances, induced him to regard these tape-worms as the *T. solium*. There were no traces of the *Cysticerci* last swallowed; and Kuchenmeister was of opinion that those only which were first taken, and which were quite fresh, had been converted into *Tæniæ*, and that those taken later, being dead, had been digested with the food. Leuckart made a similar experiment. He fed a man, thirty years of age, with *Cysticerci* from a pig, and in two months the man had *Tæniæ*. But the enthusiastic and sceptical Germans were not yet content with the proof. M. Humbert, of Geneva, experimented on himself. On the 11th December, 1854, he swallowed fourteen fresh *Cysticerci* in presence of MM. Vogt and Moulinié! Early in March of 1855 he felt the presence of *Tæniæ*, and had the satisfaction, delight, or pleasure of discharging segments of them!! Converse experiments have also been made by Kuchenmeister, Van Beneden at Louvain, Eschricht at Copenhagen, and Leuckart at Giessen, each of whom, in separate experiments, caused lambs to take the *Tæniæ* segments with their food; and, in all the cases the same result followed—namely, the occurrence of the symptoms of *sturdy* at a period of from fifteen to eighteen days after the mature segments of *Tæniæ* were given. Twenty-four hours after administration, the blood of the portal and other abdominal veins contained numerous cestoid embryos. On the fourth day, semi-transparent vesicles, $\frac{1}{80}$ of an inch, appeared in the liver, and in six days they measured $\frac{1}{25}$ part of an inch. The same experiment had likewise been performed by Dr. Haubner, of Dresden. All the lambs died of the vertiginous disease; and the *Cœnurus* cysts were found in the brain, heart, lungs, and voluntary muscles. Several of these experimenters, having examined carefully the lambs so affected, were able to detect the progressive stages of formation of the *Cœnurus* in the cortical substance of the brain, where alone these entozoa seemed to attain the true *Cœnurus* form. There were abundant traces of them in the heart, diaphragm, and other muscles, and also under the skin, in some of the experiments; but, in these situations they appeared to be abortive; while in the brain they gradually grew, and in some instances the vesicle had there attained the size of a hazel nut. The brain was in all instances marked with inflamed grooves over its surface, indicating probably the track of the embryos; for at the end of each of these tracks, in the early stages, were found the minute *Cœnuri*.

From the whole series of observations and experiments that have now been made (and of which a summary has been here given), the following general conclusions may be drawn and elementary facts stated, namely:—
 (1.) That entozoa are always introduced into animals from without; (2.)

That some obtain access to the body of animals from water, or other matters in which they have previously lived in the free condition, while others are taken along with animal food in which the entozoa have lived parasitically; (3.) That entozoa, when reaching sexual perfection in their parasitic condition, require to be in a situation which communicates with the external air—the most common position being the alimentary canal, and more rarely the pulmonary cavities; (4.) That almost all the entozoa inhabiting close cavities, or otherwise encysted in the bodies of animals, are imperfect and earlier forms of other entozoa, which may attain maturity in the open cavities of the same or of different animals, or in the free condition, that is, liberated from their cysts; (5.) That entozoa rarely propagate themselves in the same animal in which they have arrived at sexual maturity, but require a different habitation, which they reach by migrations in the various modes before referred to; (6.) That the cystic entozoa are the imperfect states of different *Tæniæ*; (7.) That *Tæniæ* are almost invariably introduced, in their earlier condition, into the bodies of animals with flesh or other animal food, in the condition of “measle;” (8.) That if the ova of *Tæniæ* be introduced into the alimentary canal of a suitable animal, through water, vegetable food, or fruit, their tendency is, after penetrating the tissues, to become encysted, and to assume the form of a cystic entozoön, such as that of a *Cysticercus*, *Cœnurus*, or *Echinococcus*; (9.) That if these cystic entozoa again are taken by certain animals with their animal food, the head part (which corresponds with that of a *Tænia*) resists digestion, and has a tendency to establish itself, and become developed into some form of *Tænia* in the alimentary canal, by the formation of segments after attachment to the mucous membrane.

The prevalence of “measle” cysts in the ration-beef served by the Commissariat throughout the Punjab, in recent years, especially at Peshawur, Meean Meer, and Jullundur, confirms and appears as a corollary to these experiments. It has challenged the attention of the authorities in India. I have had a sample sent me from Meean Meer of this beef, containing as many as 100 cysts in the pound of flesh; and the medical officer who sent it wrote me that he has found as many as 300 in the pound of ration-flesh. The largest percentage of cyst-infected cattle occurred in the Lahore division of the Punjab, in January, 1869—namely, at Meean Meer, where it was as high as 33·15 per cent.; and next in Peshawur, in February, 1869, when it was 17·99 per cent. But all statements regarding the number of slaughtered cattle *rejected as food*, on account of being cyst-infected, are extremely variable, and are totally worthless as to scientific value. No two inspectors are equally careful, and great differences exist as to the practical knowledge and experience of individual inspectors; while the general ignorance on the subject of such parasites and their propagation is almost incredible. The natural size and appearance of these cysts are represented (Fig. 41) by *a* and *b*. The head of one of these is magnified in



Fig. 41.*

* (*a*. and *b*.) Two specimens of *Cysticercus Tænia mediocanellata*, natural size, sent from the Punjab, out of ration-beef.

Fig. 42, to the extent indicated by*the scale attached, and is shown to be the characteristic head of *Tænia mediocanellata* in its embryo state. It may be contrasted with the embryo head of *Cysticercus telæ cellulose* of measily pork (Fig. 43). Up to August, 1868, a great destruction of ration-

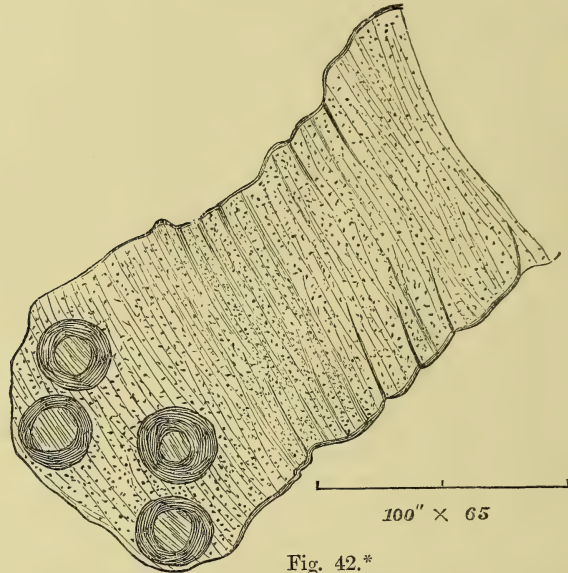


Fig. 42.*

beef had taken place in India, on account of these cysts—so much so that 17,428 pounds of meat had been destroyed in 1868 and 1869. The Government then appointed Assistant-Surgeon Cleghorn to investigate into the whole question. The extensive condemnation of meat, on account of cyst, incurred great expense to the Government,—so great that the Commissary-General feared a difficulty might be experienced in procuring the number of cattle required for rations. The Inspector-General of Hospitals,

Dr. Muir (now Sir William Muir, K.C.B., and Director-General of the Army Medical Department), also expressed his belief that the danger of eating cyst-infected beef had been much exaggerated, and that it might be eaten with safety, provided it was *thoroughly* cooked. The Commander-in-chief in India concurred in this view, and orders were accordingly issued by the Government in the sense of these opinions. Further special inquiry was, however, suggested, but was deferred, till a Report, dated June 1, 1870, by the late Assistant-Surgeon Oliver, R.A., on



Fig. 43.†

* Head of *Cysticercus Tænia mediocanellata*, from ration-beef.

† Head of *Cysticercus telæ cellulose* from measily pork, contrast with the *Tænia mediocanellata*.

the prevalence of *Cysticercus* in ration-beef at Jullundur, was considered by Sir William Muir to confirm his opinion. It was no doubt satisfactory to the authorities, and especially to the Commissary-General (*Abstract of Proceedings of the Sanitary Commissioner with the Government of India*, August, 1870, p. 236). Dr. Oliver reported that the meat was well fed, and of excellent quality; and the objects of his inquiry were,—(1.) As to the sources whence the cattle obtained the *Tænia* ova; (2.) As to whether or not any evil results followed the consumption of cyst-infected meat when properly cooked. Regarding the source of the *Tænia* ova, it was found,—(1.) That the infected cattle had been purchased by native dealers from various parts of the district, not from any particular locality. (2.) That when brought in they were lean, and, on an average, required from two to three months' feeding at the Commissariat cattle-yard before they were fit for the shambles. (3.) That their food consisted of the *grass they could pick up on the grazing grounds of cantonments*, supplemented by such an allowance of grain and *bhusá* as their condition required. (4.) They were supposed to be watered at a trough with water drawn from a well; but, it transpired that they were taken very frequently to a *large dirty tank near the yard for their water*.

There can be no doubt, from Dr. Oliver's inquiries, that both the grazing-grounds of the cantonments and the dirty water of the tank were plentiful sources of tape-worm ova. *The tank was close to the huts of the camel-drivers, many of whom were infected with Tænia mediocanellata. Human filth was often to be seen on the banks of the tank, and microscopic examination of it, and of stagnant water taken from the margin, exhibited the ova.* Dr. Oliver also says, that a few months after means were taken to secure a good supply of well-water for the cattle, *cysticerci* entirely disappeared from amongst them. The cysts were found to be most abundant in the flesh at the root of the tongue, of the rump, of the psoas muscles, and those of the gluteal and lumbar regions; but it is not possible to detect them in cattle for practical purposes before death in any of these situations—the most choice parts for food of man.

Besides the ration-beef cyst, the watery-bag or hydatid cysts largely infested the animals killed at the Commissariat slaughter-house. Of the livers, 70 per cent. were so infected (OLIVER and CLEGHORN). As these cysts or watery-bags are only seen in the internal parts, the attention of medical officers has not been so much directed to them. Hence a prominence has been given to ration-beef with cysts, rather than to the abundant existence of watery-bags or hydatids, which have been in some measure overlooked. The liver and the lungs are the parts generally infected. The larger of the hydatid cysts are found in the liver, but varying from a pea to a man's head in size. They are the inclosing bags of the embryo *echinococcus*—the little tape-worm which infests the dog tribe (see p. 199, *ante*); and considering the immense number of pariah dogs which *are fed on the refuse of animals infected with hydatids*, there can be no doubt about the source of the watery-bag infection of cattle. The extremely minute proglottis within ova of the *Tænia echinococcus* passed by the dogs must be extensively disseminated over the pastures on which the cattle graze. The refuse of animals infected with watery-bags ought to be BURNED. No dog should be allowed even a smell of such offal.

Dr. Oliver's experiments as to eating beef infected with cysts show, as the experiments of others have long ago shown, that if the vitality of the cyst is destroyed by a high temperature, it will not produce tape-worm; but if the flesh is underdone, or if "scolices" of *cysticerci* are surreptitiously introduced into the food of a Hindoo boy of low caste (as described in the Report, or any other boy), the chances are, that, between three and four months afterwards, that boy will apply (as the Hindoo boy did) for tape-worm medicine. Surely such experiments are not now required, considering what has been already done in this direction (see p. 203, *ante*, and previous editions of this book for more extended details).

Tænia mediocanellata is acknowledged to be very common amongst the Mussulman population of the Punjab, the lower classes of whom are in the regular habit of eating half-cooked beef—indeed, prefer it so. It is amongst them that tape-worm is so prevalent. The late Dr. Oliver was of opinion, from his experiments and observations, "that the question as to the safety or otherwise of eating cyst-infested beef, simply depends upon the manner in which it is cooked. If this meat is thoroughly done, and presents no rawness when cut into, the 'measles' appear like little nodules of coagulated albumen, are doubtless perfectly inert, and may be eaten with impunity. But," continues Dr. Oliver, "it is not only thorough cooking that is required to guard soldiers in India from the ill effects of eating measly meat; but there is a want of cleanliness in the general arrangement of the kitchens, and serving of meals, which must offer great facilities for the introduction of *cysticerci* into the food. Barrack cooks, unless constantly looked after, are utterly careless as to the washing of chopping blocks, tables, dishes, and the like. The dish or pot cover on which the meat is placed when raw is often used without washing for serving the piece up for dinner; and I have myself picked up a *cysticercus* from the table on which a cook was preparing food." The dangers, too, of the parasite being conveyed by the cook's unwashed hands to the plates in which the meals are served, and the common practice of using the same knife for cutting up meat, and afterwards (without washing it) for other culinary purposes, must not be overlooked. With good selection and careful feeding, there seems to be every probability that *cysticerci* would soon almost or completely disappear from our Commissariat cattle. If they were entirely stall-fed, and watered from pure wells, there could scarcely be a possibility of infection after their purchase, and no possibility whatever if all ova-infested excreta were destroyed.

Notwithstanding the very excellent report by Dr. Oliver, of June 1, 1870, the Sanitary Commissioner with the Government of India, merely observes that "it is very desirable that the special attention of the Executive Commissariat officers should be drawn to the great importance of being careful about the water used by slaughtered cattle." Moreover, he writes that "very little is yet known of these cysts or their connection with tape-worm," and suggests a careful inquiry, without arousing any alarm or suspicion. But with all due deference to the opinion of the Sanitary Commissioner with the Government of India, there are no facts in medicine more clearly, accurately, and securely established (alike by experiment and observation), than the connection of such cysts with tape-worm, and of the ova of tape-worm with such cysts (see pp. 203-205,

ante, and previous editions of this book). Knowing this, and having seen every year, for twenty years past, the deficiency of practical acquaintance with the great facts of parasitic disease amongst medical candidates who have come to Netley, I am compelled by duty to teach doctrine opposed to the practice prescribed for adoption in India. By the evidence contained in the abstracts of proceedings of the Sanitary Commissioner, it appears that so soon as the feeding and watering of the cattle are cared for in the proper manner, the cyst-infection of beef disappears. It is to this careful feeding and watering, therefore, that the executive ought to turn its attention and endeavours. Because the executive fails to secure this cleanliness, is it politic that this cyst-infested beef-ration should be ordered to be used as food, *even when thoroughly cooked*? It is by no means a settled question what amount of heat or cold is necessary to destroy the vitality of cysts like the *cysticercus* or *echinococcus* (See Owen and Rudolphi's observations at p. 157, *ante*). It is generally believed that the boiling temperature of 212° Fahr. is sufficient to destroy the vitality of these cysts, and to convert them, as Dr. Oliver describes, into nodules resembling albumen. But the boiling point is not the ordinary temperature at which good wholesome meat is usually cooked, if it is well cooked, so as to retain the nutritive juices in its interior. The outside may approach that temperature, and may even reach it in baking or roasting; but 158° to 170° Fahr. is about the usual range of temperature at which meat is cooked in perfection, so as not to be hard or shrunken. Apart from the disgust, discomfort, and disease to the soldier, the mere permission to use cyst-infested ration, and far more, the authoritative order that it must not be condemned, but must be eaten when thoroughly cooked, opens a door for the wide extension and continuous propagation of these parasites—a door that ought to be most rigidly closed. The tendency undoubtedly is to undercook animal food rather than thoroughly to cook it, or *overdo* it. Thus greater facilities are afforded for such cysts to live and become tape-worms, capable in their turn of shedding myriads upon myriads of ova, which may find their way into other cattle and herbivora grazing on foul cantonments, and drinking the *tænia*-ova-infested water of the tanks. Compare the ultimate and inevitable consequences of this policy with the policy of destroying all cyst-infested meat, and the incidental loss at first of 8,000 or 10,000 pounds of meat in a year (the average yearly amount destroyed for 1868 and 1869) at 6d. or even 1s. a pound, and the certainty that every year the loss would be less, and that such parasites would be eventually stamped out, *if care be taken as to the feeding and watering of the cattle, and destruction of all ova-infested excreta, alike in man and animals*. Cyst-infested ration-beef, and all hydatids or watery-bags, ought to be *uncompromisingly condemned to destruction by fire*. There can be no doubt that parasitic disease has a firm hold, and is most disgustingly prevalent, among the inhabitants of India (both man and beast), and especially in the district of the Punjab (see subsequent remarks by Dr. Gordon). I believe it to be increasing, not only from accounts I receive from India, but from the prevalence of tape-worm (*mediocanellata* variety) in the intestines of invalids from India who happen to die at Netley, and in whom the previous existence of a tape-worm was quite unknown. As a rule, patients

do *not* complain of tape-worm; and unless they happen to be in hospital for other diseases, the medical officer has little chance of knowing how prevalent tape-worm may be among the men of a regiment. If the medical officer desires to know this, he must make a special inquiry, and with his *own eyes must look* for the presence of tape-worm links (proglottides) in the *daily* faecal evacuations of every man. This is impossible with men in health; but it can be done in hospital. A medical officer who did this amongst the patients in the hospital writes me, that he found *six* men out of thirty (one-fifth) suffering unknowingly from tape-worm when he examined specially to determine the point. How many cases were outside hospitals in India, there are no records to show. The practical importance of this fact is obvious. All such ova-infested excreta ought to be as uncompromisingly destroyed by fire as the cyst-infested ration or hydatid. If such excreta are not destroyed, the ova inevitably find their way on the grazing-grounds of cantonments, or by surface conduits and drains into the water of tanks. If, on the one hand, cyst-infested beef is suffered to be eaten; and, on the other hand, the excreta of tape-worm-infested-men are allowed to go undestroyed, the propagation of parasitic disease broadcast over India must be the inevitable result. *Prophylaxis*, therefore, is all important; and the entrance of the *scolices* ought to be prevented. With *prophylaxis* in view, the following remarks by Dr. C. A. Gordon, C.B., are not less revolting than suggestive:—

“*Tenia* appears to be of very frequent occurrence among the white troops in Upper India, and especially in the Punjaub; and I have been told by some medical officers who have been stationed at Peshawur, our nearest cantonment to Afghanistan, that they firmly believe every third soldier has had tape-worm during the two years regiments remain there. From what I have been able to ascertain on the subject, natives are not particularly liable to tape-worm, and certainly not more so in the north-western parts of India than in Lower Bengal. This is generally attributed to their almost total abstinence from animal food; and when we consider that both Hindoos and Mussulmans—all except the very lowest classes—abhor pigs’ flesh, while our own countrymen are very partial to it, and the common soldier probably not very particular regarding the early history of the animal that is converted into pork for his use, an additional circumstance in favour of the transformation of the *Cysticercus* constituting the ‘measles’ of pork into *Tenia* is thus disclosed to us. Those who have escaped the misfortune of having had to pass some years in India can form no idea of the vast herds of lean, half-starved pigs that roam over the fields and waste grounds in the vicinity of villages, neither can they have any conception of the nature of the food on which these pigs subsist. The natives of India perform their ordinary natural functions in the open air on a piece of waste ground, left for the purpose on the outskirts of every village; and where, morning and evening, men, women, children, and pigs dot the ground at short intervals from each other. In an incredibly short space of time after the villagers have left the field it is as clean as if they had never been there, while the herd by which the clearance has been effected may be found in some shady place near or close to a tank, with the exception of a few of the more insatiable, that have gone to hunt for dead dogs, cats, cattle, and Hindoos that have paid the debt of nature since the previous meeting, and have been thrown

or left on the plain to be devoured by domestic animals or vultures. Pigs, however, are not the only animals that live in this filthy manner in India; cattle and sheep, that are so particular in their food in Britain, acquire degenerate tastes in India; and it is needless to enter into similar particulars regarding ducks, fowls, turkeys, and pigeons, all of which are more or less used as food by our countrymen there. I have thus alluded to these matters with a view to indicate some circumstances that most unquestionably tend to vitiate the quality of the animal food upon which our troops in India must subsist; and I think I have at least shown a sufficient cause for almost any amount of disease in the bodies of these animals; as also why their flesh should be more liable to become diseased in Upper India than in Lower Bengal" (*Med. Times*, No. 357, May, 1857).

Abstinence from the practice of eating raw meat is to be strenuously recommended. Cases closely inquired into will often be found to take their origin from the habit of eating animal food imperfectly cooked or *underdone*. Children have been affected with *Tænia* on being weaned, from the custom of giving them pieces of pigs' flesh to suck in an uncooked state, containing *Cysticerci*. Cooks and butchers are known to be more liable than other people to be infested with tape-worms; and in countries where raw or uncooked meat in the shape of fish, flesh, or fowl is much used, there tape-worms greatly abound—*e.g.*, Abyssinia. By contrast, this is remarkably brought out in that country. It is observed that those who there abstain from flesh are altogether free—*e.g.*, the Carthusian monks. Vegetables eaten green, such as salads, ought to be scrupulously clean; for it is through green vegetable food and fruits that the ova which lead to the growth of *Cysticerci* and *Echinococci* make their way into our bodies. Hence these immature forms of parasites are most common in cattle; while the mature tape-worms are mostly found in the carnivora. Man, being more or less omnivorous, is doubly hospitable—entertaining as a host both forms of unwelcome guests, the tape-worms and the immature cysts. In place of burying the excreta of animals known to be affected with *Tæniæ*, all such excreta ought to be burned. If they are simply buried, or merely allowed to drop and lie on the ground, multitudes of minute embryos escape destruction; and may ultimately find their way into the human body. All entozoa (not preserved for scientific investigation and experiment) should be thoroughly destroyed by fire when practicable, and under no circumstance should they be thrown aside as harmless refuse (COBBOLD).

Symptoms of the Presence of Tape-Worm and Cystic Parasites.

From what has been stated, it is obvious that these must be variable, determined by the form of the parasite and the locality in which it exists. Almost every case has some special symptoms of its own, local, reflex, or general. In the alimentary canal the tape-worms occasion uneasiness, or pain in the abdomen, sometimes spasmodic, gnawing, or biting, but more frequently a distressing feeling which cannot be described. Irritation at the mucous orifices (mouth, nose, and anus) are also charac-

teristic accompaniments. The bowels may be constipated or relaxed; the stools exceedingly dark or white; the appetite sometimes lost and sometimes voracious; sleep disturbed, and temper fretful; and often, as a result of so many combined irritating causes, a remittent fever occurs, which has received the name of "worm fever." The mind is also often so much depressed as to amount to hypochondriasis (see page 155, *ante*). The symptoms of the existence of worms in the *small intestine* are often exceedingly obscure, and simulate many other diseases; so that, until a patient has passed a portion of the parasite worm, or the whole of it, we are unable to predicate its existence with any certainty; and at no time till we actually see the parasite can we determine its species.

Medical Treatment of Tape-Worm.

For the ejection of *Tænia* from the small intestines a great many remedies have been recommended; but in the present day medical men very generally limit themselves to one or two special remedies and to the action of purgatives. The celebrated Swiss remedy, purchased by one of the kings of France, was a purgative composed of twelve grains of *calomel* and twelve grains of *scammony*, followed shortly after by half an ounce to an ounce of the *sulphate of magnesia*. In many cases such a purgative has been efficient. The *oleum terebinthine* alone has been found to act well. Half an ounce to two ounces of this medicine may make the patient slightly tipsy, but is less likely to cause strangury than a smaller dose, and produces three or four motions. In one of these the worm is usually found, the animal having, it is supposed, a great antipathy to this substance, lets go its hold, and actively attempts to escape. The dose may be repeated twice a week, if the first is not sufficient. Three-fourths of the inhabitants of Cairo are said to be infested with *Tænia*, and the native remedy is twenty to thirty drops of *petroleum*—an agent not greatly dissimilar to turpentine. The *Grenadine bark* has acquired much reputation in the West Indies; but it has not supported the hopes that have been entertained of it in this country. Others have employed, with great success in tape-worm, the *etherial oleo-resinous extract of the male shield fern* (*Lastræa Filix-mas*) in doses of twenty to twenty-four grains—a remedy which by many is still believed to be the most efficacious. The *liquid extract of fern root* is the officinal remedy of the British Pharmacopœia. It is made from the *rhizoma* or rootstalk of the *Aspidium*, or *Nephrodium Filix-mas*, according to the formula:—*Fern root*, in coarse powder, 1 part; *ether*, 2 parts, or a sufficiency: percolate and distil off the ether, and the liquid extract remains. The dose is ℥30 to ℥60, in milk, mucilage, syrup, or the yolk of an egg. Mr. Squire finds the extract of the unexpanded frond equally effective with that of the rhizome. The powder may be used alone in doses of one to three drachms. The patient ought to refrain from food for a day, taking only liquids, such as milk or beef tea; then a full dose of castor-oil is to be taken in the evening; and, on the following morning, the necessary dose of *Male fern*. A remedy sometimes used in Germany is Chabert's *bandwurmöl*, or the "*oleum Chaberti contra Tæniam*." It is obtained by the distillation of twelve

ounces of oil of turpentine mixed with four ounces of the *oleum animale fœtidum*, which is the crude oil obtained from hartshorn and animal bones. A remedy of Abyssinian origin, called *Koussou* or *Cusso*, the flower of the *Brayera anthelmintica*, has recently been much recommended; and has doubtless been of great efficacy in some instances. It is administered in the form of powder, of which half an ounce is mixed with half a pint of warm water, and the infusion, well mixed *with the sediment*, is to be taken at two or three draughts, in the morning, on an empty stomach. If the bowels fail to be moved, a brisk cathartic ought to be given in three or four hours. Another remedy of more recent recommendation is *Kameela* or *Reroo*, the *Rottlera tinctoria* of the natural order *Euphorbiaceæ*, and sub-order *Crotonææ*. One to three drachms of the powder, suspended in gruel, mucilage, treacle, or syrup, will of itself expel the worm. Dr. Clymer suggests the following formula:—

Rottleræ, ʒi. ; *Oleo Resinæ Filicis*, f ʒss. ; *Mucilag. Acaciæ*, *Syrupi Aurantii*, ā ā f ʒii. ; *Misce et signa*. One-half at bed-time, and the remainder the next evening—the patient fasting ten or twelve hours before. Should it not operate adequately, a dose of castor-oil should be given.

The duration or life of the parasite within the human intestine varies from a few months to thirty-five years (WAURUCH); and although it is considered by some absolutely necessary that the *head* of the animal should be expelled before the treatment can be considered successful, yet there are good grounds for believing that there is considerable chance of destroying the parasite if large masses near the head are brought away. The worm is then likely to perish, the most actively growing and important portions being discharged. The head of the worm ought always to be sought for during treatment; at the same time, not being easily found, the physician is not to be discouraged by not finding it. The excreta ought to be mixed with water, passed through a filter of coarse muslin in order to search efficiently for the head.

Flukelike Parasites.—They are of a flattened oval form, smooth, soft, and yellowish, or yellowish-brown, and not jointed nor segmented. They are provided with two discs; one, situated at the pointed head extremity, forms a funnel-shaped depression leading to the oval orifice of a ramifying or bifurcating alimentary canal, which has no anal aperture. The other disc is situated on the abdomen, and terminates by a blind concavity. The opening of the sexual organs lies between the two discs. Male and femal sexual organs are embodied in one and the same individual; and (as in parasites generally) they pervade a very large portion of the body of the mature adult animal. A study of these parasites is of practical importance, chiefly in relation to their ova and early embryotic forms, which are now known to be developed in the open waters, or in minute water animals. It is therefore incumbent on us to be able to recognise them, and to know something of their natural history. The generation of some of them constitutes one of the most remarkable parts of the history of the process of reproduction among *Invertebrata*. Two states or forms are known to exist—namely, a mature or

free form, and an immature or encysted form. It is only in the mature form that the generative organs are found. Such mature *Distomata* have their *habitat* in the biliary passages, especially of the *Ruminantia*, as well as of man. Among sheep the disease induced by *Trematodes* often commits great devastation, and is commonly known by the name of "*the rot*." These flukelike parasites are found in grazing cattle, and especially in the horse, ass, hare, rabbit, squirrel, antelope, deer, beaver, and man. They are chiefly injurious to man indirectly, by their prevalence in the lower animals, causing meat to be unfit for food, although such meat is largely eaten by poor people.

The odour of sheep with rot from the liver-fluke is peculiarly offensive; the flesh is wasted, flabby, and watery; and hundreds of thousands of sheep die every year, especially in marshy districts, from the effects of this parasite.

Egg of *Distoma*.*Opalina*.

Fig. 44.

It is only very recently that the facts connected with the natural history of the liver-fluke have been ascertained, and the general result may be stated as follows:—The fully grown and sexually mature *Trematodes* (as exemplified in the *Distomata*) are oviparous. The egg is $\frac{1}{180}$ of an inch long, and $\frac{1}{70}$ of an inch wide. When the egg of the *Distoma* opens (by the springing up

of a sort of hood, which gives it the operculated appearance at one end) it gives vent to an embryo which moves rapidly by means of cilia, as is the case with many infusoriæ, and especially of the *Opalina*, now regarded by Schultze, Agassiz, and Stein as the earliest embryonic form of a *Distoma*. The discovery was made by Agassiz that a genuine *Opalina* (Fig. 44, *b*) was hatched from the egg (Fig. 44, *a*) of the *Distoma*. As such they are found in sewage water (see A. H. HASSALL'S *Reports on the Water of London*); also in the fæces of animals infested with liver-flukes, their ova passing out with the bile. The eggs continue to develop in water, and each egg finally produces an embryo, which swims freely by cilia. They die in pure water, as many vermicular animals do that would be more fortunate in water which is dirty, full of organic impurities, and abounding in food fitted for them. From each of these ova is formed an embryo, in which no resemblance to the parent *Trematode* is to be recognised; but presenting the simple structure of a ciliated animalcule, like a polygastric infusorian, known as the *Opalina*. This embryo is not itself converted by any direct process of development or metamorphosis into a perfect *Distoma*, but has a progeny gradually formed from germ-cells within it, and consisting sometimes of one, but more frequently of a number of bodies which, when they arrive at maturity, present each one an external form and internal structure and locomotive powers entitling them to be considered as independent animals. Nor are these directly converted into *Distomata*; a new progeny of animals is formed as before, nearly similar to those producing them, and equally differing from the complete *Distomata*. Each individual of this new progeny, as it increases in size, has formed within it, by development from germ-cells, the third progeny of the series and

last of the cycle. These are different from their immediate parents, and in their internal organisation soon manifest the type of the true *Trematode*. They are endowed for a time with very active locomotive power, to which a long caudal appendage contributes. Their progenitors have been confined in the parasitic condition; but these are in general freed from confinement, and move about with great vivacity for a time in the water, surrounding the animals which their progenitors infested. In this state they have been long known as *Cercariæ*, having the appearance of minute worms with tails, and were classed by Mueller, the Danish zoologist, with the *Infusorias*. Regarding the history of the development of these animals, information is only fragmentary; yet so different periods have been observed in the development of different species of these *Trematodes*, that by analogy a tolerably connected history has been obtained of the whole process. These *Cercariæ* are observed to be exceedingly lively and active in water both as to body and tail; and after moving about free in the water, they begin to go through a metamorphosis of a most extraordinary kind. They either become inclosed like a chrysalis in a pupa case, or, penetrating the bodies of soft animals, or their embryos, inhabiting the water—e.g., snails, fish, larvæ of insects, and the like—they become encysted within them. They thus become parasites, for example, in the mussel family (*Mytilus edulis*); in the ovisacs of the *Gammarus*, or sand-hopper (a small crustacean abounding on our sandy shores, which may be seen leaping up in myriads from the beach); also in snails, frogs, newts, and the like. In the cavities of these animals it lives as a ciliated animal, and multiplies by division of its body.

The next change in these parasites is that in which the cilia vanish. They fix themselves, and become oval motionless bodies (STEENSTRUP, PRITCHARD, p. 270), which continue to grow, and in which a germinal mass becomes visible, of an elongated form, out of which the first form of a *Cercaria* arises. It is a single-mouthed parasite, and is known as the *Monostoma mutabile*, inhabiting the intestinal cavity of water birds. In the interior of this parasite arises another embryo, which becomes free, and passes out of the bird into the water again; and being yet furnished with cilia, it makes its way especially into the aquatic larvæ of insects—e.g., ephemera; caddice worms (*Phryganidæ*); the dragon-fly (*Libellulidæ*); and into the soft embryos of fishes like the cod; in snails, mussels, and such kinds of water animals;—all used as bait and food for fishes. Out of this ciliated embryo a *germ pouch*, as it has been called, makes its way; and from this germ pouch the *Cercariæ* broods are developed. In the eyes of fishes, they inhabit the aqueous humour. Thus it seems probable that *Distomata* enter the human intestinal canal, and that of animals as *Cercariæ*, and pass thence into the biliary passages (FRERICHS); and according to the observations of Giesker and Frey, they may also penetrate directly into the skin, and undergo development in the subcutaneous cellular tissue. Two *Distoma* were found in the interior of a tumor on the sole of a woman's foot; and in all probability, as Frerichs remarks, the *Cercariæ* had entered the sole of this woman's foot while she was bathing in stagnant portions of the lake at Zurich. In another instance this parasite was found in a cyst behind the ear of a sailor, aged thirty-nine; and in an abscess in the head of a boy twenty-five

months old (MURCHISON, in Translation of Frerich's *Diseases of the Liver*, Vol. II., p. 488). The eating of uncooked fish, whelks, shell-fish, and uncooked garden snails, are all obvious modes in which the *Cercariæ* of the *Trematoda* may find an entrance into the bodies of man and other animals.

Ten species of flukelike parasites have been found in man. They have been named as follows:—

Fasciola hepatica, in its full-grown condition measures from eight to fourteen lines in length, and from three to six lines in breadth.

Distoma crassum.—In 1843 Mr. Busk found fourteen of these *Distoma* in the duodenum of a Lascar who died on board the "Dreadnought" hospital ship in the Thames. They are thicker and larger than those of the sheep, varying from one and a half to nearly three inches in length.

Distoma lanceolatum.—Very few (only three) instances are known of its occurrence in man. The eggs are $\frac{1}{830}$ of an inch long, and $\frac{1}{330}$ wide.

Distoma ophthalmobium.—It is recorded that four specimens have been found in the eye of a child five months old, born with lenticular cataract. No one of them exceeded half a line in length; and they were situated between the lens and its capsule, where they could be recognised as so many dark spots on the surface of the lens (COBBOLD).

Distoma heterophyes was found by Dr. Bilharz, of Cairo, in 1851, in the small intestine of a boy; and on a second occasion he collected several hundred specimens under very similar circumstances. The parts infested displayed a multitude of reddish points, due to the presence of dark-coloured ova in the interior of the worms. The length of the largest specimens did not exceed three-fourths of a line (SIEBOLD, COBBOLD).

Bilharzia hæmatobia is so named by Dr. Cobbold in honour of its distinguished discoverer. It is, however, a bisexual parasite. The body of the male is thread-shaped, round, white, and flattened anteriorly. The oral sucker is triangular; the abdominal sucker at the end of the trunk is circular. Below this, at the curved margin of the abdomen, a furrowed canal exists for the reception of the female. This canal is peculiar, and renders this *Distoma* generically distinct from the *Distomata* already noticed. The genital pore lies between the abdominal sucker and the commencement of the *canalis gynæcophorus*. The female is very thin and delicate; its tail is not provided with any canal. The suckers resemble those in the male; but the genital pore and the abdominal sucker are in contact. The length of the animal amounts to three or four lines, and the male is broader than the female (FRERICHS, MURCHISON, COBBOLD). Another name has therefore been given to it—namely, the *Gynæcophorus hæmatobius* (DIESING); but by whichever name it is known, it is of remarkable interest, not less from its peculiar anatomical structure, than from its great prevalence on the borders of the Nile, and from the grave and characteristic symptoms to which it gives rise. According to Griesinger, it has been met with in Egypt 177 times in 363 *necropsies*—i. e., equal to 33 per cent. The first specimens were discovered by Bilharz, of Cairo, in the portal vein and its branches, and likewise in the walls of the urinary bladder. It

has been since observed by Griesinger, Reinhart, and Lautner in the veins of the mesentery, urinary bladder, ureters, and pelvis of the kidney, giving rise to a formidable and very prevalent disease. In the larger vessels, such as those of the liver, this *Distoma* gives rise to no derangements equal to those which result when it exists in the lining membrane of the urinary passages and the intestinal canal. There it induces hæmorrhage and inflammation. In the intestine they are often associated with appearances resembling those of dysentery, with congestion, extravasation of blood, deposit upon and beneath the mucous membrane, fungoid excrescences, and croupy exudations that occupy ulcerated patches of the bowel. In many of these cases the eggs of the creature may be found wedged in long rows within the intestinal vessels, or in and beneath exudations on the free surface of the mucous membrane. Hence it has been suspected whether the dysentery endemic to Egypt may not have to the presence of these *Distoma* the same relation as “itch” has to the *Acarus*. Such a conclusion receives a strong confirmation when we turn to the lesions produced in the urinary apparatus. Here the mucous membrane appears swollen in places which are covered with a soft, sandy, rotten mass, firmly fixed to the subjacent tissue. The microscope shows this mass to consist of the full and empty shells of the parasitic ova, imbedded in a mixture of blood, exudation, modified epithelium, and crystals of uric acid. The thickening of the submucous tissue often produces stricture of the ureter, which is followed by retention of urine and all its dangerous consequences,—degeneration of the kidneys, pyelitis, dilatation of the renal pelvis, or atrophy of the substance; or the masses themselves become the nuclei of calculous deposits, and thus aid in the chlorotic exhaustion these creatures produce in the person they inhabit, by the consumption and loss of blood they imply. Lastly, it seems not unlikely that the dislodgement of clots into the general circulation sometimes brings about pneumonia, in the way described by Virchow, and illustrated by the clinical researches of Kirkes (*Brit. and For. Med.-Chir. Review*, l. c., p. 125). Professor Virchow was good enough to show me a specimen, in his museum, of lesions in the bladder effected by the existence of this *Distoma*. The entire trunk of the portal vein is sometimes filled with this parasite, while their ova are found in the tissue of the liver; but the symptoms to which the *D. hæmatobium* gives rise are more referable to the urinary organs than to the liver. The urine is bloody, and sometimes contains the ova of the *Distoma*; and a state of profound cachexia supervenes (FRERICHS, COBBOLD, MOQUIN-TANDON). Dr. John Harley, of King’s College, London, has recently directed the attention of the profession in this country to the remarkable prevalence of hæmaturia at the Cape of Good Hope—a condition which he found associated with the fertile ova of this entozoön passed in the urine. After micturition, a little blood, never exceeding a teaspoonful, or some dark coagula, like “veins,” appear with the last half-ounce of urine. The urine itself is never bloody. Sometimes the blood-coagula will block up the urethra, and cause obstruction for a few minutes. These are all the symptoms which appear in connection with the urinary apparatus; and numbers of people of both sexes are affected in precisely the same way in certain parts of the Cape—as endemic hæmaturia—especially at

Uitenhage and Port Elizabeth. In various samples of urine sent to him by a person suffering from this affection, he invariably detected the ova of this entozoön. Of these he was kind enough to give me specimens, and a copy of the woodcut (Fig. 45). The eggs measure $\frac{1}{200}$ of an inch long, and $\frac{1}{500}$ of an inch broad. He was successful in observing the perfect ciliated embryo after its escape from the shell (Fig. 45, 2 and 3).

Dr Cobbold has discovered the same *Distoma* in the portal system of an African monkey.

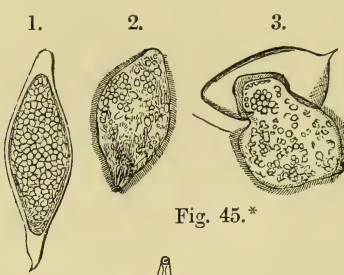


Fig. 45.*

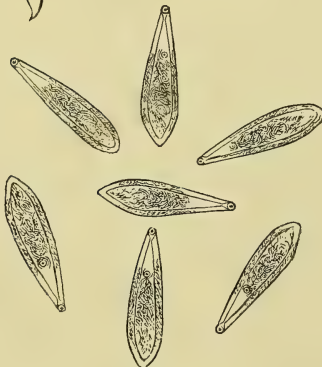


Fig. 46.† Ad Nat.

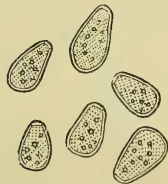


Fig. 48.§ × 300.

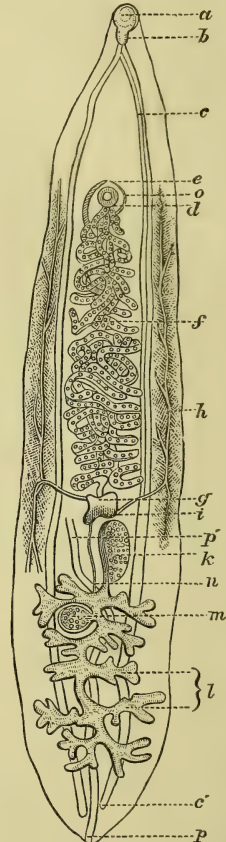


Fig. 47.‡ × 6.

* 1. Ovum of *Distoma hæmatobium*, from hæmaturia of the Cape of Good Hope (Dr. JOHN HARLEY); 2. Embryo (ciliated) from ovum capsule; 3. Embryo attached to the ovum capsule.

† Fig. 46 represents the flukes (*Distoma sinense*), drawn to natural size.

‡ Fig. 47, one specimen enlarged to about six times the natural size, showing the ventral surface. *a*. Oral sucker. *b*. Oesophagus. *c*, *c'*. Right alimentary canal. *d*. Ventral sucker. *e*. Genital orifice. *f*. Uterine folds. *g*. Ovary. *h*. Vitelline gland (right). *i*. Vitelline duct (right). *k*. Right testicle. *l*. Receptaculum seminis (?) *m*. Left testicle. *n*. Vas deferens. *o*. Termination of vas deferens. *p*. Pulsatile vesicle. *p'*. Water-vascular canal.

§ Fig. 48 represents ova magnified 300 diameters.

Distoma sinense (Fig. 46) is new to science, having been discovered by Dr. J. E. P. M'Connell, Professor of Pathology and Resident Physician, Medical College, Calcutta, on making the *post-mortem* examination of the body of a Chinaman, on 9th September, 1874, when he found in the liver, obstructing the bile ducts, a large number of "flukes." The Chinaman was a carpenter, aged twenty. He was brought to the Medical College Hospital, Calcutta, in a moribund state, having been suffering from fever of a continued type for a fortnight. He was profoundly insensible, and died two hours and a half after admission. The liver was found large, swollen, and tense; superficially of a dark purple colour, but paler, more dingy or muddy-looking on section. Its parenchyma everywhere was very soft. The large portal and hepatic veins were filled with fluid blood; and the bile ducts were particularly distinct from their large size and distension with thick yellow bile. On incising the liver in different directions, it was noticed that small dark vermicular-looking bodies escaped on to the table. These were seen to protrude from the bile ducts, which were found more or less obstructed by and containing them in large numbers; some lying free, others coiled up, and others solitary, or in groups of twos or threes, within the biliary ducts, and there was hardly any duct of medium size which did not contain two, three, or more parasites. None were found in the gall-bladder. Numerous ova were found in the biliary canals. The parenchyma of the liver was intensely pigmented with granular particles, in the interlobular tissue, and also within the hepatic cells. The presence of the flukes in the biliary ducts appeared to be the exciting cause of the acute and extensive structural degeneration of the proper tissue of the liver, and of that cholæmic condition induced by the obstruction of the biliary channels, which appears to have been the immediate cause of death. Professor M'Connell justly considers that the discovery of this *Distoma* may throw some light on the pathology of those obscure liver enlargements and jaundice which every now and then, in India, present themselves for treatment. Many Chinamen, especially in Calcutta, have applied for the treatment of obscure, hard, massive enlargements of the liver, with or without jaundice, and which, in the absence of lardaceous disease, may be due to such parasitic infection as Professor M'Connell has described. (*Veterinarian*, Oct., 1875, and *Lancet*, Aug. 21, 1875.)

The *Tetrastoma renale*, as its name implies, infests the tubes of the kidney, and was discovered in 1833 by Lucarelli and Della Chiaje. It attains a length of five lines, has an oval flattened body, and is furnished with four suckers disposed in a quadrate manner at the caudal extremity. The reproductive organs are situated near the mouth.

Hexathyridium venarum has been found in venous blood, and from the sputa of persons suffering from hæmoptysis (TREUTLER, CHIAJE, FOLLINA). It attains a length of three lines, is cylindrico-lanceolate, with six suckers biserially disposed on the under side of the so-called head (COBBOLD).

Hexathyridium pingicola was once found in a diseased ovarium. The parasite attains a length of eight lines (TREUTLER, OWEN).

The following table represents the more prominent differentiating characters of the three principal *Distomata* found in man—

	D. HEPATICUM.	D. LANCEOLATUM.	NEW LIVER-FLUKE.
<i>Length</i> , . . .	$\frac{3}{4}$ " to 1".	$\frac{1}{3}$ " (8.9 mm.)	$\frac{7}{10}$ ".
<i>Breadth</i> , . . .	$\frac{1}{2}$ " to $\frac{2}{3}$ ".	$1\frac{1}{2}$ ".	$\frac{1}{2}$ ".
<i>Oral sucker</i> , . .	Smaller than ventral.	About same size.	Larger than ventral.
<i>Integument</i> , . .	Covered by minute spines.	Smooth.	Smooth.
<i>Intestinal canal</i> ,	Branched.	Not branched. Terminates at lower fourth of body.	Not branched. Extends through the whole length of body to the caudal extremity (nearly).
<i>Male reproductive organs</i> , .	Form a series of spread-out vermiform tubes.	Form two distinct globular testes, placed one above the other in the median line <i>directly below</i> the ventral sucker.	Form one or two globular testes, occupying the lower third of the body <i>below</i> the female reproductive organs, and are connected with a very remarkable development (receptaculum seminis?).
<i>Female reproductive organs</i> ,	Occupy central and hinder parts of body <i>below</i> the testes.	Occupy the anterior and middle third of the body <i>above</i> the testes and seminal apparatus.
<i>Ovum</i> , . . .	$1\frac{1}{8} \times \frac{1}{16}$.	About $\frac{1}{10} \times \frac{1}{33}$ ".	$\frac{1}{33} \times \frac{1}{100}$ ".

Symptoms.—The symptoms to which *Distomata* give rise in the human subject must of course vary with the site of the parasite; but nothing definite is known regarding them, except in cases of *hæmaturia* and *dysentery*, already noticed. In sheep their presence occasions dilatation and catarrh of the biliary passages, accompanied by atrophy of the hepatic tissue. Jaundice rarely shows itself, and then only lasts a short time; but ultimately a condition of anæmia is developed, under which numbers of sheep die. The disease is known as one form of "the rot;" and it prevails to a considerable extent among flocks feeding on marshy and wet land near the shores of rivers.

CLASS C.—ACCIDENTAL PARASITES.

Definition.—*Internal parasites, having the habits, but not referable to the class of entozoa.*

Pentastoma denticulatum has been conclusively shown by Leuckart's experiments to be the *larvæ*, or sexually immature condition of *Pentastoma tænoides*, and is found in the liver and small intestines.

Pentastoma constrictum.—The author had two portions of lung and three portions of liver, each containing an unusual parasite, sent to him from Jamaica, in August, 1865, for the Museum of the Army Medical Department at Netley. Staff Assistant-Surgeon Edward Barrett Kearney, Esq., is the donor of the specimens; and from his history of the patient's fatal illness, the following account has been drawn up :—

“On the 11th of January, 1865, Private Isaac Newton was admitted into the hospital of the 5th West India Regiment, at Up Park Camp, Jamaica, for an attack of *tonsillitis*. He was an African, enlisted about eight months previously from the slave depôt at Rupert's Valley, St. Helena, where all slaves captured in slave-ships are kept until disposed of. He appeared to be about twenty-one years of age, and of a thin spare habit of body. On admission the tonsils were inflamed and enlarged, but not ulcerated; and there were aphthous ulcers about the tongue. He suffered from headache and pain across the back. On the morning of the 14th he complained of great pain in the abdomen, which became tympanitic. His tongue was clean, but vividly red at the edges and tip, and it felt dry to the touch. The skin was very hot, dry, and harsh; and his pulse 100, which continued to increase in quickness. Sordes soon began to appear about the mouth and teeth, and the tongue became furled and cracked. Large moist crepitation was heard over the whole surface of both lungs. He became low, and disinclined to be spoken to, and by six o'clock in the evening his mind appeared to be confused. He passed his urine and his stools involuntarily in bed. About nine in the evening of the 16th, he became suddenly worse. His pulse became weak and almost indistinct, the skin cold, the countenance sunken, and covered with a copious perspiration. He died at half-past ten that night.

“*Post-mortem Examination Fourteen Hours after Death.*—The general appearance of the body was that of emaciation, with yellowness of the conjunctiva. *Thorax.*—The subcutaneous areolar tissue over the chest and abdomen was of a deep yellow colour. The *Pericardium* contained about four ounces of deep amber-coloured fluid. The *Heart* was large and pale, but its substance was otherwise normal, and its valves healthy. The *Lungs* were both highly congested; and when cut into, a bloody frothy fluid exuded in quantity. The substance of both was friable and yellowish in colour. ‘On the anterior surface of the *right lung*, and near the edge of its lower lobe, one or two yellow specks appeared. They were about the size of a spangle, and when cut into, worms were seen regularly encysted in its substance.’ On the posterior surface of both lungs there were numerous adhesions of long standing. *Abdomen.*—The *Liver* was very large, extending into the left hypochondrium. ‘Its surface was dotted over, both posteriorly and anteriorly, with about twenty or thirty yellow specks similar to those seen in the lung.’ The hepatic substance appeared paler and rather more soft than natural. *Stomach.*—It was distended with air and fluid, containing about a pint and a half of dark-green coloured fluid. The mucous membrane was congested in patches, in the stomach, and along the whole tract of the intestines. There was no ulceration; and no appearance of *Tæniæ*, either con-

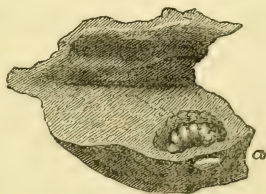


Fig. 49.

tinuous or in proglottides, could be discovered. Other *organs were healthy.*"

Description of the Parasite and the Lesions it produced.—Fig. 49 represents a small portion of the lung, with the little worm seen at *a* curled up in its cyst. The pleura has been removed, so as to expose the "rings," "markings," or "constrictions," which are characteristic of the body of this parasite. The pleura was opaque and considerably thickened, probably from irritation. The appearance of the parasite on the surface of the liver was exactly similar to that in the lung, and wherever the serous covering of the organ was sufficiently transparent, the constrictions of the parasite could be seen distinctly shining through.



Fig. 50 represents two specimens of the parasite removed from their cysts. They are of the natural size, and one of them, *a*, is much shorter than the other, the constrictions being closer together: *a* measures five lines in length, *b* measures about eight lines.* In diameter they are about one line. About twenty to twenty-three rings or constrictions can be counted on the elongated body, at tolerably regular intervals, and somewhat spirally arranged. Fig. 51 represents the two specimens of the parasite slightly magnified (about three diameters): *a* is the shorter; *b* and *c* are the posterior and anterior aspects of the longer of the two worms. The head end appears compressed, so as to be flat and square-shaped at the end. It is seen to be marked with five spots on the anterior aspect, as shown at *c*. The posterior aspect of the flattened head, as shown at *b*, is comparatively smooth. The elongated body is rounded, and the caudal end terminates in a blunt-pointed one. The constrictions appear like folds of the outer covering of the worm, each fold overlapping the one which follows, from the head to the tail. The body of the parasite is rounded, and not flat, as the tape-worms or cysticerci. Fig. 52 represents the anterior aspect of the flattened head end (*cephalothorax*) of the parasite. It is so highly magnified as to show the nature of the five spots or marks shown in Fig. 51. The dotted lines from *a* and *b* point to two pairs of hooks or claws—one pair on each side of a pit or mouth, *c*. The points of the claws, indicated by *a*, are seen nearly in profile; those at *b* are directed more towards the observer. These claws appear to be implanted in socket-like hollows or depressions, surrounded by much loose integument. These socket-like hollows appear to be elevated on the summit of the mass of tissue which lies underneath the folds of integuments surrounding the base of the hooks. These parts are regarded as

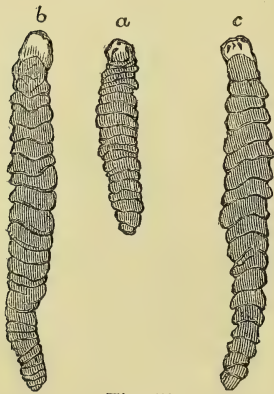


Fig. 51.

sents the anterior aspect of the flattened head end (*cephalothorax*) of the parasite. It is so highly magnified as to show the nature of the five spots or marks shown in Fig. 51. The dotted lines from *a* and *b* point to two pairs of hooks or claws—one pair on each side of a pit or mouth, *c*. The points of the claws, indicated by *a*, are seen nearly in profile; those at *b* are directed more towards the observer. These claws appear to be implanted in socket-like hollows or depressions, surrounded by much loose integument. These socket-like hollows appear to be elevated on the summit of the mass of tissue which lies underneath the folds of integuments surrounding the base of the hooks. These parts are regarded as

* With regard to the sizes mentioned, it is to be remembered that those dimensions are from the animal preserved in spirits, which causes such parasites to shrink considerably. The full size is given by Dr. Lawson from the fresh state as 1 to 1½ inches. (See following pages.)

the feet of the parasite, and the hooks are the foot claws. The pit or mouth (indicated by the dotted line to *c*) is of an oval shape, the long axis of the oval lying in the direction of the length of the worm. The lip or outer margin of the pit is marked by a well-defined thin line. There are no spines nor hooks on the integument of the elongated body.

From the description and the drawings here given, it will be seen that the parasite corresponds in its specific characters with the larval condition of the *Pentastoma constrictum*. It belongs to the family *Acanthotheca* of Diesing, and has no structural connection with the true helminth parasites found in the bodies of man and other animals. The parasite now described, when compared with the descriptions of *pentastomata* given by Frerichs, Cobbold, and other observers, demonstrates clearly that at least two species of *pentastomata* infest the human body; the *Pentastoma constrictum* being by far the larger, the more dangerous, and fortunately the more rare, of the two species. It is also still more satisfactory to know that, as a human parasite, neither of the two species has ever been detected in this country; and according to the researches of Dr. Cobbold, it is only in the encysted or larval conditions that the *pentastomata* are met with in the human body. As an embryo it becomes encysted. The cyst is composed of condensed connective tissue, and is lined by layers of loose flakes, which are evidently the remains of repeated castings of the skin of the parasite; and during the intervals of these successive moultings the worm makes considerable growth, so as to reach the size in which it is finally found. In this pupa or larval condition it occurs in the solid organs of the abdominal and thoracic cavities of man in certain geographical districts in Europe, Egypt, and the West Coast of Africa, and much more frequently in various herbivorous animals, such as the sheep, deer, antelope, peccary, porcupine, Guinea-pig, hare, rat, and domestic cat (COBBOLD). In all these animals, and in man, the larvæ usually occupy cysts immediately underneath the serous covering of the liver and the lungs; and Dr. Cobbold mentions that he has occasionally found the *Pentastoma denticulatum* free in the cavities of the abdomen and pleura of animals. Our knowledge of the natural history of these parasites is

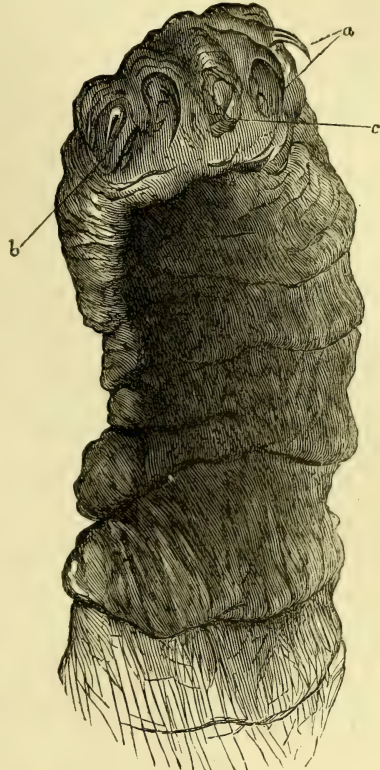


Fig. 52.

mainly derived from descriptions of the *Pentastoma denticulatum*, the larval or sexually immature condition of the *Pentastoma tenoides* of Rudolphi. We are told by Frerichs (*Clinical Treatise on Diseases of the Liver*, Vol. II., p. 276) that Pruner was the first observer who pointed out, in 1847, the existence of the *pentastoma* as a parasite in the human subject. On two occasions he found *pentastomata* in the liver of negroes at Cairo. He does not seem, however, to have determined accurately the nature of the parasite he observed; and he also subsequently found two specimens of the worm preserved in the Pathological Museum at Bologna, which had been removed from the human liver (COBBOLD). Bilharz has since repeatedly detected in livers of negroes at Cairo the parasite discovered by Pruner in 1847. Bilharz and Von Siebold made this parasite the subject of careful study; and they recognised in it a variety of *pentastoma* quite different from that which prevailed in some parts of Germany. They gave this new variety the name of *Pentastoma constrictum*—the parasite which proved fatal in the case whose history Dr. Kearney sent to me from Jamaica. It is the form of *pentastoma* endemic in Egypt, and hitherto it has only been found in the African negro. It differs from the *Pentastoma denticulatum* (the larval form of the *Pentastoma tenoides*), “in not being furnished with any integumentary armature of spines, and in its being a much larger worm” (COBBOLD, p. 402). The *Pentastoma constrictum* seems to be from eight to twelve times larger than the *Pentastoma denticulatum*, and therefore is all the more dangerous from its actual size (nearly an inch long); and when it occurs in great numbers, as in the present instance, it cannot fail to prove an extremely irritant “foreign body,” when it escapes into a serous cavity like the pleura or peritoneum—one of the modes in which it seems to cause death. The latter parasite (*P. denticulatum*) has been fully described by Frerichs, and figured by him in his *Atlas*, Plate xi., Fig. 9, as endemic in Germany in the human liver—in which organ it is considered to be far more common than the echinococcus.* Frerichs, however, regards the *pentastoma* endemic in Germany as devoid of clinical importance, because it does not give rise to any functional derangement. Not so, however, is such the innocent history of the *Pentastoma constrictum* as it affects the negro; and after the history of the case now given, the clinical importance of this parasite cannot be disregarded.

As to the mode in which it tends to cause death, the evidence in this case, from symptoms and *post-mortem* examination, seems to point to *pneumonia* and sudden collapse from *peritonitis*. The author is able also to verify this point in the pathology of this parasitic disease still more

* “In Germany,” says Frerichs, “the *pentastoma* was first found in the human liver by Zenker in 1854; it occurs, however, not only in this gland, but also in the kidneys, and in the submucous tissue of the small intestine (WAGNER). The parasite is by no means rare with us. Zenker, at Dresden, succeeded in finding it 9 times in 168 autopsies (or, according to Kuchenmeister, 30 times in 200 autopsies). Heschl, in Vienna, met with it five times out of 20 autopsies; Wagner, at Leipsic, once in 10. According to Virchow, it is more common in Berlin than in Central Germany. During six months at Breslau, I (Frerichs) met with it in 5 out of 47 dead bodies. As a rule there is only one present; in rare cases there are only two or three. It presents the form of a somewhat prominent nodule, from 1 to 1½ lines in length, which is formed by a firm fibrous capsule, easily detached from the surrounding parts. The animal lies coiled up in the interior of this capsule” (*On Diseases of the Liver*, Vol. II., p. 276).

clearly from a preparation which has been in the Museum of the Army Medical Department since 1854, but the nature of which he could not understand till the history of the case now published was so thoughtfully furnished by Dr. Kearney, together with the specimens of the parasite *in situ*. The preparation in the Museum (hitherto a puzzle to all who have examined it) consists of four pieces of liver (Fig. 52, *a, b, c, d*), and appears in the Catalogue with the following description:—

"Portions of liver, containing numerous small cysts (evidently some jointed entozoön), taken from Private George Sutton, 1st West India Regiment, who died at Bathurst, Gambia, 18th November, 1853; and for the history of the case reference is made to the quarterly report of sick and wounded from that station, dated March, 1854."

My friend, Inspector-General Dr. Lawson, has sent me (since the last Edition was printed) an abstract of the case carefully drawn up by himself, as follows:—

"This man arrived from the West Indies in May, and immediately after came under treatment for an affection of the lower dorsal vertebræ, which had commenced on the voyage. Blisters were frequently applied over the part with relief, but the general appearance did not indicate decided improvement. He continued much the same until the end of September, when œdema of the left foot and leg came on; in the course of a week the right leg was similarly affected. On 19th October a fluctuating tumor showed itself on the eighth and ninth ribs, on the right side of the back; this was opened with a small trocar, and a considerable quantity of matter evacuated, and a continued oozing went on from the wound subsequently. The patient got gradually weaker, and died on 18th November, at six A.M.

"An extensive abscess was found on the right side of the spine; the borders of the ninth, tenth, and eleventh dorsal vertebræ were denuded of periosteum and carious, and the sac of the abscess originating from them had extended upwards behind the right crus of the diaphragm, and opened into the cavity of the pleura of that side, while below it had passed behind the kidney, and for some distance under the psoas muscle. The peritoneum, though not penetrated by the abscess, contained a quantity of sero-purulent matter. The right kidney was a good deal inflamed, and the right psoas and iliac muscles were in that pale, dense, semicartilaginous condition described by Guthrie and others, as being caused by the constant irritation of fractured bones in wounds. The right iliac artery and vein were completely obstructed by a dense clot of firm coagulated lymph, and their coats were inflamed and thickened; the destruction embraced their branches, though the inflammation did not proceed farther than the top of the thigh. A number of small ulcerations were found in the mucous membrane of the colon, and a large number of worms in the liver. These worms varied in length from an inch to an inch and a half or more, and were found coiled up like a watch-spring in small sacs scattered through the whole organ."

The author is now able to identify this preparation as an example of

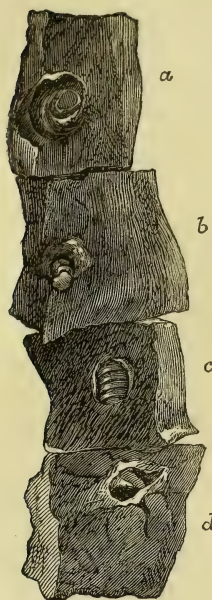


Fig. 53.

lesions produced by the *Pentastoma constrictum*. At *a*, in the cyst where the larva has been, there is contained the *débris* of integumentary exuviae; at *b* the head end of the parasite is seen peering out of an ulcerated opening in the serous covering of the liver. The edge of the opening is rounded and indurated, as if a good deal of local irritation had been maintained at the part previous to penetration of the serous covering. At *c* the ring-like constrictions of the parasite are seen shining through a very thin portion of serous membrane; and the portion of liver at *d* represents an empty cavity, whence one of these larvæ has passed out, probably into the peritoneum. The cicatricial-like contraction and puckering of tissue in the vicinity show that considerable irritation has existed previous to the exit of the parasite.

We have no information as to how the *Pentastoma constrictum* finds its way into the human body as an embryo and subsequent larva. Although in this instance the negro was stationed in Jamaica, it is most probable that he had the germs of these parasites within him when he left his native shores in some part of Africa; and that this parasite is

neither endemic in St. Helena nor in Jamaica. Reasoning from what is known regarding the propagation and development of the *Pentastoma denticulatum*, it is probable that the ova with the contained embryos are introduced into the human stomach along with uncooked vegetable food (fruits or salads), in regions where the mature animals are endemic. From the stomach the embryos, escaping from the ova, bore their way, and find a resting-place in the liver or other solid viscus, exactly like the embryo of the *tenia*. In solid organs (like the lungs and liver) they become encysted, and undergo the pupal transformation so well described by Leuckart and Cobbold in the case of the *pentastoma* endemic in Germany.

The drawings were made by Dr. Humphrey C. Gillespie, then Staff Assistant-Surgeon, from the preparations which are now in the Pathological Museum of the Army Medical Department at Netley.

Æstrus hominis.—Various insects and their larvæ are apt to infest man, such as “bots,” the *Æstrus bovis*, and the larva of a species of *musca* in Africa (KIRK). The *Æstrus hominis* is the larva of the *æstridæ*, the bot-flies, breeze-flies, or gad-flies. The females deposit their eggs on the bodies of different herbivorous animals, and each species of fly keeps to its own particular species of animal. Several cases are on record as having occurred in man.

Anthomyia canicularis are exciting causes of boils by their larvæ; and, under this head, it may be convenient to notice that my friend Dr. Albert A. Gore, while Staff Assistant-Surgeon on the West Coast of Africa, kindly favoured me with the following account of a larva or grub which is the exciting cause of the Bulama boil:—

“This small larva or grub (Fig. 54) is of a white colour, a line or two

in length, and is the exciting cause of a boil occasionally seen in the Island of Bulama and its neighbourhood. When magnified under a low power (Fig. 55), it appears to be divided into a series of joints, and covered with minute bulbous hairs. On the anterior division are placed four or five red spots (*b*), and from either side project two hollow suction tubes (*a, a*). The posterior extremity seems to be terminated by a blunt hook. In applying a higher power the bulbous hairs turn out to be a number of beautiful black hooklets (Fig. 56), which have a very pretty appearance on the white surface. A faint outline of a central cavity can be discerned. The hooklets are directed anteriorly.

“**Symptoms and Treatment.**—Attention is first attracted to the part by feeling an extreme itchy sensation. On examination a small red pimple is seen. After a while a small serous discharge oozes from its centre, which sometimes seems to pulsate. If allowed to progress, it becomes a regular inflamed boil, very painful, and often causing an erysipelatous blush, with inflammation in the neighbouring lymphatics, and tenderness of the glands to which they run. On the evacuation of the small abscess in the boil these symptoms gradually disappear, but a persistent red mark remains at the original seat of the disease. The treatment consists in poulticing until the little animal appears, when it can be withdrawn. If allowed to suppurate, it must be treated in the usual way of a boil under similar circumstances. The natives put in a mixture of salt and palm oil, which takes out the little grub.

“**Etiology and Pathology.**—This little worm cannot be the *chigoe* or *Pulex penetrans* of the West Indies, although it may result from the ova deposited by some similar aphanipterous insect. The *chigoe* chiefly attacks the toes or intervals between them, and causes a series of painful ulcers. It is also of a black colour. This small grub is white, causes a boil or two in any portion of the body, most commonly in the thigh, arm, or abdomen; it is sporadic, although endemic. They have one symptom in common—viz., the extreme itchiness. But this is complained of in nearly every case of disease resulting from insect or other living organisms.”

II.—ECTOZOA.

Definition.—*Animals living upon the skin and hair.*

Pathology.—These parasites are of three kinds—namely, lice, mites or ticks or spiders, and fleas.

(1.) The first kind belong to the family of “lice” (*Pediculidæ*), of the class *Insecta*, amongst the *annulose* or *articulated* animals. They belong to the order *Anoplura*, all of which are parasites and destitute of wings. They undergo no regular metamorphosis, as most other insects do; but in

* Natural size of larva or grub, the exciting cause of Bulama boil.

† The same grub magnified by a low power,—*a, a*. Hollow suction tubes; *b*. Fine red spots. The other dots on the body are hooklets.

‡ Two of the hooklets highly magnified. These drawings were furnished by Dr. Gore.

Fig. 54.*

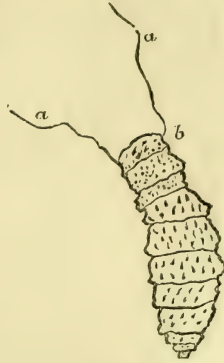


Fig. 55.†

Fig. 56.‡

their growth to maturity they shed their skin a certain number of times, which may to some extent explain the irritation and forms of lesion to which they give rise. Almost all animals—man, quadrupeds, birds, and reptiles—are liable to be infested with these parasites; and were it not for our instinctive feelings of disgust with regard to them, as opposed to our notions of cleanliness and propriety, the study of their forms and habits is of considerable interest to the pathologist. Upwards of 500 species of lice have been described—universally diffused over the animal kingdom in different climates of the world. Their superabundance upon the persons of the human race is associated with some severe lesions of the skin; and authentic cases are related of death from lousiness. A technical name has been given to this condition, namely,—*Phthiriasis*, which is a morbid state in which lice develop themselves to such an extent that a pruriginous eruption is produced (*Prurigo pedicularis*). The skins of persons liable to constitutional skin diseases in which watery or secreting eruptions (such as *eczema*) prevail, are those most favourable for the development of lice. Five forms of lice infest the skin of man. One variety is met with on the hair of the head—the *Pediculus capitis*; a second variety infests the other hairy parts of the body, but especially the pubis; and hence its name—*Pediculus pubis*; the third form lives on the general trunk of the body—the *Pediculus corporis*; a fourth is the *Pediculus palpebrarum*; and a fifth the *Pediculus tabescentium*. The first four species, although they live in close proximity to one another, yet strictly limit themselves to the regions mentioned. In a clinical point of view the *P. corporis* is the most important.

(2.) The second kind of animals associated with diseases of the skin belong to the family of “mites” or “ticks” (*Acaridæ*), of the class *Arachnida*, amongst the *annulose* or *articulated* animals. Some of these are free, others are parasitic, and vary somewhat in their structure accordingly. Those which live a parasitic life have the mouth in the form of a sucker. Such are the “ticks,” which fasten upon dogs, cows, sheep, horses, and other animals. They bury their suckers so deep in the skin, that it is impossible to detach them without tearing the skin to which they fix themselves; and they multiply so rapidly, that oxen and horses attacked by them have been known to die of mere exhaustion. The harvest ticks (*Leptidæ*), one species of which, the *Leptus autumnalis*, well known as the harvest bug, is common in autumn in grass and herbage, from which it gets on the body of man, and, though exceedingly small, produces extreme



100ths of an inch \times 10
diameters.
Fig. 57.*

irritation of the skin. One only of these parasitic *Acar*i has been found to live entirely in the skin of man—the *Sarcoptes galli*, or *Acarus scabiei*—producing the disease called the itch.

(3.) The third kind includes the common flea, *Pulex irritans*, and the *Pulex penetrans* or *chigoe*.

Phthirus inguinalis, or the *Crab Louse* (Fig. 57), has a shield-shape, and a much broader body in proportion to its size than any other form of louse; and there does not appear to be any distinct separa-

* The Crab Louse (after ANDERSON).

tion between its thorax and abdomen. It has been met with on all the hairy parts of the body, except the head, but more especially on the hair of the *pubis*. It does not run about like other lice; but grasps the stems of the hairs with its forelegs, and adheres so firmly that it is difficult to remove it without pulling out the hair. The *nits* or egg capsules are attached to the hairs in the same way as on the head. Pruriginous or eczematous eruptions, which may become pustular, are results of their existence (ANDERSON).

Pediculus capitis, or the *Head Louse* (Fig. 58), is considerably smaller than the body louse. Its legs are larger in proportion to the size of the body than those of the *P. corporis*; and the abdomen is more distinctly divided into seven segments, separated from each other at the margins by deep notches. They propagate with astonishing rapidity; and, by their irritation produce an *eczema*, from which the fluid exudes abundantly, and crusts are formed, involving the cuticular *débris* or exuviae of the lice and the remains of epidermis. The hairs become glued together; partly by the fluid from the *eczema* and partly by the secretions of the insects as they deposit their ova in the capsules which they fix to the hairs (Fig. 59). These capsules are commonly called *nits*, and they adhere with great tenacity to the hairs.

Pediculus palpebrarum, the *Louse of the Eyebrows*, is a doubtful variety.

Pediculus vestimenti.—The *Body Louse* (Fig. 60) is

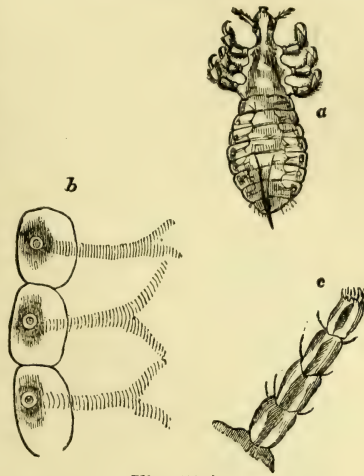


Fig. 58.*

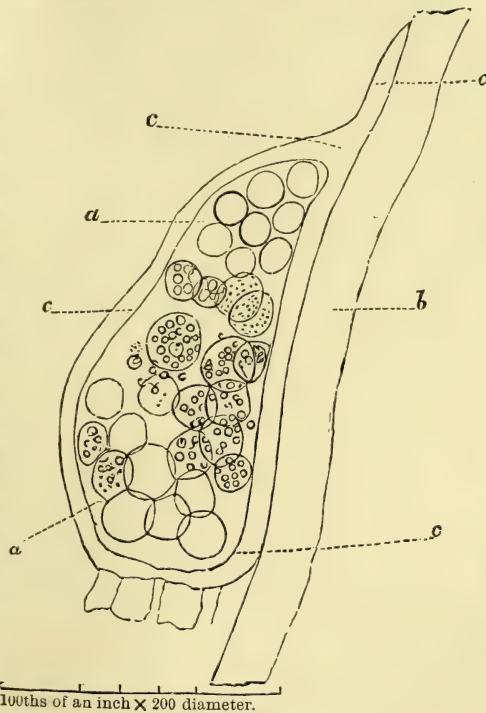


Fig. 59.†

* a. *Pediculus capitis* (male); b. Trachea and stigmata; c. Antenna (after ANDERSON).

† Nit, or Egg Capsule of the Louse, fixed to (b) a hair, by the glutinous secretion (c, c, c).

of a whitish colour, and varies from half a line to two lines in length; the body elongated and the abdominal portion broad, its margins lobulated and covered with little hairs. The thoracic portion is very narrow, and carries three legs on each side. The legs are hairy, jointed, and terminate in claws. The insect secretes itself amongst the folds of the clothing, and causes extreme itchiness of the skin where it comes to feed. Between the irritation of the insect, the *débris* of its exuviae, and the scratching of the skin by the patient, *papule* arise, the summits of which, being torn off, give rise to a *pruriginous eruption*, which may even become pustular. This eruption is met with most frequently on the neck, back, and shoulders, and round the waist—the parts most tightly embraced by the clothing, and where the clothes are most frequently gathered into folds, between which the lice are imbedded, and where they deposit their eggs, or egg capsules, which are crystalline, shining, yellowish, opaque bodies. The lice seem to multiply fastest where eruptions, such as *eczema*, prevail. A case is reported by Mr. Bryant as having occurred in Guy's Hospital, in which the whole of the body was literally covered with lice. The patient had been a governess, about thirty years of age; and the irritation was so great that excoriations and scabs were produced. On admission into hospital she was put into a warm bath, her clothes were destroyed, and every precaution taken to remove all the insects; but in two hours afterwards her body



Fig. 60.*

was again covered with lice, although she had been laid in a clean bed. She was again thoroughly washed, but the vermin re-appeared immediately. Some of the insects and their ova no doubt remained adhering to the skin, hidden amongst the scabs of the excoriations; and they are known to multiply with a rapidity proportioned to the favourable nature of the soil afforded by the morbid condition of the skin of the affected person (ANDERSON, l. c., p. 108). Bernard Valentin relates the history of a man, forty years of age, afflicted with unsupportable itching over the whole body, and having his skin covered with little tuberosities. The physician, unable to assuage the itching, made an incision into one of the small tumors, and gave exit to an enormous quantity of lice of different forms and sizes. The same operation was performed on the other tumors with a similar result (quoted by ANDERSON, p. 110). The following instance is related by Dr. Whitehead, in his work *On the Transmission from Parent to Offspring of some forms of Disease*, p. 173, and quoted by Dr. Tanner:—A farmer, forty-three years of age, strong, and of sanguine complexion, contracted syphilis in April, 1840. Seven months afterwards he suffered from secondary symptoms. At the end of 1841 he became so annoyed by the presence of lice about his person, chiefly on the trunk, that he sought again medical aid and advice. He was scrupulously clean in his habits, and had never before been troubled with these vermin. They

* *Pediculus corporis*—female (after ANDERSON).

increased in number, and produced such mental distress that fears were entertained for the integrity of his intellect. An examination of the skin showed a multitude of irritable-looking points on the front and sides of the chest, from which *nits* could be detached by lateral pressure. At this period the generation of the lice was so considerable and rapid that a flannel vest put on clean in the morning was crowded with them by the end of twenty-four hours. The usual remedies had only a temporary effect, till *iodide of potassium* and *prussic acid*, taken internally, seemed to render the system unsuitable for the further development of the vermin.

Pediculus tabescentium, or *Distemper Louse*, is of a pale yellow colour, having a rounded head and long antennæ; the thorax is large and quadrate, the abdomen large, and the segments intimately united. It is doubtfully British.

Treatment of Lousiness consists in the destruction of the insects and soothing the irritation. To accomplish the death of the parasites the following substances are efficient—namely, *carbolic acid*, *sulphur*, *mercury*, *staphisagria*, *sabadilla*, *pyrethrum*, the *essential oils* and *alcohol* (ANDERSON.)

Carbolic acid may be used as a soap (Calvert's or Macdougall's) or ointment, or as a lotion, in the following proportions:—*Crystallised carbolic acid*, two drachms; *Spirits of Rosemary*, one ounce; Rectified spirit, half an ounce; Distilled water, to make up a lotion to six ounces, with which the parts are to be sponged night and morning. *Sulphur* is used in the form of vapour baths or fumigations, or the simple or compound sulphur ointment of the Pharmacopœia. *Mercury* may be employed as simple mercurial ointment, or by fumigations with *cinnabar*, or in solution of the *bichloride*, in the proportion of two to three grains to an ounce of water, to which some alcohol has been added. *Staphisagria* is employed as an ointment in the proportion of an ounce of *staphisagria* with four ounces of lard; or an infusion of *staphisagria* may be made with vinegar. *Sabadilla* may be used in powder or as an ointment; an ounce of lard being used to incorporate a drachm of *sabadilla*.

The hair should be cut short when lice infest the head; and a lotion of the *bichloride of mercury*, or some of the ointments above named, applied at once to cause the death of the insects. The *nits* must be dissolved away by *alcohol* or *dilute acetic acid* (HEBRA). The scalp should afterwards be repeatedly washed in warm water with soap, and any eruptions treated according to their nature. Sometimes the *nits* and the *débris* of the lice are involved in the substance of these eruptions, so that care must be taken to kill any insects that may remain and be so hidden. It is also necessary either to destroy the clothes, or to expose them to a temperature of at least 150° Fahr., by steaming them, or ironing them over with a sufficiently hot iron, or to boil them. In extreme cases, such as those related above, it has been found that a mixture of *iodide of potassium* and *prussic acid* in full doses cured the diseased state of the system which favoured the development of lice in such numbers. After sixteen or eighteen doses in the case recorded by Whitehead, the cure was permanently completed.

The *Pediculus pubis* is best got rid of by rubbing a lotion of *bichloride of mercury* amongst the roots

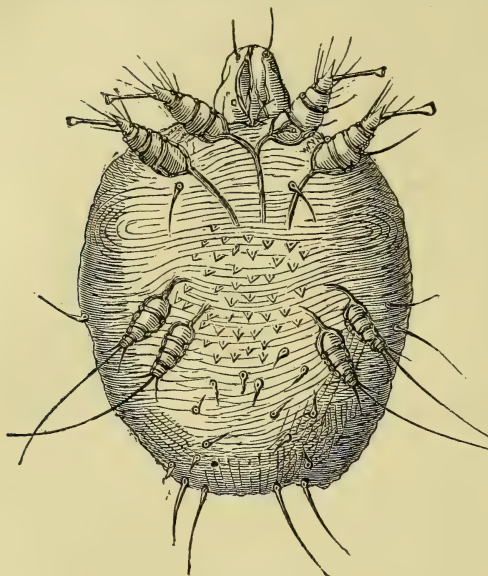


Fig. 61.*

of the hairs, taking care that it is brought in contact with every insect. The application should not only be applied to the hair of the *pubis*, but to that in the neighbourhood, such as that of the *scrotum*, *perineum*, and *anus*; and the application should be continued twice a day for a week at least. *Mercurial ointment* is equally efficacious; but care must be taken not to induce salivation: the hairs, rather than the skin, are the parts on which the lotion or ointment ought mainly to be applied.

***Sarcoptes scabiei*.**—The full-grown *itch spider*, or

Acarus is of a whitish-yellow colour, and is just visible to the naked eye. The female (Fig. 61) varies in size from $\frac{1}{7}$ to $\frac{1}{4}$ of a line in length, and from $\frac{1}{10}$ to $\frac{1}{8}$ of a line in breadth. It is of an ovoid form, broader anteriorly than posteriorly. The anterior segment carries the head and four limbs, two on each side of the head, which are set very close to it. The *head* projects considerably beyond the body, is of a rounded form, and marked by a central fissure provided with mandibles. The limbs are altogether eight in number, the four posterior limbs being placed about the middle of the under surface of the body. These limbs are of a conical form, tapering towards a point. They are each composed of several jointed segments; and the four anterior limbs are each provided with a stalked sucker. The extremity of each of the hind limbs terminates in a long curved *hair*; and several short hairs spring from beside the root of each sucker on the anterior limbs. The body is marked by numerous regularly disposed wavy lines; the dorsal surface is convex, provided with numerous little angular spines and little round tubercles, from each of which also springs a short conical spine. From each side of the body two hairs project; and four project posteriorly; so that, including those springing from the hind legs, the posterior half of the body is provided with twelve long hairs. The male *Acarus scabiei* (Fig. 62) is considerably smaller than the female; and the innermost pair of posterior limbs are provided with stalked suckers as well as the anterior limbs; while the parts corresponding to the genital organs are very distinctly marked (HEBRA, ANDERSON).

* *Acarus scabiei*—female (after Dr. T. ANDERSON).

It is now impossible to say who discovered the itch insect. Avenzoar hinted at the existence of an *insect* in the vesicles of itch; but Moufet, in his *Theatrum Insectorum*, first mentioned it in a particular manner in 1663. Hauptmann first published a figure of it, and represented it with six feet. Redi, Lorenzo, Cestoni, and Bonomo examined numbers of them, having removed them from the papules or vesicles of the skin. They discovered also the eggs of the parasite, and even observed their extrusion. Morgagni, Linnaeus, De Geer, Wichmann, and Waltz confirmed these observations; but, nevertheless, the existence of the parasite up till 1812 was still called in question. About this time, therefore, a considerable prize was offered by the Parisian Academy of Sciences for its demonstration; and M. Galès, an apothecary of the St. Louis Hospital, tempted by the reward, is said to have defrauded the Academy and gained the prize (ANDERSON). His investigations are reported to have been witnessed by many members of the Institute; but, nevertheless, he managed to conceal beneath the nail of his thumb the common cheese-mite, and having opened with a lancet the pustule of a patient affected with scabies, he dexterously produced the cheese-mite from beneath his nail, pretending to have removed it from the patient (ANDERSON). Many others attempted to find the *Acarus* in the pustules or vesicles; and the circumstance of such men as Galeoti, Chiarugi, Bielt, Lugol, and Mourouval having failed in finding the animal, occasioned fresh doubts regarding its existence. Their failures arose from having followed the cue given them by M. Galès in searching for the parasite in the papules or vesicles. Moufet had long before stated that they were not to be found in the pustules, but by their sides. Casal made nearly a similar observation; and Dr. Adams remarks that they are not found in the vesicle, but in a reddish line going off from one of its sides, and in the reddish firm elevation at the termination of this line, a little distance from the vesicle. Seventeen years after Galès' demonstrations the Academy discovered, through Raspail, that they had been defrauded; and in 1834 M. Renucci, a medical student from Corsica, showed the physicians of Paris the mode of discovering the *Acarus*, which is the same method as that which had been formerly mentioned by Dr. Adams. Since that time, as Rayer remarks, the existence of the *A. scabiei* has been placed beyond a doubt; and after the demonstrations of MM. Lemery, Gras, and Renucci (all of whom showed him the method of detecting it), Rayer has been able to extract it several times himself. Raspail has given an excellent description and figure of the parasite. M. Albin Gras enters into researches as to the share it has in producing the eruption, and he instituted experiments on the *Acarus* itself, which have an important bearing on the treatment of *scabies*. The habits and natural history of the parasite have been carefully investigated by Hebra;

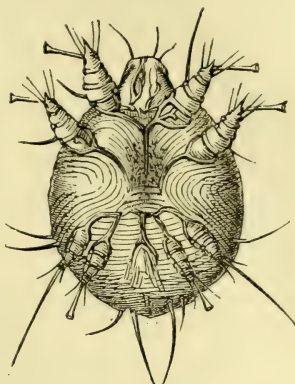


Fig. 62.*

* *Acarus scabiei*—male (after Dr. T. ANDERSON).

and Dr. Anderson has given an excellent account of these investigations, from which this description of the parasite and the disease is mainly taken. The discovery of the male *Acarus* is claimed by several observers. According to Hebra, it was first discovered by Danielssen and Boeck in Norwegian scabies. According to Dr. Anderson, M. Bourgogne, the maker of microscopic preparations in Paris, claims to be the discoverer. According to Devergie, the honour is due to M. Lanquetin, a pupil of St. Louis. It is the female only which burrows in the epidermis of the human skin. All the male *Acari* go free on the surface of the epidermis, where sexual intercourse between male and female *Acari* is said to take place. When an impregnated female is placed on the surface of the skin it seeks a suitable spot to penetrate, and raising its head at right angles to the surface, it digs, burrows, or eats its way between the scales into the deeper layers of the epidermis, where it imbeds itself, derives nourishment, and goes through the process of parturition till she dies. Having found a suitable place, an egg is laid, and each day another, the animal penetrating a little farther each time, leaving its deposited eggs to occupy the space previously inhabited by itself. The direction of the canal is oblique, the portion first formed being, of course, nearest the surface. As the old epidermis is thrown off, new layers of cuticle being formed from the deeper strata, the first-laid eggs are gradually thrust upwards to the surface, where they are finally extruded, while the recently deposited ova remain in the canal close to the parent female, whose instincts lead her to make the canal in such a way that her eggs reach the surface about the time the young ones are ready to come out of the shell. The newly-hatched *Acari* (males and females) having arrived at the surface, crawl about the skin, and enter into sexual congress. The females in due time become impregnated, and, like their parent, repeat the process of burrowing and parturition just described. The length of time which intervenes between the laying and hatching of an egg is said to be *fourteen* days; and as the *Acarus* is found to lay one egg daily, there are rarely more than fourteen eggs in one canal at a time. The canals (cuniculi) which the female *Acari* burrow have a serpentine shape, and vary from half a line to three lines long. Hebra, and Dr. Reid, of Glasgow, have seen them three or four inches in length; and Hebra mentions that they sometimes completely surround the wrist like a bracelet. These canals have generally a whitish dotted appearance, the dots corresponding to the ova in the canals; and at the extremity of each canal is a little whitish elevation, which corresponds to the site of the parturient or defunct female *Acarus*. This whitish elevation is generally about a quarter of a line distant from the papule or vesicle; and the skin should be cleaned before endeavouring to detect it. After the death of the mother *Acarus*, the epidermis which covered in the canal gives way, as the cuticle grows and desquamates; there is then left at first a depression, or open rugged furrow, bounded on each side by a ragged edge of epidermis; and as these edges become dirty, the remains of the canal present a dirty ragged line. Besides the *Acarus* and its eggs, numerous little oval or rounded blackish spots are seen in these canals, which are supposed to be the excreta of the parasites; and after the female has once entered its canal it is unable to recede, owing to the spines on its body, which project backwards. It therefore dies in the canal when parturition is finished.

The eggs of the *Acarus* vary much in size, according to their age and development; and just before the larva has burst its shell the egg is almost as large as the male *Acarus*. In the earliest stages the egg is very small, and filled with a granular-like matter (Fig. 63, *a*).

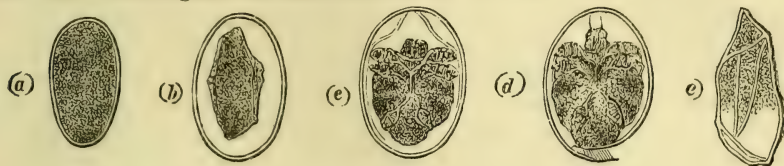


Fig. 63.*

It grows in the canal; and as it increases in size its contents seem to shrink and recede from the shell, and to have a distinct enveloping membrane. The bright yellow colour of the embryo contrasts strongly with the clear, almost colourless, walls of the egg-shell (Fig. 63, *b*, *c*, *d*). The head and legs of the embryo soon become distinctly visible, and at last the whole form of the *Acarus* (Fig. 63, *c*, *d*). Finally, the shell bursts, and the young *Acarus* escapes, leaving its shrivelled envelope (*e*) behind. The larva or young (Fig. 64) differs from the full-grown insect in the possession of two hind limbs only, in place of four. By and by, however, it casts its coat, and then appears with eight legs; and sometimes even the full-grown *Acarus*, with its eight legs, may be seen inside of its old six-legged skin, and thus renders the history of its development complete.



Fig. 64.†

Demodex folliculorum is found inhabiting the sebaceous sacs and hair follicles of the human skin (Fig. 65). By some naturalists this parasite is referred to the *Acaridae*, but by recent investigations it is considered to be more nearly related to the *Rotifera*, its parasitic habits causing it to resemble some of the numerous forms of the *Crustacea*. It is not known to cause disease or any specific lesion.

Pulex penetrans, *Chigoe* or *Jigger*, is a species of flea, and one of the greatest pests in tropical countries, and especially in South America and the West Indies. The female burrows in the skin of the feet till quite out of sight, preferring the bare spot between the toe and the nail, and there remains to rear a numerous progeny. It

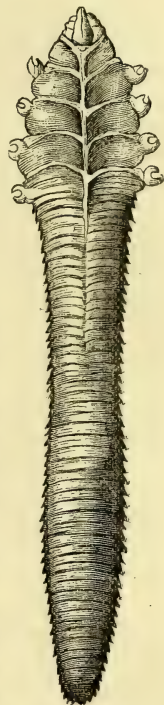


Fig. 65.‡

* (*a*.) Egg in the first stage; (*b*.) In the second stage, their granular contents being yellow; (*c*.) Egg in the third stage, the form of the *Acarus* becoming apparent; (*d*.) The egg in the fourth stage, the *Acarus* having broken the shell; (*e*.) Egg-shell after the escape of the *Acarus* (after Dr. T. ANDERSON).

† Larva or young *Acarus scabiei*—having only two hind legs (after Dr. T. ANDERSON).

‡ *Demodex folliculorum* (after Dr. ANDERSON).

sets about its work so quietly, and insinuates itself so gently, that the only perceptible sensation is a slight, but not unpleasant, irritation (Rev. J. G. WOOD). When the female has completely penetrated under the skin, the vast number of ova begin to undergo development, and so enlarge the parent-abdomen of the *Chigoe* to about the size and shape of a pea. Even when so large, the only external sign is the swelling, with livid bluish colour surrounding it, and up to this time it may escape observation. Those, however, who live in *Chigoe*-infested districts are careful to examine their feet every day, and dislodge the insect at an early period with the point of a needle. It is very difficult to extract them when fully swollen; for if the distended abdomen burst, and a single egg is suffered to remain, the larva will be hatched in the wound, severe inflammation results, and a painful, festering, troublesome sore remains. If such a consequence supervene, oil of turpentine dropped into the wound is the most efficient, though painful remedy, but preferable to the long-continued and future painful sores. The negroes use red pepper, which they rub well into the hollow after extracting the rest of the *Chigoe* from the feet of children; and the children are so afraid of being peppered that they soon learn to apply early to have the *Chigoe* removed before it has completely penetrated the skin.

III.—ENTOPHYTA AND EPIPHYTA.

These mainly consist of *fungi*; but as *fungi* are often confounded with *algæ* and *confervæ* the following three definitions may be given:—

Definition I.—*Algæ* are represented by sea-weeds and multifarious green vegetable forms of simple cell-structure. They are met with in all streams, ditches, ponds, or even in the smallest accumulation of water standing for any length of time in the open air. They are common also on damp walls, or on the ground in all permanently damp places. They are represented by the following groups:—(a.) *Floridæ*; (b.) *Fucoidæ*; (c.) *Confervoidæ*.

The following were once classed as *algæ*, but are now regarded as the *mycelia* of various *fungi*, and not independent organisms, namely:—*Cryptococcæ*; *Leptomitæ*; and *Phæonemecæ*.

Definition II.—*Confervæ*, or *Confervoid Algæ*, are chiefly marine, and consist of plants having unbranched filaments, composed of cylindrical cells, the length and diameter of which have a very variable relation in different species. They are produced from *Zoospores*, having two or four cilia.

Definition III.—*Fungi* are a class of cellular flowerless plants, growing in or upon damp mould; in or upon the wood and herbaceous parts of living or dead plants; upon living or decaying animal substances, or in solutions containing organic matter. They do not appear capable of assimilating inorganic food, and are in this respect distinguished from healthy specimens of all other plants, as also by the total absence of colour, depending on the presence of *chlorophyl* or of its red modifications. They are allied by certain forms with the *algæ* and with the *lichens*; but are distinguished from all outwardly similar forms of *algæ* by the spore-bearing fruit always being elevated into the air, when mature, although the *thallus* or *mycelium* may be aquatic. The *lichens* possess green conidial

cells in the thallus, which fungi have not. They are entirely aerial *encrusting* plants, while the fungi have their vegetative structures immersed in the medium in which they grow.

As influencing disease, *fungi* have certain peculiarities:—(1.) They all absorb oxygen, and give off carbonic acid. They have great powers of decomposing salts of metals, as well as of destroying organic substances. (2.) All of them contain a greater or less proportion of poisonous alkali entering into their composition. (3.) They take part in yeast formations producing diarrhoea (PAREIRA'S *Materia Medica*, p. 588).

The Structural Elements of Fungi are—(1.) *Mycelium* (thallus fibrils), or vegetative structure, consisting of threads of various shapes and sizes, composing a mass of exceedingly delicate, sometimes jointed and branched, colourless, interlacing filaments. Some are large convoluted tubes, sometimes septate, and they form together a cottony or felt-like mass when growing in or upon any permeable structure; or cloudy flocks when growing in liquids undergoing fermentation or putrefaction. The contents of the tubes and threads are granules or cells.

(2.) *Reproductive structures or fruit* of fungi differ extremely in appearance in the various tribes. A filament rises up from the general body of the mycelium, and at its terminal end is borne the fruit, composed of a modification of one or more terminal cells. Thus the fruit may either be an enlarged, terminal, solitary cell; or a shortly-jointed tube; or a clustering of minute cells seated upon a receptacle; or a radiate arrangement of minute cells; or a large sac filled with minute cells.

Reproductive cells or fruit may be of at least three different forms in *coniomycetous* fungi.

(a.) *Conidia* (naked spores), rising direct from the mycelium, or from the *stroma* found on the mycelium. The stroma consists of an indefinite number of minute cells, and is the early condition or nuclear form of the fully developed fungus. It accompanies all fungi in a state of active growth. The nuclear cells sometimes exhibit a molecular movement of their contents.

(b.) *Conceptacles*, or hollow cells (organised immediately on the mycelium), the inner wall of which is lined by filaments (stylospores) terminating in minute cells or spores; or by *spermatia*, which are much smaller than the stylospores, and of a linear form (ultimately confused into a gelatinous mass). The functions of these spermatia are unknown, but are supposed to be of a fertilising nature.

(c.) Another form of spore is found inclosed in *asci*.

Structure of the Spores of Fungi.—They are round or oval, solitary or arranged in rows, single or many-piled, or in groups of various sizes. They often show a dark spot, or a nucleus or nucleolus, in their interior. The spore has an outer coat composed of cellulose, and an inner coat or utricle inclosing a liquid containing floating granules, which are coloured blue by iodine. Spores may be confounded with many other cells, such as fat, blood-disks, corpuscles of various fluids, nuclei of epithelial cells, pus, earthy particles. They are unaffected by *ether*, *chloroform*, *alcohol*,—all of which dissolve fatty cells, and render epithelium transparent. *Ammonia* renders the spores a little more colourless. It also dissolves pus and the secretions of eruptive diseases (which often contain granules

and cells very like spores), converting them into a gelatinous mass. A hot solution of *potash* with *alcohol* dissolves impetiginous crusts, fat, pus, hair, and epithelium. Earthy particles, in granular, duplicate, or quaternate forms of carbonate and phosphate of lime, are destroyed by *acids*.

The greatest care must be taken to distinguish fat-cells from spores, and diffused molecular fat from sporules, or nuclear forms of fungi. Beware also of taking minute fibres from handkerchiefs or towels for mycelia. The appearances which may be assumed by fatty concretions and myeline have been well shown by Drs. Lewis and Cunningham in their investigations regarding the fungus disease of India. They show that under the influence of various reagents origin may be given to an abundance of that ill-defined substance which Virchow has termed *myeline*, which is especially prone to occur where portions of fatty matter, freshly removed from alcoholic preparations, are subjected to the action of *liquor potassæ*. Multifarious and highly complex forms of tubes, with filaments, globules, and cysts, appear to become developed—appear to shoot out and to grow from mere globules and aggregations of fat—and so from their appearance they are liable to be mistaken for fungoid growths. Hence the necessity of great caution in the interpretation of such phenomena. The use of reagents is absolutely necessary in determining fungi, to escape from constant sources of fallacy, and the following are the most useful reagents for determining their character:—

Iodine (solution for granules of spores), *Ether*, *Chloroform*, *Spirits of Wine* (absolute alcohol), *Ammonia*, *Potash*, *Acids* (to dissolve earthy carbonates), *Glycerine*, *Sulphuric Acid*.

Although much has been written on the influence of *fungi* in the production or aggravation of disease in the animal as well as in the vegetable kingdom, and also as to their influence in the transmission, propagation, or portability of disease, yet the subject is involved in much confusion; because, in a few instances only have the persons recording their experience been sufficiently acquainted with the botanical nature of *fungi*, on the one hand, and with the nature of diseases, on the other, so as to give anything like a complete history of the cases which have fallen under their observation. Mere *mycelia* have been described as perfect plants, and mistakes have been made in important points of structure. Productions of an undoubted fungic nature have been referred to *algæ*, although agreeing with them neither in habit nor in physiology. The commonest moulds have received new names; and several conditions of the same species have been recorded as productions differing in their mechanism and physiological laws (BERKELEY). *Fungi* are the most numerous of all plants in regard to genera and species, and their growth may be associated with most extensive injury to animal and vegetable life; and as they are now proved to be capable of propagation by implantation from animals and vegetables to man, they demand from the physician a most careful study. The epiphytic diseases of plants as well as animals have hitherto been almost neglected by the pathologist; yet how do we know that the *blights* of plants, or the causes of them, are not communicable to animals and to man? It is known how intimately the diseases of man and animals are related with the occurrence of famines and the prevalence of unsound or unwholesome food; and of famines with the diseases of vegetable and

animal life, as much as with the destruction and loss of food. The black sporules of *Ustilago hypodytes*, which cause disease in grasses in France (the grass smut), and those of *Ustilago vittata*, which cause similar disease in the grasses of India, are known to produce most injurious effects upon the haymakers in the former country (LEVEILLE). In places favourable to the multiplication of *fungi* they often commit extensive ravages. Among the silk-worms in the silk manufactories of Italy, *fungi* are the cause of more extensive destruction of such animals than we have any correct idea of. Under the names of "mildew," "blight," "smut," "brand," and the like, *fungi* are described as committing extensive damage among living plants, as the farmer and orchardist know too well. It, therefore, behoves the pathologist to study carefully the nature of those epiphytic diseases in animals and vegetables, as well as in man, whenever he has an opportunity, and especially in India. It is not in all cases easy to determine whether they are the cause of morbid states, or whether, as some think more likely, the diseased tissue has merely afforded a suitable nidus for their development, either as a result of accident or of disease, alike in animals and in plants. It is certain that wherever the normal chemical processes of nutrition are impaired, and the incessant changes between solids and fluids are less active;—then, if the part can furnish a proper soil, the cryptogamic parasites will appear. The soil they select is for the most part composed of epithelium or cuticle, acid mucus or exudation—as a fact, it is unhealthy; and "it has yet to be shown that the living matter of the tissues of any animal, so long as it retains its vitality undiminished, is liable to succumb to the attacks of a fungus." (LEWIS and CUNNINGHAM, l.c.) Acidity, however, though favourable for their growth, is not indispensable; since some of the vegetable parasites grow upon alkaline or neutral ground, as on the ulcerations of the trachea, or in fluid in the ventricles of the brain. Certain atmospheric conditions seem favourable to the occurrence of such vegetable parasites. For example, *Tinea tonsurans* may be quite absent for years in places, such as workhouses, where it commonly exists, and then for several months every second or third child in the place gets the disease. It has been observed that some of these parasitic diseases can be propagated by transference of the plant, as in various forms of *tinea*, and that the disease can be cured with the greatest readiness by the chemical agents which are most destructive to vegetable life. Parasites of the nature of *fungi*, associated with the skin diseases described as "ringworm," have been shown to be capable of transmission from animals to man. It has been proved by Devergie that *T. tonsdens*, for example, is transmissible by contagion from horses and oxen to man, and that the parasite has given rise either to the same form of *tinea* disease or to another; but he ascribes both species of parasitic disease to the growth of one and the same parasite—namely, the *Trichophyton*. Von Bärensprung, of Berlin, bears similar evidence. He rubbed on his forearm some of the scales of *tinea* from one of the lower animals, containing abundance of the spores and mycelium of the fungus *Trichophyton*. No effect was produced for several days; but after a longer interval considerable itching called his attention to the part (irritation having set up a diseased state), which he found occupied by a well-marked spot of *T. circinatus*, about the size of a sixpence. In

three weeks the patch increased to the extent of a crown-piece (*Brit. and For. Med-Chir. Review*, July, 1857, p. 263). Instances have been noticed of grooms being attacked by *T. circinatus* and *sycosis* after grooming horses affected by *T. tonsurans*. "A dragoon came to the Dispensary of the St. Louis Hospital affected with *T. circinatus* on the front of the right forearm. He stated that five or six of his comrades had contracted this affection as well as himself from grooming diseased horses. A visit to the barracks showed three horses with round patches absolutely identical with *T. tonsurans*. These were situated on the withers, shoulders, back, and belly. The hairs in the centre of each patch were broken off close to the skin, and there was a whitish, squamous, crust-like production, which was traversed by the hairs. The presence of sporules was detected by the microscope. The dragoon who showed the horses showed also his daughter, a girl of eight or ten years of age, the side of whose nose exhibited a patch of *T. circinatus*" (BAZIN, quoted by Dr. ANDERSON, *On the Parasitic Affections of the Skin*, p. 51). With regard to the transmission of *favus* from the lower animals, I am informed by Dr. Anderson that, in the dispensary practice of Glasgow, the physicians often find it traceable to contagion from mice, cats, and dogs similarly affected; but that mice especially are the animals which seem to be the ultimate source of the disease.

On the other hand, it is shown that animals may contract parasitic diseases of the skin from human beings similarly diseased. Dr Tilbury Fox mentions an instance of a white cat, a great pet with the children of a family of nine, which contracted the *mange* and *T. tarsi* from *T. tonsurans* affecting five of the children. The fungus of the *mange* in the cat is the same fungus as that of *Tinea* in man—namely, the *Trichophyton*.

The principal vegetable parasites associated in man with special morbid states are enumerated at p. 150, *ante*; but it would have been better if these *fungi* had been described, in the first instance, without specific names. The fact of specific names having been assigned to each of them has drawn attention from the important part which these *fungi* perform in the work of decomposition. They may be forced to fructify by placing them in a globule of water surrounded by air, and placing them in a closed cell; and until the fungus has thus come to maturity, it is worse than useless to give them names—so many different forms in an undeveloped state being all capable of reference to one common mould (BERKELEY). The plants forming on mucous membranes, or in the contents of cavities lined by mucous or serous membrane, are in most cases secondary formations only, and their exact pathological significance is yet undetermined. The pathognomonic sign of all fungic parasitic lesions of the surface (cutaneous or mucous) in man and animals is the infiltration or destruction of hairs (*tinea*) and epithelial textures (*muguet*, *thrush*, *oidium*) by the *sporules* of a fungus, and which, by union or by growth, form elongated branches, or *mycelium*.* The diagnosis of such *fungi* on the skin, hair, or epithelium, can only be effected by a careful and skilful microscopic examination; and it is always absolutely necessary to use *liquor potassæ* and other re-agents, mentioned at 238, *ante*, in the examination of all *tonsurant* appearances of the hair, of all *idiopathic* bald

* These *fungi* have sometimes been popularly but erroneously termed *confervæ*. The *confervæ*, for the most part, grow in salt or fresh water.

patches, and of all brown or yellow-coloured scurfs, for sporules are frequently detected which had escaped observation before *liquor potassæ* had been used (BAZIN, FOX, ANDERSON). Sufficient time must also be allowed for the parts to become transparent under the action of this reagent. The parasitic lesions of the skin are, as a rule, unsymmetrical, and hence they differ materially in this respect from syphilitic eruptions. They differ also no less essentially from the eruptive diseases of the skin. An eruption is no necessary part of these parasitic lesions; but, from the irritation established in the true skin, eruptions of various kinds may occur. Eruption thus often precedes the detection of a fungus, and, as a rule, very often follows its existence; and they who dispense with the microscope in the diagnosis of skin affections cannot avoid confounding severe eruptive with parasitic lesions, because they disregard the pathognomonic evidence of such lesions already indicated (Dr. T. FOX). The term *herpes*, therefore, as applied to these parasitic affections, is an objectionable term; because it has been already used to indicate a vesicular eruption, namely, *herpes zoster*, which invariably exhibits large typical vesicles.

There seems to be a peculiar (unhealthy) condition of nutrition best fitted for parasitic growths of a vegetable nature, just as some constitutions and states of the system are best suited for the propagation and development of entozoa; but the exact circumstances which predispose to the growth of these *entophytic fungi* upon the human body are not better known than those which predispose the body to receive and develop certain morbid poisons of a specific kind, known to multiply during the course of the disease, and to throw off material capable of propagating and spreading the same kind of disease. With regard to the parasitic diseases of the skin and hair, a failure of the vital powers to carry on the healthy processes of life seems ordinarily to be one of the inviting causes of such a development of true *fungi* as would constitute a disease. A special nidus or soil is necessary, just as a predisposition is necessary in the case of the spread of specific, contagious, or infectious diseases; yet care must be taken in both instances not to confound the co-operating cause with the special or peculiar poison or germ. Both Robin and Bazin recognise a condition of the hairs (dependent, perhaps, on constitutional causes) which appears to be essential for the growth of the plant; for sometimes the disease disappears spontaneously, and the fungus dies without its being destroyed by any specific treatment.

Varied opinions still prevail regarding the nature of vegetable parasitic affections, and especially as to whether several distinct fungic growths are concerned in the production of the parasitic affections of the skin; or whether those affections are due to one and the same parasite. It has been shown that all the forms of ringworm are due to the same parasite,—namely, the *Trichophyton* (BAZIN, ANDERSON); but there are not a few who hold that there is only one parasite productive of *all* the vegetable parasitic affections of the skin, amongst whom are the names of Hebra, Tilbury Fox, Lowe, and Jabez Hogg. There are numerous facts which seem to them to justify the belief that there exists but one essential fungus, whose sporules find a soil for development and growth upon the surface, or even within more secluded portions of the human body; and that

varieties in the growth of that fungus are due to differences in the constitution of the individual, to the moisture, exudation, soil, or temperature under which the development of the fungus takes place. The exact nature of these differences is not understood; but the production of irritant acids and gases are constant accompaniments of the growth of such parasites, by the chemical action of the vegetable cell; for it does not undergo development without exciting a chemical decomposition in the pabulum on which it feeds; and the different stages in its growth give rise to alcoholic, acid, and putrefactive fermentation. Of the latter there is ample evidence in many of these parasitic skin diseases, and especially in favus, the odour of which closely resembles that of some *methylamine* compound (LOWE). Thus their irritant action very soon may establish an eruption. But the ratio of eruption to parasite is not constant; for an amount of fungus which will simply produce death of hair in one person or part of the body, may in another produce irritation, eruption, or violent inflammation. These different results may be due to two causes, —(1.) Constitutional peculiarity in different individuals; (2.) Peculiarity of structure of a part as regards density, heat, moisture, and chemical and anatomical composition of the part. On the other hand, Dr. Anderson contends that the fungi met with in *favus*, *tinea tonsurans*, and *pityriasis versicolor*, are each different; in other words, that the *Achorion Schönleini*, the *Trichophyton*, and the *Microsporon furfur*, are not identical. The following is a summary of the proof he adduces in favour of their non-identity:—

“(1.) In all cases of successful inoculation with *Achorion*, *Trichophyton*, and *Microsporon furfur*, the same parasitic disease has been produced as that from which the parasite was taken.

“(2.) Of the innumerable cases occurring in the human subject illustrative of the contagious nature of favus, *tinea tonsurans*, and *pityriasis versicolor*, which have been recorded, there is no authentic case in which one of these diseases gave rise to one of the others.

“(3.) The difference in the appearance of favus, *tinea tonsurans*, and *pityriasis versicolor*, when fully developed, is so very striking, as to lead to the belief that they are produced by separate parasites.

“(4.) There is no authentic instance on record of the transition of one of these diseases into one of the others.

“(5.) The difference in the appearance of the *achorion*, *trichophyton*, and *microsporon furfur*, is sufficiently striking to enable the observer in many cases to form a correct diagnosis from the microscopic examination alone.

“(6.) Of the numerous instances on record of the transmission of favus and *tinea tonsurans* from the lower animals, by contagion or inoculation, favus has always given rise to favus, and *tinea tonsurans* to *tinea tonsurans*.”

It is not to be hastily concluded that a disease is non-parasitic because a fungus has not been demonstrated in the part where it was expected to exist. The minuteness of the sporules, and the care required in the investigation, sufficiently explain the various opinions which have prevailed on the nature of these parasitic lesions (LOWE). To appreciate the ravages which may be produced by such minute cells, the reader may be reminded of the immense force which the growth of such cells is capable

of producing through continuous and gradual development; for, being of so minute a form, and its agency so apparently invisible, its intrinsic power is apt to be underrated. An *Agaric* growing under a stone of more than a hundred pounds' weight will eventually raise it from its bed to the height of several inches; and the cells of that *Agaric* are not widely different from the *sporules* of a fungus. Again, the *mycelium* of a fungus (probably identical with that found in skin diseases) has been known to raise a cask of wine, the fungus feeding on the wine as it leaked from the cask (HARVEY, quoted by LOWE, in *Lancet* of August 13, 1859).

The variations of form in these different vegetable parasites, associated with the skin diseases to be afterwards described, are obviously so slight that they seem insufficient to warrant different species being made out of them. They may all be initial or undeveloped forms, referable to the *Aspergillus glaucus* or *Penicillium*; the initial forms of both of these being isomorphous (LOWE); and both of them are also equally indifferent about the matrix where they grow, as long as the conditions for their growth are fulfilled (BERKELEY). Peculiarities have also been observed in the growth of the fungus, which may be due to the stage of development and conditions of growth at the time of examination (*Path. Society Trans.*, Vol. VII., p. 395). The same *fungi* during their growth assume very different forms and appearances. It thus happens that the same species has not only been described under different specific names, but even referred to different genera. Fries has traced no fewer than eight genera to mere degenerations or imperfect states of one particular fungus (*Thelophora sulphurea*); and Nees von Esenbeck states that the same fungoid matter which develops a certain fungus in winter (the *Sclerotium mycetospora*) will develop another fungus in summer (the *Agaricus volvaceus*). Professor Henslow showed that some of the supposed species of *Uredo* are forms of *Puccinia*, *Aegma*, and the like.

Thus the *Dimorphism of fungi* presents a difficulty at the outset in connection with the identification and interpretation of the structures seen in textures which are believed to be so infested. In many fungi of the *coniomycetous* kinds (where spores are the principal part) the same fungus appears under two or more distinct forms—so distinct, that some have been regarded as different species belonging to different genera far removed from each other, and bearing different names. The golden-coloured turmeric-like powder on the under surface of the rose leaves has its *mycelium* penetrating the tissue of the leaves, and the powder on the surface is composed of the spherical spores. The plant fungus was believed to be here complete, and has had the name of *Uredo rosæ* given to it. But later in the season there are produced on the same mycelium certain dark-brown spots, which have been found to be aggregations of complex fruits of the same plant. These brown spores are borne on long stalks, and are septate or divided by transverse partitions into a complex fruit, to which the name of *Puccinia rosæ* has been given. Thus *Uredo rosæ* (the yellow spores), and afterwards the *Puccinia rosæ* (the brown fruit), were believed to be distinct and different fungi. Now they are known to be different forms of fruit on the same plant.

Multiplicity of form is thus characteristic of the fructification of fungi. These forms are one or other of the following:—

(1.) *Naked spores*, or "*conidia*" (of which there are at least four forms), growing on the external cells of stroma, and reproducing only torula-like forms or *promycelium*.

(2.) *Naked spores*, or *conidia*, becoming sacs or *pycnidia*, and containing true minute spores (sporiferous), sometimes septate or bead-like, or monili-form strings, associated with true spores, and named *stylospores*.

(3.) *Spermatia*, or minute bodies, produced in cysts or spermogones, and not known to germinate.

(4.) *Conceptacles*, containing "*sporidia*" in hyaline sacs. These "*sporidia*" are secondary sporules, and are thus produced, within *asci* or *sporangia*, on the germinating threads from the true spores.

In such fungi the *mycelium*, or root-part, consists of fine thin filaments, spreading through all parts of the plant infested by the parasite; and the distinctions are taken from the characters and forms of the true spores.

The artificial cultivation of *fungi* has lately attracted attention, and it has for its object to distinguish the species of fungus under observation. The identification of *fructification* and *fruit* is necessary to distinguish the species; and the only safe method is,—*First*, To identify the germ to be cultivated (the individual "*micrococcus*," or larger globule, or spore). *Second*, To take a specimen of "the micrococci," or one or more globules or spores, and isolate them in a closed cell, containing suitable food, to germinate in; and with a pellicle of air surrounding the food and the germs, into which, as they grow, they may throw out their aërial forms of fruit or other reproductive parts by which they may be identified. My friend, Dr. Maddox, recommends the following solution for cultivating microscopic fungi (on glass slides):—"*Dextrine, phosphate of soda and ammonia*, microcosmic salt, of each *two grains*; grape sugar, sixteen grains; saturated solution of acetate of potash, twelve drops; distilled water, one ounce, placed in an ounce-and-half phial, set in a saucepan, with sufficient water to come to the shoulder, and boiled for half an hour or longer. Surround the bottle very loosely with bits of rag or hay. If wished to increase the temperature, add salt to the water, or use a dense fluid, as oil. When cold, pour off into clean *zij* stoppered bottles. I find most of the fungi grow well, keeping their colour in this fluid. Placing the growing slide in a porous (battery) cell, and setting this in a vessel with an inch depth of water, keeps the object moist. This cell can be opened or covered from the light." (*Monthly Journal of the Royal Microscopical Society*, June, 1870.) This is the only way to determine what originates from such bodies as are termed "*micrococci*." *Third*, To take care that the *menstruum* contains no other germs than those to be cultivated.

The growth of moulds on moist substances ought first to be made a subject for practical study by beginners. The pastes of wheat or of rice flour, where such moulds grow, should be examined at different depths, when it will be seen that numberless different modifications of form are assumed by the same fungus in different parts of the matrix. The general unsatisfactory character of cultivation-experiments are mainly due to two elements:—(1.) The difficulty of the work, and the loose mode of identifying what has been called "*micrococci*," or even the larger "*yeast-like cells*." (2.) The uncertain methods of cultivation of these,

and their uncertain identification with known forms of fungi. When a spore is taken for artificial cultivation, it is almost sure to carry with it germs of *Bacteria schizomycetes*, or other germs, as these exist in great abundance wherever *fungi* are, and they rapidly develop into an immense number of individuals.

Spores of certain fungi will endure the moist heat of boiling water without losing powers of germinating; their behaviour is similar with regard to the power of resisting frost, so that their endurance of high and low temperatures is very remarkable (BERKELEY's *Fungology*, p. 32). Hence the very indestructible characters of spores. The smallness of the reproductive cells or germs of fungi, and their facility of distribution, are important to be remembered in connection with their relation to disease-propagation. Some species are constantly recurring in the same places; but there is nothing like chance about their characters and growth. So rapid are they in development, that their appearance seems to favour the idea that they cannot have grown from seeds, germs, or spores. But notwithstanding the recent revival of the question of spontaneous generation, and of Professor Bastian's and Huxley's results, the belief in such generation is surely exploded,—all the more surely after the recent repetition of the experiments by Professor Frankland, of St. Bartholomew's Hospital, and of Dr. Hartley, of King's College. The most careful experiments show that without pre-existent germs no organised beings are ever produced from solutions containing material fit to nourish animal or vegetable life. Where experiments are not careful, and proper precautions are not taken to exclude germs, they grow and will exist in myriads. Observations on the growth of germs must be made very often, in some cases hourly. Only very little material should be employed at a time (a single germ only, if possible), in order that the field of observation may be well known and clear. The object is to *see* actually that a given growth proceeds from a given origin; and not to conclude that when two or more forms exist in the same preparation, they necessarily have a developmental relation to each other, or a common origin, even although, at the beginning of the experiment, there appears to be only one kind of element present. Mere co-existence, without organic connection, affords no grounds for such a conclusion.

The apparatus required for culture-experiments must be of such a kind as will afford—(1.) Means of protection from dust; (2.) Means of applying heat; (3.) Means of growing in the dark; (4.) Means of frequent observation.

An account of the parasitic diseases of the skin will be found under “DISEASES OF THE CUTANEOUS SYSTEM.”

CALCULUS AND CONCRETION.

LATIN EQ., *Calculus et Concreta*; FRENCH EQ., *Calcul et Concretion*; GERMAN EQ., *Stein und Concretion*; ITALIAN EQ., *Calcolo e Concrezione*.

Definition.—(a.) *Calculus* is a formation brought about by the deposition of all or of certain components of a fluid in which they are naturally held in

solution or suspension; (b.) Concretion is a formation (1.) arising out of a liberation of phosphates and carbonates of lime, or other salts, from their normal combinations, so as to encrust or penetrate parts and sever minute textures (ROKITANSKY); or (2.) a formation arising from stagnation of a secretion, when mineral and protein compounds alike become solid.

Pathology.—Calculi are especially prone to form in secreted fluids, and either consist purely of specific ingredients proper to the secretion, or these ingredients blended with other elements. Numerous causes contribute to their separation; for example,—(a.) Extreme concentration of the fluid from loss of water; (b.) Inspissation and exsiccation of secreted and exuded matter, such as *mucus, ear-wax, smegma preputii, bile, pus*; (c.) Chemical conversion of the fluid, as when the free acid of normal urine retains the phosphatic earths in solution, and when such urine is rendered alkaline, as by the presence of mucus, or of pus, or of exudation; or by conversion of urea into carbonate of ammonia, the phosphatic earths becoming precipitated. If lithates present in the urine become decomposed by excess of acid in the urine, uric acid, being less soluble, is thrown down as concretions or minute calculi. The soluble phosphate of magnesia also becomes a concrete precipitate the moment it enters into combination with ammonia, as *ammonio-phosphate of magnesia*.

The principal and best-known elementary substances which, either in their purity or in combination with others, compose the more bulky calculi-concretions or concrements, are as follow:—

1. *Concretions of Protein Substances* emerge from their solutions as a structureless or clod-like lump, or mass, in various degrees of coagulation, or as elementary granules, represented under the highest magnifying powers by a concrete dot or point. Whatever undergoes spontaneously rapid and firm coagulation is described as *fibrine*; that which coagulates more slowly and less perfectly, under the influence of heat, is known as *albumen*. The protein deposits are insoluble in *ether* and *mineral acids*. *Acetic acid* renders them translucent, and ultimately dissolves them. *Caustic potash* and *fuming hydrochloric acid* slowly dissolve them, the latter with a lilac tint. A watery solution of iodine colours them yellow.

2. *Concretions of Fats* occur in the elementary form of dropsy, or as amorphous solid particles, granules, or crystals. There are different kinds of fats, recognisable by their forms and chemical relations, namely:—

(a.) *Elain* or *olein*, occurring in variously-sized drops, either free or within cells. The fat is usually set free out of emulsion-like compounds; and as *elaic acid*, out of saponaceous compounds; or out of combination with other fats, as in exudations or in medullary cancers. The drops resist the action of water and of acids, but dissolve on being boiled with potash, and still more readily in ether or heated alcohol.

(b.) *Margarine* and *margaric acid* occur as microscopic, needle-shaped crystals, generally aggregated so densely in stellate groups or bundles as to appear as round, almost black, spherical masses (Fig. 66). *Margarine* emerges in these shapes from its solution in *elain* after cooling either within flat-cells (Fig. 67) or free. Crystals of *margaric acid*, soluble in concentrated heated alcohol, are developed out of the *margarine* of the fat by free acid, and are usually products of decomposition. They are found

in gangrene, and are apt to take up colouring matter. *Margaric acid* (Fig. 68) crystallises in minute tufts, composed of very small and much-curved crystals.



Fig. 66.*

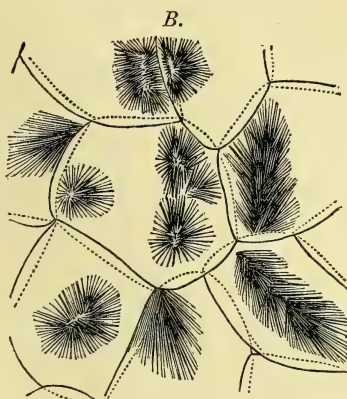


Fig. 67.†

(c.) *Cholesterine* occurs in flat tubular crystals or laminated plates (Fig. 69, a), representing rhombic planes. It also occurs in the form of granules of fat. It almost always occurs with other fats, and often in great



Fig. 68 ‡

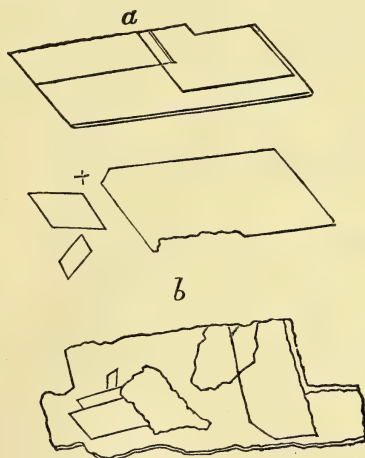


Fig. 69.§

* *A. Crystals of margarine* (after ROBIN and VERDEIL).

† *B. Fat-cells, inclosing crystals of Margarine* (WEDL).

‡ *Margaric acid* (BEALE).

§ *Cholesterine plates*—*a*. Regularly laminated, and viewed obliquely; at +, in the middle solitary plate, one edge is truncated; *b*. Irregularly laminated, partially injured forms: $\times 300$ diameters (after WEDL).

abundance. It is always present in bile; and colourless gall-stones consist almost entirely of this substance. It is also found in the atheroma of arteries; in the serous fluid of ovarian and other serous cysts; in the fluid of old hydroceles. In general terms, it may be stated that wherever tissues are in a state of fatty degeneration, there cholesterine may be found as the product of decomposition of their elements. Thus, cholesterine is found in exudations, tubercle, stratiform coagula in the inner coats of arteries, epithelial cells of the air passages, in bronchitis (BEALE), epithelium and oil-globules in coats of uriniferous tubes in fatty degeneration of the kidneys. It may be extracted from many tissues in health—*e.g.*, crystalline lens of the eye (BEALE). It is non-saponifiable, and is coloured dark red by the action of sulphuric acid. These crystalline fatty concretions are also frequently found in morbid growths, such as *lipoma*, and very commonly in various fluids and solids of the body.

3. *Pigment Concretions* from blood, urine, or bile, assume the following forms:—

(a.) *Black, brown, russet-yellow* molecular granules, often adherent to microscopic crystals of *ammonio-phosphate of magnesia*.

(b.) *Bile-pigment*, as a finely granular precipitate of a yellow-brown colour, insoluble in water and in most acids; soluble in a boiling potash solution, with a greenish-brown tint. Nitric acid destroys it, after causing it to pass through phases—first, of green, then of blue, and lastly of red coloration.

4. *Concretions of Uric Acid and Urates*, simply, or in the following combinations:—

(a.) *Uric Acid*.—The fundamental type of this crystal (Fig. 70) is the rhomboid prism—often cut down to a rhombic plane. Frequently the crystals are seen grouped into concretions as rosettes, and are difficult of solution in water. They are insoluble in acids, alcohol, and ether. Potash causes their gradual solution, and they are generally soluble in alkaline fluids. It occurs—

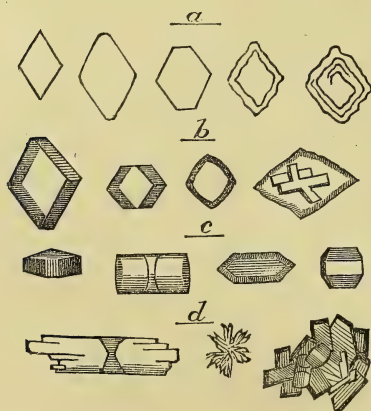


Fig. 70.*

(1.) As a sediment in the urine.
(2.) As concretions of minute crystalline grains, of a gritty feel, but indistinctly visible. Such concretions are commonly known as "gravel."

(3.) As amorphous or crystalline concretions, from a size clearly visible to a very large size, when they are named "calculi."

* *Crystals of uric acid*.—*a*, Rhomboidal, truncated, hexahedral, and laminated crystals. *b*, Rhombic prism; horizontally truncated angles of the rhombic prism; imperfect rhombic prisms; on the last crystal in this row is seated a group of rectangular crystals. *c*, Prism, with a hexahedral basic surface—barrel-shaped figure; prism, with a hexahedral basal surface. *d*, Cylindrical figure; stellate and superimposed groups of crystals: $\times 300$ diameters (WEDL).

(b.) *Urate of Ammonia* (Fig. 71) occurs as a finely-granular precipitate, coloured of a dingy yellow, yellow-red, russet or rose-tint. It also occurs in the form of globules. It is difficult of solution in cold water, less so in hot. Acids will separate the uric acid, which, under the microscope, will be seen to assume their characteristic form, as shown in the previous figure.

5. *Concretions of Lime Salts* are composed of the following forms:—

(a.) *Basic Phosphate of Lime*, occurring as a gelatinous granular mass, soluble in acids, both in fluids and in solidified formations; a soluble combination of protein substances with gluten, out of which it separates, especially in putrefactions and ossifications, in the form of molecules.

(b.) *Carbonate of Lime*, in a granular deposit in cell incrustation or stratification, either alone or in combination with the former. It is soluble in acids with effervescence.

(c.) *Oxalate of Lime* occurs in octahedral crystals, sometimes remarkably minute. They are insoluble in water, alcohol, ether, and acetic acid; but soluble in hydrochloric acid. They are found in urine and in the urinary bladder (Fig. 72).

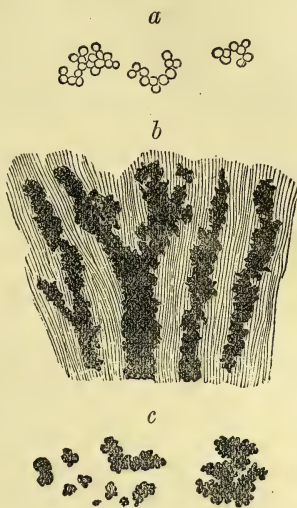


Fig. 71.*

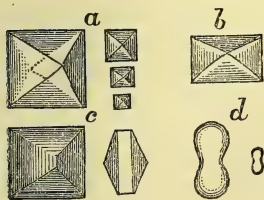


Fig. 72.†

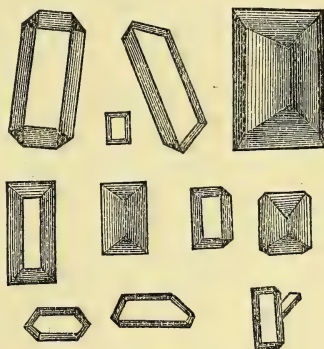


Fig. 73.‡

6. *Concretions of Ammonio-Phosphate of Magnesia* are composed of crystals of various shapes (Fig. 73). When rapidly formed, they cluster

* a, *Urate of ammonia* in the form of globules; b, *Urate of ammonia* as an orange-yellow, fine, sandy concretion in the tubes of the kidney of a child, forming divergent, hard, irregular, black streaks, with lateral branches and twigs imbedded in the tubular substance: $\times 60$ diameters; c, Angular molecules of urate of ammonia, aggregated into black concretions.

† a, Quadratoctahedrals of oxalate of lime; b, The basal plane of an octahedron, forming a rectangle; c, Compound forms; d, Imperfect forms (dumb-bell crystals): $\times 300$ diameters (WEDL).

‡ The more usual forms of triple-phosphate of magnesia and ammonia: various metamorphic, hemihedral forms of the fundamental figure—the rhombic vertical prism: $\times 300$ diameters (WEDL).

together in stellate groups of needle-shaped crystals, or represent denticulate leaf-like forms. When slowly developed, they constitute trilateral prisms, in which both angles, corresponding to the same lateral edge, are truncated. These crystals are readily soluble in acids, even acetic acid. Wherever a development of ammonia takes place, the wide dissemination of phosphate of magnesia determines the formation of the insoluble triple phosphate.

In the formation of calculi and concretions these elementary constituents are classed by Rokitsansky under two series, namely:—

(1.) Those made up essentially of *protein substances*; or into such as consist of *gluten* and *fat*, with the *phosphates* and *carbonates of lime* and *magnesia*.

In this series are included the following:—

(a.) *Protein concretions*, as coagula within blood-vessels (vegetations); free bodies in serous cavities.

(b.) Gluten-like colloid substances, commonly encysted, or conified concretions, as the valve-vegetation of the heart.

(c.) *Fat*, as in the contents of fatty cysts, or accumulations of fat in serous cavities, or in combination with—

(d.) *Bone-earth concretions*, as in cretification and ossification of fibroid and cartilaginous textures (see “Mineral Degeneration,” p. 125, *ante*).

(2.) Into such as have a more varied composition, and are especially marked by the specific substances which they contain,—*e.g.*, bile, urine, &c. In this second series of concretions there are two varieties:—

(a.) Genuine stony concretions or calculi, which, when diminutive, are termed “gravel” or “sand,” resulting from the *precipitation of one or of several of the specific components of a secretion*, the animal matter entering into their composition as a bond of union or cement. The size of such calculi are extremely varied, from a fine but just perceptible sand-grain to that of a concretion filling up the largest secretory canals and reservoirs. The smaller are usually spherical; the larger have their shape in some measure determined by the form of the canals or reservoirs in which they occur; as, for example, gall-stones in the bile-ducts, and calculi in urinary bladder. Where many concretions co-exist, they acquire more or less smooth facets, from reciprocal pressure and friction; as, for example, numerous calculi in the gall-bladder or in the urinary bladder. The consistence and specific weight of calculi depend on their composition. They may be free in a cavity, or firmly impacted, or glued to a surface by the medium of a fibrinous exudation. In structure they are extremely varied. A nucleus is sometimes recognisable, composed of an amorpho-granular precipitate, round which concentric strata or crystalline formations accumulate; or they may be altogether crystalline, as the lithic acid calculus; or of cholesterine, as the white gall-stones. Sometimes a foreign body may form a nucleus, introduced from without, as into the urinary bladder; or a coagulum of blood or fibrine, or inspissated bile or bile-pigment, to a cholesterine calculus in gall-bladder. Examples of such calculus formations are to be found in calculi of the urinary bladder, salivary calculi, calculi of the lachrymal sac, prostatic calculi, gall-stones, and intestinal concretions.

(b.) *Inspissation* and *desiccation* of some fluid of secretion. The ingredients

of the secretion from the substance of concretion or calculus, including a considerable amount of animal matter, combined with the specific elements of secretion. In proportion to the inspissation is the hardness of the concretions. Cyst-like dilatation of follicles gives space for such concretions to accumulate; for example, the follicles of the skin, mucous follicles, the tonsils, nasal and pharyngeal cavities, glands and prepuce, intestinal concretions, especially in diverticula of the intestines, inspissation of colloid or other cyst-contents.

A microscopical analysis of concretions is essential for a determination of their source and character, and ought never to be omitted (See WEDL, p. 121, for an instructive illustration of this part, too long for quotation.)

MALFORMATIONS.

LATIN EQ., *Deformitas Ingenita*; FRENCH EQ., *Vice de Conformation*; GERMAN EQ., *Missbildung*; ITALIAN EQ., *Vizio di Conformazione*.

Definition.—*Deviations from the normal development of the organism of the body occurring in the earlier period of gestation, or at least previously to the termination of fetal existence.*

Pathology.—The imperfections consequent on such primitive malformations result in more or less permanent deformity, to which the harsh name of *monstrosity* has been given; and the doctrine of such *congenital deformity* is now comprehended under the scientific name of "Teratology" (Τέρας, signifying monster). The superstitions, absurd notions, and strange causes assigned to the occurrence of such malformations are now fast disappearing before the lucid expositions of those famous anatomists who have made the development and growth of the ovum a subject of special study. It is sufficient to mention here the names of J. Muller, Rathke, Bischoff, St. Hilaire, Burdach, Allen Thomson, G. and W. Vrolik, Wolff, Meckel, Simpson, Rokitsansky, Von Ammon, Von Beer, Balfour Foster, as sufficient evidence that the truths of science will in time dispel the mists of ignorance and superstition. Nevertheless, much mystery still enshrouds the origin of malformations; and opinions concerning them may be considered to raise two main issues, namely,—(1.) Are they due to original malformation of the germ? or, (2.) Are they due to subsequent deformities of the embryo by causes operating on its development?

With regard to the first issue, it is believed that the germ may be originally malformed or defective, owing to some influence proceeding either from the female or from the male; as in the case of the repeated procreation of the *same kind* of malformation by the same parents, deformities on either side being transmitted as an inheritance. Further evidence of original germ-defect is held to exist in the hereditary deformities extending over more than one generation; such, for example, as harelip, excessive number of fingers, hypospadias, and the like.

With regard to the second issue, namely,—whether subsequent deformation may not follow an originally well-formed germ, it has been said,—(1.) That such deformation may be produced by mental impressions on pregnant women; but of this there is an absence of positive proof. (2.)

It has been proved that external injury, suffered by women during pregnancy, will bring about deformation, as in the congenital deformity of *hydrops ventriculorum cerebri*. (3.) The late Sir James Simpson has shown how diseases of the ovum and foetus will bring about deformation of the child. The diseases are chiefly—acute and chronic inflammation of the placenta, attachment of pseudo-membranes to the foetus, and adhesions of the foetus to the placenta. Self-amputations are thus also explained. Moles, mother's marks, and cutaneous spots are ascribed to morbid states of the coats of the ovum. (4.) A very generally recognised cause of malformation consists in impeded development of the foetus, the cause of which is not always obvious, but is for the most part concealed. The impediment may be confined to one part or extended to many; to more than one region of the body, for example, and to more than one apparatus. To understand the results of impeded development, the student requires to know the natural forms through which the several parts of the foetus pass in their normal development to completeness. If he does not know these, he cannot appreciate an "*arrest of development*" where it ceases at a certain point, and advances no farther. The increased growth and progressive nutrition of the normal portions of the body also impart important modifications to the final result. Transient forms of the human foetus are comparable to persistent forms of many lower animals; hence, malformations resulting from arrest of development often acquire an animal-like appearance. Many human forms are unfit for life after birth.

The following elementary facts in "teratology" are essential to a proper understanding of malformations:—(1.) Dissimilar parts are never fused or united; only parts that are not originally dissimilar, but which are developed from a common mass. (2.) Malformed parts are restricted to their determinate place. (3.) No malformed organ ever loses entirely its own character, nor a malformed animal its generic distinction—a distinct gradation and natural order are observable throughout. Thus, there are different degrees of malformation in the same deformity, varying from the greatest possible degree to the very least. (4.) Deformities do not take place by chance, and double deformities are always of the same sex. No suitable classification of deformities can yet be given; but taking *embryogenesis* as a basis, a grouping may be made, useful alike for physiological and medical practice. Such is that originally given by F. A. Von Ammon and W. Vrolik. The same principle seems to have guided the College of Physicians in the following grouping of malformations (p. 237, Appendix to *Nomenclature of Diseases*):—

I.—MALFORMATIONS RESULTING FROM INCOMPLETE DEVELOPMENT OR GROWTH OF PARTS.

(a.) OF THE BODY GENERALLY.		Upper and lower extremities absent. Lower extremities absent. One lower extremity absent. Hands and feet articulated to scapulæ and pelvis. Fingers and toes deficient in number.	Referable to very early periods of develop- ment.
Head absent, or rudimentary.	(Nine types are described by Vrolik.)		
Cranium defective.			
Lower jaw absent or defective.			

(b.) OF THE NERVOUS SYSTEM.

Brain absent, with exposure of base of skull.

Brain rudimentary or incompletely developed—several types, according to incompleteness.

Spinal cord absent or imperfect, with more or less exposure of spinal canal.

Continuity of nerves with nerve-centres incomplete.

(c.) OF THE ORGANS OF SPECIAL SENSE.

Eyes absent.

Eyes imperfect.

Eyelids incomplete. Eyelids remaining united. (Symblepharon.)

External ear absent. Pinna adherent.

Meatus externus closed.

Internal ear imperfect.

Nose absent.

Nose imperfect.

Nose resembling a proboscis.

(d.) OF THE VASCULAR SYSTEM.

Heart absent.

Cavities of heart deficient in number.

a. One auricle and one ventricle.

b. Two auricles and one ventricle.

Septa incomplete.

a. Auricular.

b. Ventricular.

Orifices obstructed or imperfect.

a. Right auriculo-ventricular aperture.

b. Pulmonic aperture.

c. Left auriculo-ventricular aperture.

d. Aortic aperture.

Foramen ovale prematurely closed.

Ductus arteriosus prematurely closed.

Origins of aorta and pulmonary artery transferred.

Origin of ascending aorta from left ventricle, and of descending aorta from right ventricle, through the ductus arteriosus.

Commencement of descending aorta, contracted or obliterated.

Foramen ovale persistent.
Ductus arteriosus pervious.
Cardiac valves imperfect.
Pericardium absent.

(e.) OF THE RESPIRATORY SYSTEM.

Lung (one or both) absent.

Pulmonary lobes deficient in number.

Larynx and trachea absent or imperfect.

(f.) OF THE DIGESTIVE SYSTEM.

Esophagus impervious.

Intestine impervious, or deficient in various regions.

Anus impervious.

Anus in unusual situations.

Liver preternaturally small.

Gall-bladder absent.

Biliary ducts impervious.

Urachus patent. Vitelline duct patent.

(g.) OF THE URINARY SYSTEM.

Kidney (one or both) absent.

Kidney lobulated.

Ureters absent or impervious.

Urachus persistent.

(h.) OF THE MALE ORGANS OF GENERATION.

Penis diminutive, resembling clitoris.

Prepuce abbreviated—elongated.

Testicle (one or both) absent.

External organs absent.

(i.) OF THE FEMALE ORGANS OF GENERATION.

Ovary (one or both) absent.

Uterus absent.

Vagina absent.

Vagina impervious.

Vagina a cul-de-sac.

External organs absent.

[Hymen imperforate.]

II.—MALFORMATIONS RESULTING FROM INCOMPLETE COALESCENCE OF THE LATERAL HALVES OF PARTS WHICH SHOULD BECOME CONJOINED.

(A.) ON THE ANTERIOR MEDIAN PLANE.

Fissure of the face.

" " iris. Coloboma.

" " lip.

a. Single harelip.

b. Double harelip.

" " palate.

a. Hard palate.

b. Soft palate.

Fissure of the nose. Naso-buccal fissure.

" " sternum.

" " diaphragm.

" " abdominal walls.

" " pubic symphysis.

" " anterior wall of urinary bladder (with extroversion of the posterior half).

Epispadic fissure of the urethra.

Hypospadic fissure of the urethra.	Fissure of the spinal column. Spina bifida.
Fissure of the scrotum.	a. Complete.
	b. Partial.
(B.) ON THE POSTERIOR MEDIAN PLANE.	1. Cervical region.
	2. Lumbar "
	3. Sacral "
Fissure of the skull.	Fissure of the spinal cord.

III.—MALFORMATIONS RESULTING FROM COALESCENCE OF THE LATERAL HALVES OF PARTS WHICH SHOULD REMAIN DISTINCT.

Lower extremities conjoined. Syreniform fœtus.
 Fingers or toes conjoined. Coalescence—webs.
 Monoculus. Cyclops.
 Double kidney.

IV.—MALFORMATIONS RESULTING FROM THE EXTENSION OF A COMMISSURE BETWEEN THE LATERAL HALVES OF PARTS (CAUSING APPARENT DUPLICATION.)

Double uterus. | Double vagina.

V.—MALFORMATIONS RESULTING FROM REPETITION OR DUPLICATION OF PARTS IN A SINGLE FŒTUS.

Supernumerary fingers and toes.
 „ cavities to heart.
 „ valves.

VI.—MALFORMATIONS RESULTING FROM THE COALESCENCE OF TWO FŒTUSES, OR OF THEIR PARTS.

Fœtus, more or less perfect, contained within another fœtus (<i>fœtus in fœtu</i>).	1. The middle parts united. The upper and lower distinct.
Fœtus, more or less perfect, constituting a tumor covered by integument.	2. The upper parts united. The lower distinct.
Double fœtus.	3. The lower parts united. The upper distinct.
a. One perfect. The other an appendage.	
b. Both more or less perfect.	

VII.—CONGENITAL DISPLACEMENTS AND UNUSUAL POSITIONS OF PARTS OF THE FŒTUS.

Transposition of viscera—[Influence of fœtus as always developed lying on left side, as a rule, to be considered here.]

Hernia or ectopia of the brain.
 „ „ heart.
 „ „ lungs.
 „ „ intestines.

Varieties :

Through diaphragm. *Syn.*, Diaphragmatic hernia.
 Through abdominal walls. *Syn.*, Abdominal hernia.
 Through umbilicus. *Syn.*, Umbilical hernia.
 Extroversion of posterior wall of bladder.
 Testis retained in abdomen.
 „ „ inguinal canal.

In the case even of double or duplex deformities only *one* germ seems to be concerned. In illustration of this, Dr. Allen Thomson has given the



Fig. 74.*

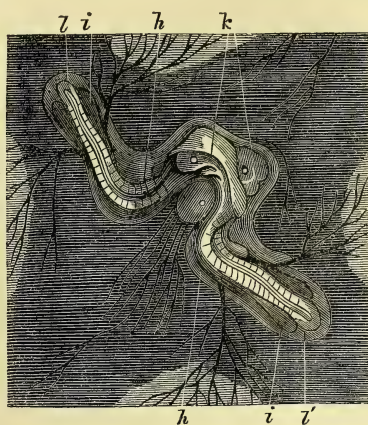


Fig. 75.†

following demonstrations (Figs. 74 and 75). He has shown that on one yolk, and on one germinal membrane or blastodermic vesicle, two primitive grooves may be formed, which, in their ulterior development, shall probably (certainly, if they live?) form a double monster, as may be seen in Fig. 75, taken from a goose's egg after five days' incubation. The formation of such a primitive groove in a single ovum is sufficient to explain the origin of the principal types of double monsters. Such malformations of the ovum have been overlooked by the College of Physicians, and are omitted in the Table just quoted.

They are the earliest examples of double deformity that have ever been recorded.

FUNCTIONAL DISEASES.

LATIN EQ., *Vitia Naturalium Actionum*; FRENCH EQ., *Maladies Fonctionnelles*; GERMAN EQ., *Functionelle Krankheiten oder Affectionen*; ITALIAN EQ., *Malattie di Funzione*.

Definition—*All those diseases in which the concurrent living action of the parts or organs (i.e., the concurrent exercise of function) is not maintained, or in which the contractility, tonicity, nutrition, secretion, sensation, or motion of parts may be increased, diminished, or perverted, and that without fever or inflammation.*

* From a fowl's egg, after sixteen or eighteen hours' incubation, magnified four times.—*a*. The germinal area of the cicatricula. *b*. The transparent area, containing two primitive traces of embryos; *c*, *c*. Primitive grooves of the double embryonic trace, on each side of which are seen the laminae dorsales (after A. THOMSON).

† Double embryo removed from a goose's egg, after five days' incubation, magnified four times.—*g*. The common heart; *h*. Rudiments of the superior; *i*. Of the inferior extremities; *k*. The common cephalic fold of the amnios; *l*. The caudal folds (after A. THOMSON).

Pathology.—Hitherto the complex morbid processes which have been described embrace nearly all the important forms of organic disease which the eye can appreciate, assisted or not by the microscope, or which can be appreciated by other physical aids, such as by chemical analysis or reagents. Morbid changes which are not visible, or which are not yet ascertained by any physical means of observation, are described as *functional* or *dynamic*. They embrace all those diseases in which the action, the secretion, or the sensation of a part is impaired, without any primary alteration of structure of the organ or tissue affected, so far as our imperfect means of research can ascertain. It is possible, however, as physical aids to the senses increase and become more practicable, thereby improving the means of observation, that such so-called functional changes may eventually be shown to depend upon some concomitant anatomical alteration of structure (molecular it may be), which at present is not appreciable. The appreciable morbid forms of disease which have been described, and which are either permanent or more or less persistent after they are formed, are described in the science of morbid anatomy as *lesions*. They are common to a greater or less number of individual diseases, hereafter to be considered and described in this Text-book. It will then be seen that such lesions do not essentially constitute the disease, but are rather the results of disease, and are serious or otherwise according to their nature, their site, and the amount of structural change involved. It is the duty of the pathologist and the physician to connect them with signs and symptoms of disease, with the object of adopting remedial measures for their prevention or cure, or for the relief of such inconveniences as they may cause.

But the class of complex morbid states, known as "*functional*," are often not less strikingly formidable in their symptoms than those attended with obvious organic lesions; they are, in many instances, the cause of much suffering. Usually of long duration, without fever, they are generally difficult of cure, having a strong tendency to recur, and to terminate eventually in visible organic changes. The occasional exciting causes of such diseases act upon the vital functions, and will be considered in detail under the various organs whose functions are thus specially implicated. They have hitherto been described under the name of "*Neuroses*;" but, year by year, as a knowledge of morbid anatomy extends the number of these functional diseases diminishes. They were so named because "*functional*" diseases were believed to have their origin in the nervous system, and were indicated by disordered sensation, volition, or mental manifestation, without any evident lesion in the structure of the part, and without any material agent producing them. Broussais attributed them to a state of irritation of the brain and spinal marrow. In the nomenclature of the College of Physicians of London, the functional diseases of the nervous system constitute a group, which embraces all affections of which the cause is either undefined or variable, whether its probable seat be the brain, the cord, or the nerves. Its components are so far heterogeneous as to include such diseases as hydrophobia, epilepsy, neuralgia, tetanus, cramps, chorea, shaking palsy, hysteria, catalepsy, trance, and hypochondriasis.

FEVER.

LATIN EQ., *Febris*; FRENCH EQ., *Fièvre*; GERMAN EQ., *Fieber*; ITALIAN EQ., *Febbre*.

Definition.—*A morbid condition of the whole system in which the temperature of the blood is raised above the normal standard.*

Pathology.—This very complex morbid state accompanies the so-called febrile diseases and specific fevers as part of their phenomena, with more or less defined regularity. It is, however, variously modified according to the specific nature of the disease of which it is an essential element; and it also takes part in the expression of many diseases, both local and general. "The process of fever, as regards the width and depth of its practical interest to persons concerned in the treatment of disease, is certainly second to none which the body suffers; for at every turn of medical practice, it is apt to present itself for treatment as a more or less prominent part of the patient's total illness; and in a large proportion of such cases, it is an influence which involves the issue of life or death" (SIMON).

The practical object aimed at in the exposition about to be given, is to fix the scientific principles which ought to guide clinical investigation in determining the *Natural History* OF FEVERS generally; and, especially, the scientific principles according to which we must define the differences which subsist among specific fevers; and so aid in determining the conditions under which they are generated or propagated—their development, course, or progress, and their defervescence. The nature of the febrile process from clinical and experimental observation shows that the characteristics of fever relate to five points, namely—(1.) Disintegration of the living substance of the body. (2.) Increase and diminished constancy of bodily temperature. (3.) The origin of fever. (4.) Its progress; and, (5.) Its termination. It is a more or less continuous process; and begins by the entrance into or action on the organism of some affecting or infecting cause. Then follows a period of latency or incubation, after which the first indications of fever begin; when it may vary indefinitely in its course, its progress, its duration, and in the local inflammations, lesions, or complications which accompany it. Every case of fever, therefore, has its period of onset, accession, and declension, each of which is characterised by certain distinctive phenomena, namely—(1.) The *onset*, by shivering and rise of temperature of the internal parts of the body. (2.) The *accession*, by continued pyrexia and exhausting disorder of the bodily functions. (3.) The *decline*, by epicrisis or defervescence, or cooling down and restoration of the functions of the body to their normal conditions and relations. *Convalescence* ought to commence when the febrile process is at an end, therefore convalescence does not form a part of the febrile process (SANDERSON, *Public Health*, No. vi., 1875).

"A hot skin, a quick pulse, increased respiratory acts, intense thirst, a foul tongue, anorexia, scanty and high-coloured urine," are phenomena common to many diseases; and when they are present, it is said that the patient is *feverish*, or that he suffers from *fever* or *pyrexia*. The cause of such fever or pyrexia is due,—(1.) To an abnormal condition of the blood

by which its temperature is increased, and which is induced by the specific poisons of the general diseases, to be considered afterwards in the Sections A and B of the General Diseases. These poisons are either generated within the body, like gout or rheumatism; or they are received into the body from without, like the poison of small-pox, typhus, enteric fever, ague, cholera, and the like. (2.) To a local injury, such as a wound, an amputation; or to a local lesion, like an inflammation of a living stomach or intestine; to a carbuncle or a boil. In fever due to the first set of causes, the symptoms constitute the prominent and almost the only appreciable phenomena, and they run a more or less definite and acute course. Such diseases have been emphatically termed "fevers," or sometimes *specific, essential, primary, or idiopathic fevers*. When diseases marked by local lesions—such, for instance, as the local inflammations—are attended by the symptoms just stated, then the *pyrexia, fever, or feverish* symptoms which attend them are said to be *secondary or symptomatic*; and the physician is accustomed, when he deals with such cases, to abstract the symptoms of fever from the other symptoms proper to the special affection. In other words, he prescribes for, and tries to cure the special affection, and not the *fever*, because he knows that when he has subdued the local disease the *fever* will subside. Not so, however, with the fever of specific diseases, like *small-pox, typhus, or enteric fever*. The physician cannot cure such a fever; but he may guide its course, by judicious management, as an experienced pilot may guide a ship and preserve it through a storm; and this is true of all specific fevers.

It is to the nature of *fever* (whether *primary, idiopathic, and specific*; or *secondary and symptomatic*), considered in its general or abstract relations, that the attention of the student is here directed, and not to any particular fever, such as *ague, typhus fever, traumatic fever*, or the like. It is to the *pyrexial symptoms* which are common to many diseases (such as to *small-pox, scarlatina, measles, typhus, ague, pneumonia, nephritis, meningitis*), and which, "like shadows to substance, are necessary to the very existence of such diseases, but yet are not, *per se*, any one of them." The presence of fever is thus so common to many diseases, that they have been divided into two great classes—the *febrile* and the *non-febrile*; and in diagnosing the nature of any disease, one of the first things to determine is the presence or absence of fever. Then, What is fever? Galen defined fever as a preternatural heat—"Calor præter naturam." Subsequently many other additional clauses were added to this definition, such as a "quick pulse," "turbid urine," and the like; but still the definition so improved does not meet the requirements of every case. Mere quickness of pulse does not constitute fever; and of all the clauses and phrases in the usual definitions of fever, "preternatural heat" is the only one whose accuracy is unimpeachable. In all cases, therefore, where fever is present, there are two points to be determined; namely,—(1.) *The amount of the preternatural heat*, ascertained by accurate measurement at two or three different hours in the course of the day; (2.) *The amount of the tissue-change*, as represented by an estimation of the amount of all the excreta relative to the body weight. In the diagnosis of fevers it is the exact sequence of phenomena we desire to know, as well as the meaning and correlation of these phenomena;

and symptoms sufficiently characteristic usually become superadded to the febrile phenomena, by which we are able to define the specific nature of the disease or the fever as a whole, and so to clench the diagnosis as to say of this case or of that,—“It is an *enteric fever*,” or “It is an *ague*,” or “It is a *rheumatic fever*,” or “a *pneumonia*,” or “a *dysentery*,” or any other form of illness where pyrexia is present. It is not very long since we were able to do this. Up till within a comparatively short time ago, the classification and diagnosis of “fevers” were not such as to distinguish and separate their varied forms and varieties from each other. “Common continued fever,” for example, was a comprehensive name which included many very different types of fever. The phenomena, then, for investigation in determining the pathology of fever, relate—(1.) To the textural disintegration of the bodily substance; (2.) To the general disturbance of bodily function, of which the disorder of temperature is the chief, expressed through—(a.) the production, and (b.) the discharge of heat in fever—“febrile pyrexia;” (3.) To the varieties or types of fever; and, (4.) To their differential diagnosis. Of these in their order.

I. Of the facts which relate to the textural disintegration of the bodily substance.

In fever the body wastes. By methods of measurement and comparison we determine how this waste takes place, how the living substance of the body is disintegrated;—a disintegration which is really the characteristic or central attribute of the febrile process. Measurement without comparison is useless; hence a standard for comparison must first be established. Thus, *in health* the discharge of nitrogen in the excreta is exactly counterbalanced by its introduction in our food—the body being then in a normal state of nutrition, whether the actual amount of nitrogen discharged be large or small which passes through the body in a given time. In all such observations, therefore, it is necessary to determine—(1.) The amount of nitrogen which enters by food; (2.) Amount of nitrogen which passes away by excreta (see PARKES' *Hygiène*). By balancing the one amount against the other, we may learn whether the amount stored in the body is being wasted, on the one hand, or retained and accumulated, on the other. We judge whether *integration* or *disintegration*—(evolution or involution)—is going on; not by absolute income or expenditure, but by nitrogen exchange; by the excess or defect of nitrogen expenditure as compared with nitrogen income. The experimental investigation of the disintegration of tissue from this point of view, is mainly due to Dr. Senator, of Berlin. His object was not merely to determine the exchange of nitrogen, but also its relations with the production of heat and the discharge of carbonic acid. For this purpose he *compared* the exchange of material and of heat in the febrile state with that of health. The animals experimented on had, therefore, to be brought into a condition of nutritive equilibrium—*i.e.*, they were carefully fed on a certain diet, which was increased or diminished until the body weight and the daily discharge of nitrogen by urea, and the “insensible” loss, became severally constant. This regulated diet having been maintained for several days, food was then withdrawn for forty-eight hours, during which period the normal *discharge* of carbonic acid, water, urea, and of heat, were measured and body weight

determined. The regulated diet was then recommenced, and continued till nitrogen-equilibrium was once more established. This having been accomplished, fever was induced by the subcutaneous injection of fresh pus, and the several determinations were repeated as before, *under exactly similar conditions as regards nitrogen*, during a second period of forty-eight hours (*i.e.*, no food was given). The febrile condition induced was well marked; the accession by rigors, and the skin felt hot to the hand. The temperature (rectum) began to rise in from one to two hours after injection of infective liquid, the pyrexia continuing about two days. The animals were listless, and had no desire to eat; but would drink water with avidity whenever it was given them. Each experiment gave two similar periods of observation for comparison; namely—two forty-eight hours periods of observation, separated from each other by an interval of several days, during which period the production of heat, the changes of bodily weight, the daily quantities of urine and urea excreted, and the respiratory and cutaneous discharges of carbonic acid and water were determined.

Relation of Amount of Excreta to Ranges of Temperature in Febrile Diseases.—The particular and preternatural degree of heat, and the waste by disintegration of tissue in every febrile disease, are thus represented by—*something*. The physician sees the fevered patient wasting before his eyes. Every tissue is wasting, and, in correlation with the excessive generation of heat, how is this waste expressed? As a rule, it is expressed by the amount of *excreta*; and the preternatural heat is the result of exalted vital and chemical action above the standard of health. But the source of the heat in fever differs in one respect from that in health, when the elimination of nitrogen is entirely regulated by the amount entering the body with the food (PARKES). In fever the increase does not come from the food, and is out of all proportion to the food taken. It is the fixed albumen of the tissues, which breaks up into circulating albumen, to be transformed into *urea* and the other nitrogenous *excreta* (MURCHISON). Hence the muscles waste, and the brain and nervous system atrophy. Cerebro-spinal fluid (so commonly seen after death in fever cases) exudes from the blood to fill the vacant space caused by the waste of those parts which are inclosed in unyielding cavities. Such fluid is not the result of inflammation. In extremely prolonged cases of fever, granular degeneration occurs in the muscles, and in the ganglia of the sympathetic. The action of the heart and of the circulation is impaired, from the impaired nutrition and degeneration of the heart; hence cardiac action is weak, and the blood stagnates in the capillary circulation in the advanced stages of fevers. And so now it has been demonstrated, both by clinical and experimental observation, that the morbid development of heat, *as measured by a thermometer*, is associated in some cases with MORE abundant, in other instances with LESS abundant, *excreta* from the body than in health; that the temperature and the amount of the excretions bear some as yet undetermined relation to each other; that the loss of weight of the patient is due to increased and rapid elimination of material with increased tissue-change, associated with the increase of temperature; and that it far exceeds what can be accounted for by the mere loss of food. So far as physiological facts have elucidated the normal generation

of heat in the healthy body, so far has the abnormal generation of heat essential to the *febrile* state been clearly made out. In health the normal temperature produced by vital and chemical change in the body is represented in the excretions by so much *urea*, *sulphuric acid*, *carbonic acid*, *excretive volatile acids* of the skin, and the like; but in the febrile body a higher temperature is represented in the excretions, in some cases by a larger, and in others by a smaller quantity of *urea*, *sulphuric acid*, and *probably carbonic acid* (PARKES). In the study of special diseases the student ought frequently to estimate the quantity of *excreta*,* especially the *urea* and *uric acid*, passed by the urine, as one of the best methods for enabling him to appreciate the changes which are going on in the body. (See Dr. PARKES *On the Composition of the Urine*; see also "Croonian Lectures on Elimination of Nitrogen," *Lancet*, 1871, Vol. I.; and the directions given on the "Examination of the Urine," towards the end of the second volume of this text-book, for obtaining quantitative results by the volumetric method.) But these numbers in the footnote, as hitherto stated, are far too great, inasmuch as the question to be determined is—what quantities of urea and carbonic acid ought a normal person in health to discharge from his body *when in bed and on a fever diet*. This fundamental consideration (*in italics*) has been neglected in clinical observations on fever; and unless the influence of diet is duly estimated in health under conditions as to diet and rest similar to those of a febrile patient, the mere determination of the daily discharge of urea is of no value whatever. The general result of an extensive series of observations is, that in the early stage of fever, a patient excretes about three times as much urea as he would do on the same diet if he were in health; the difference between the fevered and healthy body consisting chiefly in this, that whereas the former discharges a quantity of nitrogen equal to that taken in, the latter wastes the store of nitrogen contained in its own juices,—a disorder of nutrition which is an essential constituent of the febrile process. The discharge of carbonic acid is augmented to about 50 per cent., and more carbonic acid is formed in fever than in health (SANDERSON). Two grains of urea per pound weight of the body is the *minimum* eliminated as the product of vital work alone (HAUGHTON and others). The urinary solids may be estimated by multiplying the number of ounces of urine passed in the twenty-four hours into the number representing the specific gravity, the product being the weight of solids in grains. A convenient table (see pp. 262, 263) is given by Dr. Haughton, founded on many observations of urine both in health and in disease, of specific gravities from 1003 to 1028. The results are approximations to the daily excretion of urea in all cases where sugar and albumen are absent.

* Here the following general results may be given as a standard for comparison:—

Average quantity of Urine passed in twenty-four hours,	.	52 $\frac{3}{4}$ to 56 ounces.
" amount of Solids	" "	95 $\frac{1}{4}$ grains.
" " Urea	" "	512 "
" " Chlorine	" "	126·76 "
" " Free Acid	" "	33 "
" " Phosphoric Acid,	" "	45·80 "
" " Sulphuric Acid,	" "	31·11 "
" " Uric Acid,	" "	8·5 "
" Specific gravity,	.	1·020

REV. SAMUEL HAUGHTON'S TABLE FOR THE DETERMINATION OF UREA IN URINE (See p. 261).

Fluid Ounces.	SPECIFIC GRAVITY.																									
	1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028
20	35	36	43	57	71	85	100	103	106	119	130	136	142	151	160	196	233	241	249	257	265	274	276	278	279	280
21	37	38	45	59	74	89	105	108	111	124	136	142	149	158	168	205	245	253	261	269	278	288	290	292	292	294
22	38	40	47	62	78	95	110	113	116	130	143	149	156	166	176	215	257	265	274	282	292	301	303	305	306	308
23	40	41	49	65	81	97	115	118	121	136	149	156	163	173	184	225	268	277	286	295	305	315	317	319	320	322
24	42	43	51	68	85	101	120	123	127	142	156	163	170	181	192	235	280	289	299	308	319	329	331	333	334	336
25	43	45	53	71	88	106	125	129	132	147	162	170	177	188	200	245	291	301	311	321	332	342	345	347	348	350
26	45	47	55	73	92	110	130	134	137	153	169	176	184	196	208	254	303	313	324	334	346	356	359	360	362	364
27	47	49	57	76	95	114	135	139	143	159	175	183	191	213	216	264	314	325	336	347	359	369	372	374	376	378
28	48	50	59	79	99	118	140	144	148	165	182	190	198	221	224	274	326	337	349	360	372	383	386	388	390	392
29	50	52	61	82	103	122	145	149	153	171	188	197	205	228	232	284	337	349	361	373	386	397	400	402	404	406
30	52	54	64	85	106	127	150	155	159	177	195	204	213	226	240	294	349	361	374	386	398	411	414	416	418	420
31	55	56	66	87	109	131	155	160	164	182	201	210	220	233	248	303	361	373	386	398	412	425	428	429	432	434
32	55	57	68	90	113	135	160	165	169	188	208	217	227	241	256	313	373	385	398	411	425	438	442	443	446	448
33	57	59	70	93	116	140	165	170	175	194	214	224	234	249	264	323	384	397	411	424	438	452	455	457	460	462
34	58	61	72	96	120	144	170	175	180	200	221	231	241	256	272	333	396	409	423	437	451	466	469	471	474	476
35	60	63	74	99	124	148	175	180	185	206	227	238	248	264	280	343	407	421	436	450	464	479	483	485	488	490
36	61	64	76	102	127	153	180	185	191	212	234	244	255	271	288	352	419	433	448	462	477	493	497	499	502	504
37	63	66	78	105	130	157	185	190	196	218	240	251	262	279	296	362	430	445	461	475	490	507	510	513	516	518
38	65	68	80	108	134	161	190	195	201	224	247	258	269	286	304	372	442	457	473	488	503	520	524	527	530	532
39	67	70	82	111	138	166	195	200	206	230	253	265	276	294	312	382	453	469	486	501	516	534	538	541	544	546
40	69	72	85	114	142	170	200	206	212	236	260	272	284	302	320	392	465	482	498	514	530	548	552	555	558	560
41	71	73	87	116	145	174	205	211	217	241	266	278	291	309	328	401	477	494	510	527	543	562	566	568	571	574
42	74	75	89	119	148	178	210	216	222	247	272	285	298	317	336	411	489	506	523	540	557	575	580	582	585	588
43	75	77	91	122	152	182	215	221	228	253	279	292	305	324	344	421	500	518	535	553	570	589	593	596	599	602
44	76	79	93	125	156	186	220	226	233	259	286	299	312	332	352	431	512	530	548	566	584	603	607	610	613	616
45	78	81	95	128	160	191	225	231	238	265	292	306	319	339	360	441	523	542	561	579	597	616	621	624	627	630
46	80	82	97	130	163	195	230	236	243	271	299	312	326	347	368	450	535	554	573	592	611	630	635	638	641	644
47	82	84	99	133	166	199	235	241	249	277	305	319	333	355	376	460	546	566	586	605	624	644	648	652	655	658

48	84	86	101	135	170	203	240	246	254	283	312	326	340	362	384	470	558	578	598	618	637	657	662	666	669	672
49	85	88	103	139	174	207	245	251	259	289	318	333	347	370	392	480	581	590	611	631	651	671	676	680	683	686
50	87	90	106	142	178	212	250	257	265	295	325	340	355	377	400	490	581	602	623	644	665	685	690	694	697	700
51	88	92	108	144	181	216	255	262	270	301	331	346	362	385	408	499	593	614	635	656	678	699	704	708	711	714
52	90	94	110	147	185	220	260	267	276	307	338	353	369	393	416	509	605	626	648	669	692	718	721	725	728	738
53	92	96	112	150	188	225	265	272	281	313	344	360	376	400	424	519	616	638	660	682	705	726	731	735	738	742
54	94	98	114	153	192	229	270	277	286	319	351	367	383	408	432	429	628	650	673	695	718	740	745	749	752	756
55	95	99	117	156	195	233	275	283	292	325	358	374	390	415	440	539	636	657	678	702	725	747	752	756	760	764
56	96	100	119	159	199	238	280	288	297	331	364	380	397	423	448	548	645	674	698	720	745	767	772	776	780	784
57	98	102	121	162	202	242	285	293	303	337	371	387	404	430	456	558	662	686	710	733	758	781	776	790	795	798
58	100	104	123	165	206	246	290	298	308	343	377	394	411	438	464	568	674	698	723	746	772	794	800	804	808	812
59	102	106	125	168	209	251	295	303	314	349	384	401	418	445	472	578	685	710	735	759	785	808	814	818	822	826
60	104	108	128	171	213	255	300	309	319	355	391	408	426	453	480	588	697	722	748	772	798	822	828	832	836	840
61	106	109	130	173	216	259	305	314	324	360	397	414	433	460	488	597	708	734	760	784	811	836	842	846	850	854
62	108	110	132	176	220	263	310	319	329	366	404	421	440	468	496	607	719	746	772	797	824	849	856	859	864	868
63	109	112	134	179	223	267	315	324	335	372	410	428	447	475	504	617	730	758	785	810	838	863	869	873	878	882
64	110	114	136	182	227	271	320	329	340	378	417	435	454	483	512	627	742	770	797	823	851	877	883	887	892	896
65	112	116	138	185	230	276	325	335	345	384	423	442	461	490	520	637	754	782	810	836	864	890	897	901	906	910
66	114	118	140	187	234	280	330	340	351	390	431	448	468	498	528	646	766	794	822	849	877	904	911	915	920	924
67	115	120	142	190	237	284	335	345	356	396	437	455	475	505	536	656	778	806	835	862	891	918	925	929	934	938
68	116	122	144	193	240	288	340	350	361	402	443	462	482	513	544	666	790	818	847	875	904	931	939	943	948	952
69	118	124	146	196	244	292	345	355	367	408	449	469	489	520	552	676	802	830	860	888	917	945	953	957	962	966
70	120	126	149	199	248	297	350	361	372	414	456	476	497	528	560	686	814	843	872	901	930	959	966	971	976	980
71	121	127	151	201	251	301	355	366	377	419	462	482	504	536	568	695	826	855	884	913	943	973	980	984	990	994
72	122	128	153	204	255	305	360	371	382	425	469	489	511	544	576	705	838	867	896	926	956	986	994	998	1004	1008
73	124	130	155	207	258	310	365	376	388	431	475	496	518	551	584	715	849	879	909	939	969	1000	1007	1012	1018	1022
74	126	132	157	210	262	314	370	381	393	437	482	503	525	558	592	725	861	891	921	951	981	1014	1021	1026	1032	1036
75	128	134	159	213	266	318	375	386	398	443	488	510	532	566	600	735	872	903	934	964	995	1027	1035	1040	1046	1050
76	130	136	161	216	269	323	380	391	404	449	495	516	539	573	608	745	884	915	946	977	1008	1041	1049	1054	1060	1064
77	132	138	163	219	273	327	385	396	409	455	501	523	546	581	616	755	895	927	959	989	1021	1055	1062	1068	1074	1078
78	134	140	165	222	276	331	390	401	414	461	508	530	553	588	624	765	907	939	971	1002	1034	1068	1076	1082	1088	1092
79	136	142	167	225	280	336	395	406	420	467	514	537	560	596	632	775	918	941	984	1015	1047	1082	1090	1096	1102	1106
80	139	144	170	228	284	340	400	412	425	473	521	544	568	604	640	785	930	964	996	1028	1060	1096	1104	1110	1116	1120
	1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028

The table is one of double entry, and is used by finding the daily excretion of urine in fluid ounces and its specific gravity by means of a carefully graduated urinometer. The data being found, and the numbers corresponding at the intersection of the columns referred to in the table, the excretion of urea is represented by these numbers in grains.

The most opposite statements have thus been made regarding the amounts of the excretions in fever, compared with the amounts excreted in health; and at present many excellent observers hold that these excretions are always, and of necessity, increased; others, no less exact, affirm that they are invariably, or almost always, diminished. Such discrepancy of statement is due, in the first instance, to the difficulty of collecting and measuring exactly the amount of all the excretions. "Two of the excretions, the cutaneous and the pulmonary, cannot be collected and measured with anything like the accuracy necessary in such an inquiry—even in health such an inquiry is difficult, and in fever it is almost impossible." But by careful and accurate observation at the bedside, it is possible to obtain very close approximative data to found conclusions upon relative to the increase or diminution of the excretions. It is to be assumed that when the respirations are not quickened (*i. e.*, about eighteen times a minute, or about one act of respiration for every four beats of the pulse), and when the skin is not evidently sweating, the excretions by these two organs are not increased; and, on the other hand, an increased excretion by these organs may reasonably be inferred, if the exercise of their function is unusually active, and if there are tolerably copious perspirations. The other two excretions—namely, the urine and intestinal discharges—can be measured with accuracy, and the urine in particular is a valuable index of the metamorphoses of tissue. The *urea* alone represents two-thirds of the whole quantity of *nitrogen* which passes off; and there is evidence that the increased excretion of urea precedes the rise of temperature, as demonstrated by Dr. Sidney Ringer (*Med. Ch. Trans.*, XLII., p. 361). As a rule, in the early stages of a fever the temperature goes on increasing to a maximum, and during that period the quantity of *urea* is greatest; and with any unusual elevation of temperature there is also an unusual amount of urea excreted; and which, to some extent, is a measure of the disintegration of—(1.) the stored, and (2.) the tissue albumen—namely, that of the blood-corpuscles and plasma, as well as that of the tissue juice or lymph as the stored albumen, and of the albumen which composes the material of protoplasm (including that of blood corpuscles) as the tissue albumen. The *sulphuric* acid (the sulphates of the food being accounted for) represents almost entirely the oxidation of sulphur; and the oxidised phosphorus of the body passes out in great measure, though not altogether, as urinary phosphoric acid. Therefore a careful daily examination of the urine (which must be collected separately and completely) and of the intestinal discharges, with an approximative estimate of the pulmonary and cutaneous excretions, give sufficiently extensive materials for the question at issue. The products excreted are thus of such a kind as to be eliminated, some by the *lungs*, some by the *skin*, some by the *bowels*, and some by the *kidneys*, and rarely by two or more modes of excretion; for when the discharges from the *skin* or *bowels* are profuse, those by the *kidneys* are usually deficient. The facts hitherto so carefully observed (but chiefly

in regard to the excretion of urea only) justify *one* conclusion, namely—*"That increase of temperature may be attended with increased elimination, and therefore presumably with increased tissue-change"* (PARKES, ALFRED VOGEL). In another series of observations, it has been shown by Dr. Parkes that a *second* conclusion is equally legitimate, namely,—*"That the products of metamorphosis, as judged of by the excreta, may be diminished in febrile cases."* These statements, apparently discordant, are capable of explanation. In the first place, it is evident that more chemical change may go on in the body than is represented by the excreta. The metamorphosis of blood or of tissues may not be carried to the point of forming those principles which can alone pass through the eliminating organs. A vast amount of imperfectly organised compounds may be formed and retained in the system, circulating with the blood or thrown upon certain organs." *Thus there may be increased metamorphosis with increased temperature, but with lessened elimination.* Several pathological facts point to such a conclusion. For example—it is in such febrile cases, with diminished excreta, that at a later period of the disease copious discharges from one or other of the eliminating organs occur; as, in a case of pneumonia in which severe spontaneous diarrhoea comes on; and, many other cases may be quoted, with similar diminution of the excretions at the period of increased febrile heat, in which violent purging, sweating, or diuresis, with increase of urea and of sulphuric acid, subsequently occurs. Such discharges during the progress and towards the termination of a febrile disease, have been termed *critical*, the event being called a *crisis*; and the particular day on which it happens, counting from the day of seizure, has been called a *critical day*. The term *crisis* or *critical* is applied because the occurrence of such discharges is usually coincident with more or less sudden fall of temperature, and general improvement in the condition of the patient, whose convalescence dates from the critical day; when, in common language, his disease is said to have "*got the turn.*" In such cases, therefore, a large amount of partially metamorphosed substances are retained until they are suddenly discharged, and the system freed from the noxious compounds. Coincident with the critical discharge, the temperature is found to fall. In another class of febrile cases retention of the products of metamorphosis is not followed by such a fortunate *critical* issue. At a later period in the history of a case with diminished excreta, it is not uncommon for *secondary inflammatory* affections to occur, as if the blood were more contaminated; and it is sometimes observed that in a patient whose excreting organs are acting copiously, there occurs, more or less suddenly, a diminution of excretion, when a simultaneous or subsequent development of local disease becomes manifest. The *urea* or other less oxidised products of metamorphosis then continue to circulate in the blood; and, permeating the tissues, give rise to symptoms of uræmic poisoning, or to what is called "*the typhoid condition*"—a condition which may also be greatly aggravated by the non-elimination of carbonic acid consequent on congestion of the lungs (MURCHISON). That such non-elimination induces the typhoid state, Dr Murchison is of opinion for the following reasons:—"1. All modern pathologists are agreed that the cerebral symptoms of most fevers are independent of any appreciable structural lesion of the brain or its

membranes. 2. The typhoid symptoms of febrile diseases often closely resemble those of uræmia resulting from disease of the kidneys. The two conditions are, in fact, often mistaken for one another; and excepting that the temperature is increased in the one case and not in the other, it may be sometimes difficult to distinguish them. The symptoms in both cases are probably due to the presence of similar morbid materials in the blood, the difference being that in pyrexia these materials are generated in excess, while in renal disease the kidneys are unable to eliminate the normal quantity. 3. In many fevers presenting typhoid symptoms—such as typhus, relapsing fever, yellow fever, and the secondary fever of cholera—urea has been actually found in the blood and in the cerebral fluid. 4. Lastly, the advent in fever of grave cerebral symptoms, and in particular of convulsions, which may be regarded as the acme of both uræmia in kidney disease and of the typhoid state in fever, is often preceded by albuminuria, or by a marked diminution, or even suppression of urine. When stupor, delirium, and coma show themselves in the course of a contagious fever, it is the custom to refer them to the action of the fever-poison on the brain; but the cerebral functions are more probably deranged, not by the fever poison, which was the first and necessary link of the pathological chain, but by the accumulation in the blood of the products of metamorphosis, and the consequently perverted and defective nutrition of the brain and nerves. Hence it is that the symptoms in the advanced stages of many fevers and local inflammations (the typhoid state) are closely assimilated, although the primary maladies have been widely different. The precise grouping of the symptoms will vary in different cases of the same disease, while in diseases essentially distinct it may be identical" (*Brit. Med. Journal*, l. c.).

Thus it is evident, from carefully recorded observations, "*that diminished excreta in fever are to be referred to retention of such excreta, and not to a want of formation; and that while the amount of excreta (capable of being measured) may in fact be small, the amount of tissue-change may nevertheless be great.*" The importance of the practical lesson so often insisted on by the older physicians,—"*never to lock up the excretions,*"—is thus demonstrated with scientific exactness and authority. Another general and practical conclusion is, that the *febrile heat* cannot be appreciated, even by the amount of the excretions as a whole, nor yet by any ingredient of them in particular; but must be estimated in correlation with them and with diet, age, and body-weight. Under the same degree of animal heat, and in the same disease, different patients pass very different quantities of *urea, uric acid, sulphuric acid, phosphoric acid, cutaneous and intestinal excretions*. The same observation may be made regarding men in health. No two persons pass exactly the same amount of excretory products. The nature of these excretory products shows that it is the albuminous or nitrogenous tissues of the body which are being destroyed in the febrile state; for the excretory products of the urine are the representatives of azotised structures. The amount of these excretory ingredients varies considerably from day to day in fever exactly as in health. Often there is a regular gradation of increase and decrease: the urea, for instance, may, for two or three days, slightly but regularly diminish in amount, and then suddenly augment to its highest point, again slowly to fall. The same

fact may be observed with the sulphuric acid; and Dr. Parkes was led to believe that, both in health and in disease, a certain periodicity, having a range of three or five days, is connected with these gradations of increase and diminution. The largest amount of *urea* excreted in twenty-four hours in the febrile state is recorded of a case of pyæmia by Alfred Vogel—namely, 1,235 grains, an excess of 723 grains at least; but a much greater excess if diet is to be estimated, as it ought to be. The largest amount observed by Dr. Parkes was in a case of typhoid fever, in which it amounted to 885 grains, an excess of more than 373 grains. The largest amount of *sulphuric acid* recorded by the same observer, when no medicine was taken, was in a case of rheumatic fever. It amounted to 52·668 (normal 31·11, excess 41·558) grains; and under the influence of liquor potassæ in the same disease, he has known this excretion rise to 70 grains—more than twice as much as in health. The largest amount of *uric acid* excreted during a febrile disease in twenty-four hours, as recorded by Drs. Parkes and Garrod, has been 17·28 grains—normal being 8·5, excess 8·78. “The amount of tissue destroyed in order to furnish such quantities of excreta must be enormous; and if it is recollected that little or no food is taken by the *feverish* patient, and, therefore, that no materials are supplied for the reconstruction of the textures, thus melting away three times more quickly than in health, the rapid loss in weight in fever, and the impaired nutritive condition of every organ at its close, will be at once evident.”

The elimination of the nitrogenous detritus appears to be often prevented “by some morbid condition of the large glands, and especially of the kidneys, either of old standing or consequent on the febrile attack. Disease of the kidneys is a serious complication of all fevers. As long as the kidneys are equal to the increased work thrown upon them, the blood is properly depurated, and the typhoid state is warded off. But if the kidneys be unequal to the task, either from the large amount of effete material to be eliminated, from previous disease of the secreting tissue, or from congestion and other changes, resulting (as they often do) from their increased work, then the blood becomes polluted, and convulsions or the typhoid state supervene. This explains why albumen so commonly appears in the urine in the course of all acute diseases of a severe form, and why it is so justly regarded as an unfavourable symptom. It also explains why albuminuria is looked upon as a contra-indication to any serious surgical operation. The surgical fever which follows an operation is attended by an increased metamorphosis of the blood and tissues; and if the kidneys be diseased, the products of this metamorphosis will be partly retained in the blood, and induce the typhoid state with all its dangers. “When convalescence from fever is fairly established, and the patient is regaining weight, the elimination of nitrogen, and also the temperature, are often below the normal standard” (MURCHISON, *l.c.*).

It is not yet determined where the increased destruction of the albuminous textures takes place; that is, whether it occurs in the blood or in the organs themselves. It is only known that both the albumen and the red corpuscles of the blood are lessened in amount at the end of a febrile disease; and of the various tissues none appear to waste so fast as the muscles, and especially the involuntary ones (*e. g.*, the heart in typhus

fever). The fixed albumen of the muscles, brain, and nerves breaks down into circulating albumen, to be in its turn transformed into urea and other nitrogenous excreta. Hence the muscles waste and the brain becomes atrophied. On microscopic examination the muscular fibre can be seen in the act of disintegration. The fat of the body is also rapidly absorbed in fevers; and while it is known that much of the metamorphosis of these tissues takes place in the normal way, it is also probable that there is an unhealthy or perverted metamorphosis which leads to the appearance of compounds in the excretions, either altogether foreign to the body, or foreign in respect of place and time. There is evidence of this in the peculiar smell of the perspiration, in the peculiar colouring-matter of the urine, as well as in the occasional excretion by it of hippuric as well as lactic, valerianic, and other organic acids.

In determining the pathology of fever, it is of importance to know what molecular changes occur in the muscular tissue during the febrile state. In enteric fever—where the fever lasts so long, and in which, as a rule, the temperature is very excessive—observations are of recent date, and the literature is already voluminous. The following may be recorded:—(1.) Virchow observed, especially, in the *rectus abdominis* what he describes as opaque or cloudy swelling (parenchymatous inflammation, see p. 64, *ante*.) This process has been noticed frequently in febrile diseases generally. He mentions, also, increase of the number of nuclei, and the cleaving or splitting up of muscular substance into homogeneous clods or lumps. (2.) Zenker calls the change waxy degeneration, and denies its inflammatory character. He contends that it forms the highest stage of granular metamorphosis of muscular tissue; that in this purely degenerative process, some muscular substance, turning waxy, includes numerous nuclei, and forms then lifeless lumps. He denies any new formation-process in the muscular fibres. (3.) Waldeyer has shown not only the existence of passive metamorphosis (waxy and granular) in the muscles of enteric cases, but also an abundant increase of nuclei, to such a degree that new tubes or sacs are formed completely filled with nuclei or cells. He did not sufficiently investigate the relation between the increase of nuclei and the waxy formations, therefore his objections to Zenker's theory are not of much weight. Yet Zenker's theory meets with contradictory facts—*e.g.*, the process of waxy formations in muscles has been seen in cases where a very evident new formation of cell-elements in the muscular tissue exists—*e.g.*, in traumatic inflammation of muscles, in trichinosis and similar processes (BENNETT, VIRCHOW, BILROTH). (4.) Popoff, under Virchow's supervision, undertook to clear up these contradictions. In twenty-eight cases of infectious disease—namely, 8 of *Fever recurrent*, 6 *Enteric*, 5 *Typhus*, 6 *Puerperal*, 4 *Cholera*, 1 *Pyæmia*, 2 *Hydrophobia*—he examined the *rectus abdominis*, the *diaphragm*, *pectoral* muscles, and *adductors* of thigh. Sections were made—(a.) in recent state, (b.) after hardening in Muller's fluid (25 grammes *bichrom. potass.*, dissolved in 1000 cc. of water, in which 10 grammes of *sodium sulphate* have been also dissolved), carmine tinging, and preservation in glycerine. The changes he found most extensive in the *diaphragm*, consisting of granular, waxy, and parenchymatous inflammation (cloudy swelling of Virchow). Frequently he observed an abundant production

of nuclei. All these changes were present in enteric cases; and in other cases they were frequently to be noticed either singly or combined, without any rule or significance as to any disease in particular. The waxy lumps were transparent as the muscle itself—therefore not a degeneration. He noted also the presence of extravasation and hæmorrhage in the muscles, showing an active participation of the vascular system. The walls of the blood-vessels were frequently found in a state of inflammation—the endothelium thickened, the cell-elements swollen, and the waxy and parenchymatous degeneration very trifling (Dr. LEO POPOFF, in VIRCHOW'S *Archives*, Vol. 61, Part 3, p. 322).

II. *Of the facts which relate to the general disturbance of bodily functions, of which the disorder of temperature is the chief, expressed through—(1.) the production, and (2.) the discharge of heat in fever; and next, the retention of water, the condition of the urine, the blood, and the nervous system.*

The constancy of animal temperature depends on the existence of heat equilibrium—i.e., on the processes by which heat is produced and those by which it is liberated, balancing each other. Increased heat production is not a sufficient cause for high temperature of the body. Heat, like carbonic acid, urea, and water, are each products of chemical work done in the living body. To determine whether or not its production is increased or diminished, continuous measurement must be had recourse to. There are two methods, namely:—(1.) To estimate thermogenesis from what is known as to quantity and “heat value” of the material daily and hourly consumed in the body; or (2.) To measure the quantity of heat daily or hourly discharged from the body—this quantity being, if the temperature is constant, identical with the quantity produced. Heat value of food taken and measurements of temperature ought to be determined simultaneously. The temperature of the body is, in fact, the result of the opposing action of two factors:—1st. Development of heat from the chemical changes of the food, and by the conversion of mechanical force into heat, or by direct absorption from without; and, 2nd, and opposed to this, evaporation from the surface of the body, which regulates internal heat (PARKES' *Hygiène*, p. 432).

But with regard to the clinical investigation of fever cases, no means of observation have been so exactly discriminative in distinguishing one disease from another when fever is an essential element, as accurate observation on the temperature of the patient determined by the thermometer. In acknowledging this great fact, it is important to observe that the absence of such exact observation, and the trusting to general signs alone, have hitherto led to great confusion—a confusion which has been unfortunately increased by a pernicious system—still too common—of naming “fevers” from the place or locality where supposed varieties of fever have prevailed as epidemics; and by the use of local or provincial native names. For example, the Walcheren Fever, Levant Fever, Mediterranean Fever, Crimean Fever, Bulam Fever, African Fever, Fernando Po Fever, Lisbon Fever, Bengal Fever, Pucca Fever of India, Gall-sickness of the Netherlands, Hong-Kong Fever, and other names not less barbarous, may be quoted. Except as matter of history, and as beacons to warn us from a great danger to science, let these and

such-like names be consigned to oblivion. With the exact means at the disposal of the physician as aids to diagnosis (which are about to be described), every variety of illness where fever takes a part may be accurately distinguished, its type recognised, and its place fixed in nosology; or, if it should be anomalous, its exact departure from the type may be not less accurately defined and described.

The phenomena which thus call for special clinical investigation are those which are strictly related to the origin, development, and progress of the febrile state. Most of the febrile diseases commence with *shivers* or *rigors*—a symptom which, when fully expressed, is always of great importance. This *rigor* or shivering may be so severe as to induce chattering of the teeth, and to shake the bed on which the patient lies. On the other hand, it may be so slight as merely to induce paleness of the face and skin generally, with a feeling of coldness, or as if cold water were running down the back. In children the shiver or rigor may be represented by convulsions. Such shivers mark, as a rule, the commencement of an illness more or less severe. Hence it is always of importance to find out when the sensation of *chill*, *shiver*, or *rigor* was first experienced, in order to *fix the date of commencement of the illness*. It may be also here observed, that like other secretions, such as urea, the discharge of heat may be diminished or abnormally retained, rather than increased, as in the first stage of fever. The facts afterwards to be ascertained are not less significant of the abatement, subsidence, or “*defervescence*” of the febrile state, than of the advent of local lesions, which may be constant, as part of the disease, or merely an accidental complication. The term “*defervescence*,” in fever, signifies the period during which the temperature of the febrile body is declining to its normal amount from that intense degree of heat attained during the accession of the febrile phenomena. This “*defervescence*” may be sudden, when it is regarded as a “*crisis*,” or it may be gradual, and is then described as a “*lysis*”—the “insensible resolution” of the older authors; or it may be partly sudden and then slow, when it may be described as “*wave-like*,” with gradual and sometimes regular alternations of high and low temperature. More than one hundred and twenty years ago (1754), Antonius de Haen (the first teacher of clinical medicine in the Hospital of Vienna) impressed his pupils with the necessity of attending to the temperature of the body in disease, *as measured by a thermometer*, instead of being estimated merely by the sensation of heat imparted to the hand laid on the skin of the patient. He showed that even in the cold stage of ague, with the teeth chattering and the body shivering, the temperature of the blood is rapidly rising, although the pallid skin may really be colder than usual—its supply of blood being diminished by the contraction of the blood-vessels. He first demonstrated with *measured accuracy* how much the heat of the blood, and therefore of the body, is augmented under the influence of the febrile state. When the crude appliances and the rough instruments of days long ago are compared with the delicacy and refinements of “the instruments of precision” of the present day, it may be of interest to observe, how the progress of knowledge and the powers of modern research have not suffered the valuable pathological lessons to be lost sight of, which are to be learned from the clinical use of the thermometer, as De Haen taught in his

time. But the determination of the amount of heat in fever cases is stamped by a much more early appreciation of its importance and value than even the time of De Haen; for, ever since the days of Hippocrates, the Physician and the Surgeon have been in the habit of applying the hand to the skin of the patient, to appreciate the presence of abnormal heat. When the hand of the physician alone is used to judge of the temperature of a patient, or when the feelings of the patient are alone taken as a measure of his temperature, it can easily be understood how such kinds of observation are extremely fallacious, doubtful, and unsatisfactory. But the practical application of the thermometer in place of the hand, while it is obviously a more accurate method, has been long in coming into general use, mainly on account of the difficulty of getting instruments sufficiently sensitive and trustworthy—instruments, in fact, of sufficient precision. The time and trouble required to work with crude and inefficient instruments soon brought them into disuse and discredit; but now the instruments required may be obtained so delicate and accurate, and the time taken to apply them is so insignificant, that we have no excuse for neglecting to use them. When it is remembered also that Galen's definition of fever is still the one whose accuracy remains not only unimpeachable, but fully demonstrated and recognised; that it describes fever to consist in "a preternatural heat,"—it is obviously essential that we should be able to determine the temperature, and so learn the significance of such increase of temperature in every case of disease where fever may be present. The careful physician *counts the pulse and the respirations* in all cases of illness; it is not less incumbent on him *to determine the exact temperature*. By means of a delicate thermometer he has in every case of fever an accurate measurer of its amount; and the student of medicine, as one of the earliest clinical lessons in hospital wards, should be taught to look to the excreta, and to the various physiological conditions of the patient, for the products of the metamorphosis of tissue which are more or less equivalent to the amount of heat which he may measure by his clinical thermometer in each disease. Ever since the publication of the second edition of this work, when the thermometer as a means of determining the temperature in cases of fever was first expounded in any English text-book for students, I have been gratified to find that the importance of this instrument in the diagnosis and prognosis of disease, has become more and more apparent, and its use more and more universal. There is ample evidence of this in the contributions to the literature of this subject since the second edition of this text-book appeared. The thermometer is now as much a necessity, and ought to be as much a companion, to the medical man as the stethoscope.

The thermometry of disease is practically shown to be important from two points of view, inasmuch as,—(1.) *The continuous daily use of the thermometer greatly facilitates the clinical recognition of diseases, and aids us in acquiring an accurate knowledge of their varieties.* It aids the busy practitioner in coming to more certain and safe conclusions than heretofore; and so relieves him of much anxiety of mind in doubtful cases. (2.) *The use of the thermometer tends to elucidate the course, tendencies, and results—in short, THE NATURAL HISTORY—of all diseases where fever is present.*

Animal heat has been determined in two ways—namely, either by the ordinary mercurial thermometer, or by the thermo-electric apparatus. The latter may indicate fractions of a degree, and in this respect surpasses the powers of the most delicate mercurial thermometers. MM. Becquerel and Breschet employed such an apparatus to determine the temperature of internal parts. But notwithstanding the greater delicacy of the *thermo-electric apparatus*, a sensitive mercurial thermometer, finely graduated and compared with a standard one, is the only instrument of practical usefulness, as yet, for ordinary clinical purposes—and for obvious reasons.

It is necessary to have an instrument which will determine the temperature in the mouth, axillæ, rectum, or other parts of the body. Such an instrument is named a "*clinical thermometer*;" and it must be of unquestionable veracity. If the instrument is not absolutely accurate, its errors require to be known. Whatever thermometer is used, it is necessary to compare it with a standard one, and note the differences *between every degree*. Differences in the diameter of the bore (in calibration), throughout the entire length of the thermometer, seem to be almost unavoidable, as yet, in their manufacture. The only safe rule, therefore, is that each instrument ought to be sent to Kew for verification, to be returned to the owner with a certificate containing the corrections for its several readings, if any are requisite. A fee of half-a-crown is charged at Kew for this certificate of verification; and if the makers would do this, and charge the fee to the price of the instrument, it would save a great deal of trouble. A thermometer is bad, and all but useless, if the differences between various degrees are unequal; but it is quite serviceable if the same sum is to be added or subtracted for *each* degree. The price of such an instrument need not now render it difficult nor expensive for a student to acquire a competent practical knowledge of "the thermometry of disease."*

A good clinical thermometer ought to have a uniform and correct scale, having a range from 90° to 112° Fahr., exhibiting fifths Fahr. of degrees, one-fifth of an inch apart from each other. It ought to be a sensitive mercurial maximum self-registering one (known as "Phillip's Maximum"); which does not require to be read *in situ*, but may be removed from contact with the part, and read when convenient. The bulb of mercury should be as thick as the diameter of the stem, and not more, so as to expose a sufficiently large surface to the part of the body whose temperature is to be determined. *This Straight Thermometer*, which is self-registering, *must have its index set before* commencing to take an observation. The index is the bit of mercury detached from THE COLUMN IN THE STEM OF THE INSTRUMENT. This index is to be set by bringing the bit of detached mercury down into the clear part of the stem, just below the lines which indicate the degrees. This is done by taking the bulb and stem of the instrument firmly in the hand, and then by *a single rapid swing of the arm* the index will come down the stem; and this *swing*

* I have found the ingenious arrangement by Giessler of Bonn, the best and most lasting form of clinical thermometer; while Cassella of Holborn Bars, Hawksley of Blenheim street, London; Harvey and Reynolds of Leeds, are most careful makers of clinical thermometers in this country.

of the arm must be repeated till the top of the index is at least below the lines which indicate the degrees. After the index has thus been set, the bulb of the instrument is then to be applied to the skin in the axilla, or between the thighs, or any part which is completely covered; and being retained in close apposition with the surrounding soft parts for a sufficient length of time, the instrument is to be carefully and gently removed, when the *top of the index*—i.e., the end farthest from the bulb—will denote the maximum temperature during the period the instrument has been in perfect contact with the parts. The patient should have been at perfect rest in bed for at least *one hour* before observations on temperature are made, and he ought to lie on the side, so as to close completely the axillary space which is the seat of the thermometer, converting it into a close cavity. The observations ought to be continuous daily, and regularly taken at the same hour every day, throughout the whole period of sickness. The most useful periods for observation are—(1.) Between seven and nine o'clock in the morning; (2.) At noon; (3.) Between five and seven o'clock in the evening; (4.) At midnight. For most practical purposes it is sufficient to note the temperature twice daily,—morning and evening, with an occasional observation at midnight. In all observations of temperature the pulse and the respirations should be noted at the same time. In less important cases, the physician may make at least one observation daily himself, and leave the others to the friends of the patient or the nurse, if either of them are sufficiently intelligent. This arrangement, however, is only justifiable so long as the observations correspond with those typical of the particular disease, and so long as they are in harmony with the other general signs of its course; but as soon as notable deviations from these conditions are observed, the physician ought to make the observations for himself. A difference of 2° Fahr. is not of any practical importance unless it is persistent. In prolonged and severe cases an examination of the records of temperature made during the course of the disease will recall to mind the nature of the case more effectively than the most detailed written history. For this purpose it is desirable to exhibit on paper the daily thermometric changes, in the form of an angular line or a curve, and to note in the proper places short memoranda of the more important incidents or therapeutic events which have taken place during the progress of the disease. Details illustrative of the changes in the pulse and the respiration and amount of excreta, ought to be entered in the same sheet, as exhibited on the next two pages. In chronic cases, when febrile attacks and their concomitant dangers may be expected, as well as in acute cases, after return of the normal blood-heat, *one* daily observation will be found sufficient. This single observation may be best made in the afternoon, evening, or midnight. It is advisable also to induce nurses, friends, or other attendants on the sick (whenever they seem apt pupils), who may make notes of any considerable excitement or restlessness, or take notice of hot hands, or increased heat of head, rather to consult at once the thermometer than trust to their sensations. They may thus, perhaps, tranquillise the patient and his friends when the instrument does not indicate any material increase of heat; but the sudden appearance of any considerable increase of temperature should always be regarded as a fact of vital importance.

It has been recommended by some to place the thermometer under the tongue; but the cavity of the mouth is the worst place in which the thermometer can be put in patients not accustomed to such investigations, because the temperature there is continually varying according to the quantity and temperature of the air used in respiration; and if the atmosphere is cold, and deep inspirations are made, large differences may be observed, compared with the temperature in the axilla. Observations made with the thermometer in the mouth are therefore not generally trustworthy, unless verified by observations in the axilla or rectum. *Three to five minutes* are found quite sufficient for the application of the thermometer, if certain precautions are taken. The simplest and most convenient way is to heat the instrument before inserting it into the patient's axilla, just as the surgeon heats the catheter before he introduces it into the urethra. Neglect of this precaution is apt to lead to an *under-statement* of temperature. It may be heated by holding the thermometer in the warm hand till the mercury shows a temperature of 98° ; and after the instrument is properly placed, be satisfied if *two observations at intervals of one to two minutes give exactly the same result*. The rapidity with which the mercurial column rises depends on the degree of temperature present; hence the rapidity of the rise of temperature ought to be noted, as well as the maximum height. If the temperature be above the normal standard, a sensitive thermometer will indicate that fact within the first minute; and as the quickness of the rising depends upon the existing temperature, the physician is able, after some experience in the use of a particular instrument, to form an approximative judgment of the amount of rising of temperature to be expected in any particular case from the slowness or rapidity of the rise of the mercury after half a minute.

It is necessary to know *the Fluctuations of Temperature within the limits of HEALTH; and the correlation of the Animal Heat with the Pulse and the Respiration*. Several observers in Germany, France, England, and the tropics, have now determined these fluctuations with great accuracy, so that ample and sufficient data are on record to furnish a standard for comparison in cases of disease. With reference to the normal range of temperature, our most trustworthy information is mainly due to Valentin, Traube, and Jürgensen, in Germany; to Edwards, Becquerel, Breschet, and Bernard in France; to Dr. William Ogle, in England; to Dr. Alexander Rattray, Surgeon in the Royal Navy, and to Dr. Edward Laurie, in India, as regards natives and Europeans in tropical and temperate climates. It is generally agreed that the ranges of temperature vary in different parts of the human body; but, as a general practical result, it is equally agreed that in temperate regions the normal temperature, at *completely sheltered parts of the surface* of the human body, amounts to 98.4° to 99° Fahr. in the *rectum*, or a few tenths more or less at different times of the day; and a rising above 99.5° , or a depression below 97.3° Fahr., are sure signs of *some kind of disease, if the increase or depression is persistent*. Dr. Edward Laurie's observations show that there is no difference between the temperature of healthy natives and Europeans in India—namely, 98.3° or 98.4° in the morning, at 6 A.M., with a rise of from 1° to 2° during the day. Valentin proved by many experiments that all warm-blooded animals surrounded by an atmosphere

of 50° to 68° Fahr. have a temperature of about 99·5° in the back of the mouth, the rectum, or other accessible *internal parts*; and at *completely sheltered parts of the surface* it is about 97·5° to 98·4° or 98·5°. According to Ringer and Stewart, in persons under twenty-five, the average maximum temperature is 99·1°; over forty years of age it is 98·8°; in the second childhood of old age it again rises (ALBUTT).

Dr. Rattray's observations were made by placing an ordinary Fahrenheit thermometer under the tongue thrice a day during a voyage from England to Bahia (lat. 11° S.) and back —*i. e.* across the equator—extending over sixty days (fifty-three in the tropics, and seven in the latitude of England, 51° N.) During this period the temperature of the air in the shade on the verge of the tropics was 72° Fahr.; at the equator, 84° Fahr.; and the average of the tropics generally, 76·9° Fahr. The atmospheric humidity ranged from 0° to 7·5°; the average being 3·8° of a Mason's hygrometer. In England, with a temperature ranging from 60° to 70° Fahr., the average temperature of the body was 98·3° Fahr.; it rose in the tropics to 98·6°, and in the equatorial doldrums to 99° Fahr., and occasionally even to 100° Fahr. In the tropics the temperature of the body is greatest during the afternoon, when the sun is high and the body most active, and least in the morning. The pulse is likewise highest and lowest at these times. The totals show 99° degrees Fahr. to be the most frequent bodily temperature, while 99·5° and 100° Fahr. form 22 per cent. of all the observations, and the range of temperature about 2° Fahr. daily (*Proceedings of Royal Society*, No. 122, p. 513, June 16, 1870.)

The following records are also believed to be trustworthy:—The *minimum temperature* is from 1.30 A.M. to 7.30 A.M. (JURGENSEN.) The lowest temperature occurs about daybreak. At this time a rise begins, which continues till late in the afternoon, and commences while yet the body is in complete repose, and when no food has been taken for ten or twelve hours; but coincidently with this rise there is an increase in the exhalation of carbonic acid (OGLE) and of urea; so that the rise is doubtless due to increased chemical change or resumed activity of organic functions, as the intensity of sleep diminishes towards morning. The *highest range of daily temperature* is maintained between 9 A.M. and 6 P.M. After this time the temperature falls slowly and continuously, if no alcohol be taken; but if alcohol be taken the fall is more sudden (OGLE). Otherwise food seems to have little influence on normal temperature, except, perhaps, with infants. Although the records may vary somewhat, yet one elementary fact stands clearly out—namely, that in a healthy man the limits of fluctuation, under various conditions, are very narrow, and independent of external temperature. This constancy of the bodily temperature is a consequence of the remarkable regulation of evolution and loss of its heat. The average variation in the course of twenty-four hours is about 1·5° Fahr. Dogs have a temperature similar to that of men. A knowledge of thermometry, therefore, in the diseases of animals, will prove not less valuable in veterinary pathology than in human, perhaps more so, inasmuch as animals being deprived of speech, are unable to express their feeling. In the cattle plague of 1866-67, the use of the thermometer was found of great practical value in diagnosis.

The following collateral circumstances mainly influence animal heat in our daily life; and require to be remembered, in order that erroneous conclusions may not be drawn from thermometric observations in disease:—(1.) *Active exercise* (not carried to the extent of exhausting fatigue) raises the temperature proportionally to the degree of muscular exertion made. (2.) *Exposure to cold* without exercise lowers the temperature. (3.) The amount of heat is also at first reduced after a *full meal* and after alcohol; but it rises again as digestion advances. According to Dr. William Ogle, the rise is most marked after breakfast, less so after lunch, and the reduction of heat after dinner is a mere retardation of the fall which without it would occur. (5.) Alcohol (claret) causes an immediate rapid fall, which is temporary, and a reaction occurs by which the temperature is carried to as high a point, or even higher, than it would have reached if no alcohol had been taken. Tea causes an elevation of temperature. (6.) Sex, race, latitude, seasons, weather, habits of life, and idiosyncrasies go for very little in influencing the temperature of *healthy persons*. (7.) On the other hand, the temperature in disease is more readily and rapidly affected—more sensitive, so to speak—than either the pulse or the respiration, and the increase or variations are presistent as long as disease exists. Hence the very great importance of noting the temperature in all diseases. The amount of abnormal increase of temperature is usually proportionate to the degree of frequency of the pulse, and to the other signs of general disease. Yet such congruity of phenomena is sometimes in part or wholly absent or incomplete; and in the cases in which a disproportion or incongruity exists between the increase of temperature and the pulse, or other febrile phenomena, it is the accurate measurement of the temperature which is most of all to be relied upon. As a general rule, the correlation of pulse and temperature may be stated as follows, namely:—*An increase of temperature of ONE DEGREE above 98° Fahr. corresponds with an increase of about EIGHT beats of the pulse per minute, as represented in the following table:—*

A temperature of 98° . .	Corresponding with a pulse of	72 (Health)
— 99° . .	Ought to correspond with a pulse of	80
— 100° . .	—	88
— 101° . .	—	96
— 102° . .	—	108
— 103° . .	—	112
— 104° . .	—	120
— 105° . .	—	128
— 106° . .	—	136

This statement is, however, in some respects arbitrary, and is rather given for the convenience of comparing different diseases with some standard than as always accurate. In some diseases a high temperature is found with a low pulse, and a low temperature with a high or rising pulse. The pulse, too, sometimes rises in rapidity when the temperature falls, or falls when the temperature rises. Dr. John Beddoe, Physician to the Bristol Royal Infirmary, records a peculiar case of enteric fever, in which the most notable point was the co-existence of an elevated temperature with an abnormally slow pulse, and but a moderate rate of wasting.

In children the records of temperature are contradictory. For example:—

According to the observations of M. Roger (*Archiv. Gen. de Medicine*, July, 1844) and Dr. Holland, the temperature of children is somewhat higher than adults, when placed in conditions favourable to sustenance. At birth the temperature of the infant is the same as that of the mother, but quickly falls to $93\cdot4^{\circ}$ or $95\cdot5^{\circ}$, rising in the course of twenty-four hours to $97\cdot7^{\circ}$ —*i.e.*, more than *half* a degree below adult heat (MACLAGAN). Between four and six years of age, M. Roger found the temperature to be $98\cdot9^{\circ}$ Fahr.; and between six and fourteen years, $99\cdot16^{\circ}$ (CARPENTER). Dr. Bennett states generally, that in children the heat of the body is about 2° higher than in adults. On the other hand, among numerous written statements sent me on this subject from actual observation, I find results are varied, and many are opposed to the preceding statements. For example, during an epidemic of measles in Glasgow, in 1866, Mr. James P. Cassels, while using the clinical thermometer, was much struck with the low temperature recorded in some cases after complete recovery. But it is important to remember that during recovery from all febrile diseases (and measles is no exception) there is a tendency to lowering of the temperature below the normal standard. The result of six observations on a baby, $16\frac{1}{2}$ months old, and in perfect health since birth, *taken when asleep*, and every source of error carefully avoided, shows temperatures *below* those of adult life, and the evening temperatures almost invariably lower than the morning ones. Dr. James Finlayson, when House Surgeon to the Hospital and Dispensary for Children at Manchester, made an extensive series of observations which confirm these general conclusions (*Glasgow Med. Journal*, 1869).

Having satisfied ourselves as to the delicacy and accuracy of the thermometer, and obtained a standard for comparison, we are prepared to appreciate, by measurement, the ranges of temperature in febrile disease. The maintenance of a normal temperature, within the limited fluctuations just noticed, under every varying influence, gives a complete assurance of the absence of anything beyond local and unimportant disturbances; and, long before the subject was worked out so thoroughly as it has been, it was often casually observed that any acute disease, however slight, elevates abnormally the temperature or animal heat; "and its undue degree of elevation is some criterion of the intensity of the diseased action" (as Dr. Davy clearly enunciated, *Phys. Researches*, Vol. I., p. 56). In short, it is now placed beyond a doubt, that while this preternatural heat varies in amount in different diseases in different persons, and at different times of the same day, it is this preternatural heat which is the essential symptom in fever, which proves fever to be present, and which exists to the extent of 4° , 6° , or even 8° Fahr. over the natural limits of health, and must be estimated by the temperature in the axilla or rectum, as indicated by the thermometer. This preternatural heat is never absent in fever, and without it fever cannot be said to exist. Rigor, which is also sometimes present, is a mere peripheric phenomenon. The coldness of the skin, so much complained of by the patient, is usually a subjective sensation, produced by the state of the peripheral nerves, and is not due to any actual decline of temperature; for even "while the outer parts feel cold to the bystander, the inner parts are abnormally warm. While the outer parts freeze, the inner burn" (VIRCHOW, PARKES, JENNER). But whether it is the production or the discharge of heat which is primarily disordered is still a question—the

former is the function of living protoplasm, the latter a function of the organs of circulation, respiration, and secretion.

There are many cases now on record in which the physician, without thermometric observation, does not appreciate the existence of fever or of danger. Wunderlich gives numerous examples of this; but long before he brought this subject so forcibly to the notice of medical men, we have the testimony of Dr. John Davy in this country, given quite incidentally, and therefore all the more valuable as an unbiassed testimony, of the usefulness of the thermometer in detecting latent disease not otherwise indicated by general symptoms. When Dr. Davy was collecting his extensive observations on the normal temperature of the body, he was surprised to find that one person exhibited for many weeks a persistent temperature of 104° Fahr. This person was a lunatic soldier; and Dr. Davy remembered that the insane do not seem to suffer from cold nor heat like ordinary individuals, and that there are certain organic lesions which are apt to occur in them unaccompanied by the usual symptoms. For example, tubercle and cavities of the lungs occur without cough or difficult breathing; and although no warning nor any indication may be given, the disease runs its course, terminating in death as certainly and as rapidly as if indicated by the ordinary train of symptoms. Discovering, then, as it were by accident, that the temperature in this lunatic was as high as 104.5° Fahr., and that his pulse was rapid, Dr. Davy's attention was more particularly aroused; but although the man made no complaint, and had a good appetite, with his digestive functions, so far as was known, acting well, yet disease of the lungs was thus discovered, and was confirmed by the examination of his chest yielding the usual physical signs of disease. The lunatic died in a month of acute tuberculosis, not otherwise expressed by symptoms beyond the great, persistent, and continuous elevation of temperature thus incidentally noticed. There were ulcers of the *larynx* found after death, but there had been no affection of the voice; there were *romicæ* and *tubercles* in the *lungs*, but there had been no cough; there were *ulcerations* of the *intestines*, but there had been no diarrhoea; there was disease of the *testes*, *vesiculæ seminales*, and *prostate*, of a severe kind, but these lesions had been equally latent during life, except hardening and enlargement of the testicle with pain,—all which conditions were only casually observed. In this very instructive case a temperature of *six degrees* Fahr. above the normal standard was the earliest indication of disease (*Researches Physiological and Anatomical*, Vol. I., p. 203).

But it is mainly to the late Professor Wunderlich, of Leipsic, that we are indebted for an elaborate exposition and persevering advocacy of the usefulness of daily records of the temperature of fever patients, and the constant employment of the thermometer as a means of diagnosis at the bedside. When the physician once becomes accustomed to the investigation of disease by the thermometer, he regards its daily employment as indispensable, for it imparts a certainty to his observations, attainable by no natural penetration, and which no other method of investigation can convey. Dr. Murchison (our greatest authority on continued fevers) considers that the "utility of this instrument for distinguishing different forms of fever has been greatly overrated; but for deciding as to the presence or the increasing or declining severity of fever, it is invaluable." Wunderlich

gives some striking instances of disease being indicated by thermometric observation before it could be detected by any other means:—*In ague*, several hours previous to the paroxysm, the temperature of the trunk of the patient's body begins to rise; and when the disease *seems* to have disappeared, an increase of temperature may be detected periodically unaccompanied by any other symptom. So long as this periodic rise of temperature continues, the patient is only apparently, but not really cured. *In enteric fever*, during the exacerbations especially, the rise of temperature or its abnormal fall may indicate what is about to happen three or even four days before any change in the pulse, or other sign of mischief, has been observed. A sudden and marked reduction of temperature has thus denoted hæmorrhage from the slough of Peyer's patches in typhoid fever several days before it appeared in the stools. A case of this kind is recorded by Dr. Parkes. It occurred in a female twenty-five years of age. Diarrhœa was considerable, and blood was largely passed in fluid stools the night before the seventeenth day of the fever. On the morning of that day the temperature was as low as 93° Fahr., rising in the evening to 101° Fahr. It is by *continuous daily observations* that the most important results have been arrived at. Certain febrile diseases have thus been found to have typical ranges or daily fluctuations of temperature throughout their course. In pure unmixed and uncomplicated cases this is found to be so constant, that the differential diagnosis may be established by accurate observation of the temperature continuously from day to day. This has now been determined, especially in cases of malarious fever, typhus, enteric fever, small-pox, scarlatina, measles, rheumatism, pyæmia, pneumonia, acute tuberculosis. In each of these diseases the temperature is one of the most certain (although not the only) means for determining the real state of the patient as regards morbid disturbances or complications; and a careful observation of temperature from day to day, considered in relation with other signs, is indispensable for judging as to the prognosis. Frequently it affords the only ultimate means of deciding in doubtful cases, and often it is the best corrective of a too hasty conclusion; for example, the characteristic variations of the temperature, in a typical case of enteric fever, are of such a kind that they are not found in any other disease. Intestinal catarrh, severe forms of pneumonia, malarious fever, acute tuberculosis, meningitis, some stages of Bright's disease, may each simulate enteric fever, and may exhibit some of its most characteristic symptoms; but observation with the thermometer as to the patient's temperature from day to day, will at once, or after a very few days at most, establish the distinction with certainty.

A remarkable inequality in the distribution of the temperature over different parts of the body (face, hands, feet, &c.) may occur during the shivering preceding fever, in collapse, and in the agony of approaching dissolution. Sometimes, also, such unequal distribution may occur in disorders of the chest and abdomen, in some local skin diseases, and in partial paralysis, apoplexy, and injuries to the nervous centres, as in Mr. Teale's case (about to be noticed), which cannot be regarded as similar to that of febrile pyrexia. This fact is not of importance or utility for diagnosis or prognosis; but it requires to be known, in order that erroneous conclusions may not be drawn.

When it is found, in a bad case of *enteric fever*, that some morning about the third week the temperature has fallen to 99.5° , the reparative stage has begun—the healing of Peyer's patches; and when a similar fall of temperature is observed in the evening, convalescence has commenced. In *pneumonia*, when a marked fall of temperature occurs in the evening, the period of crisis has arrived. In *measles*, when the maximum severity of the eruptive stage has been reached, the temperature falls. A sinking from a considerable height down to a normal temperature suddenly (within twenty-four hours), occurs in a few *eruptive fevers*,—*measles*, *variola*, rarely in *pneumonia*, *typhus*, and *pyæmia*. In *scrofula*, especially in its acute form, with deposition or growth of *tubercle*, the persistent maintenance of an uniformly high temperature will alone show that no arrest in the progress of the disease has occurred.

The correlation of pulse, respiration, and temperature is of importance to be determined in many acute diseases. In *pneumonia*, if the mean of the temperature is not above 104° Fahr., and that of the pulse is not above 120 in a minute, and the mean of the respirations not over 40 in the same time, the case must be considered a slight one; and if the patient is otherwise healthy, he will surely begin to get well in from eight to twelve days, without any medical treatment beyond attention to antiphlogistic regimen. In *typhus fever*, a falling temperature with a rising pulse forbodes danger. *Convalescence* is known to commence when the disease-process ends; and this precise point can only be fixed by continuous thermometric observation. The morbid process does not end till the normal temperature of the body returns, and maintains itself unchanged through all periods of the day and night. Regularly continuous observations of the temperature exhibit the precise point at which the disease-process terminates, and the degree of its complete development. When this point has been determined on, a retrospective view may be taken of the character of the disease, as to the purity of its typical form or its complexity, and a prognosis may be hazarded as to the probability or doubtfulness of recovery. The morbid process has not terminated till the normal temperature of the body returns, and remains unchanged in the evenings and throughout all periods of the day. The transition from the febrile state into defervescence being either slow (lysis) or rapid (crisis); and regularly continuous defervescence is always a sure sign of convalescence. Irregular defervescence, on the contrary, indicates a disturbed and protracted course of convalescence, which requires careful watching and judicious nursing. It is of practical importance to know that the fall of temperature during the period of recovery, in cases of considerable morning remissions, as well as in those of continued defervescence, may be abnormally large, and may sink as low as 28° R. = 95° Fahr., or even lower. Such events constitute collapses during defervescence, which must be counteracted by artificial heat, the administration of warm drinks, or even of such stimulants as wine or camphor, unless some unexpected new danger should interfere with an otherwise favourable course of the disease. During convalescence the recurrence of a high temperature is generally the first sign of an approaching relapse, or the onset of a new disease, the characteristic symptoms of which it may precede by several days. The persistence

of even an inconsiderable degree of abnormal temperature after apparent return to health, is a certain, and frequently for a long time the only, sign of incomplete recovery, or the existence of some lingering secondary disease. The temperature should therefore be closely watched during convalescence; and the thermometer should be applied every alternate evening at the very least. As long as the temperature remains normal, nothing need be feared; but every rise of temperature should act as a warning. It may be due to mere error in diet, or to leaving bed too early; but in such cases the temperature soon sinks again, on greater precautions being taken.

Regularly continuous observations on the temperature alone, or in connection with other symptoms, may enable the physician to predict a fatal issue with certainty, or the probably near approach of death. On this point some qualifying statements are necessary: (1.) The temperature may rise continuously and considerably above $106\cdot2^{\circ}$ Fahr., when it is generally a bad sign; or it may even reach 110° Fahr., when a fatal issue is almost certain; and it not unfrequently happens that, after the apparent occurrence of death, the temperature still continues to rise one or two-tenths Fahr., or even a degree, the cooling of the body taking place very slowly. Wunderlich records a case of spontaneous or rheumatic tetanus in which the temperature exceeded the maximum that has ever yet been observed in any disease. The heat only began to increase within the last twenty-four hours before death; but the other symptoms before that time had been very violent, the respirations being accelerated, and the pulse at 102. During the night previous to death the temperature suddenly rose $3\cdot3^{\circ}$ Fahr., while the velocity of the pulse and the frequency of the respirations diminished, and the other symptoms did not increase in severity. Shortly before death, the heat rose to $110\cdot75^{\circ}$ Fahr., the pulse being then at 180; and at the moment of death the thermometer was at $112\cdot5^{\circ}$ Fahr. After death the temperature still rose, and was found to be $113\cdot8^{\circ}$ Fahr. an hour after the fatal event. It then slowly diminished; and thirteen and a half hours after death the temperature had not yet fallen to the normal average of the living body. (2.) The temperature may become more or less moderated, while the pulse is increased in frequency, and the other symptoms become more and more threatening. Such diminution of temperature, amidst conditions which do not harmonise with it, must be regarded as a pretty certain sign of approaching dissolution (see cases published by Dr. Muller of Dundee, in *Brit. and For. Med. and Chir. Review*, Oct., 1868). (3.) A decrease of temperature *below* the normal is rare. It happens sometimes transitorily, announcing thereby a favourable crisis, by preceding the return to a normal temperature. It is also met with sometimes during the morning remission of *remittent fever*; also during the *apyrexia* of *intermittents*; in acute collapse, preceded or not by fever; in chronic wasting diseases; and sometimes, also, on the approach of death, especially in typhus fever, in which the cardiac symptoms have been dangerous, and in pneumonia. (4.) Exceptionally high temperatures occur in acute tetanus and in certain spinal injuries, and to the nervous centres—temperatures up to $111\cdot2^{\circ}$ Fahr.; and (as in a case reported by Dr. J. W. Teale, of Scarborough), 108° to 122° Fahr. at varying periods; those which occur during the process of dying in acute specific fevers—

as for instance, $110\cdot06^{\circ}$ Fahr., the maximum of temperature in cases of typhus; and at $107\cdot36^{\circ}$, the agony of death commences. No patient in typhus whose temperature has risen above $107\cdot15^{\circ}$ Fahr. (death line) has recovered. Shivering in the morning may be followed, in typhus, by a temperature of $106\cdot91^{\circ}$ without death; but a temperature in typhus of $106\cdot24^{\circ}$ in the morning is sure to be followed by death. At $106\cdot12^{\circ}$ Fahr., one half going above this temperature dies. Favourable cases range from $105\cdot8^{\circ}$ to $104\cdot66^{\circ}$ Fahr., as average maxima; and the average maxima of *morning* temperatures of the same class of cases, especially typhus fever, are from $105\cdot08^{\circ}$ to 104° Fahr.; (5.) Low temperatures are to be found in cases of autumn diarrhoea, with symptoms like typhus or typhoid, in which the temperature may be $97\cdot7^{\circ}$ Fahr.; and cases of cholera die whose temperature descends to this degree; and $97\cdot52^{\circ}$ to $93\cdot02^{\circ}$ Fahr. are ranges of minima temperature of cholera cases, $89\cdot0^{\circ}$ Fahr. being the lowest temperature observed in any case of cholera.

Opinions vary, however, as to the fatal influence of temperature alone. Liebermeister has declared that a temperature of $107\cdot6^{\circ}$ Fahr. is followed by speedy death; and according to Weihardt, a temperature of $108\cdot6^{\circ}$ causes the blood in the vessels to coagulate. But that the danger to life is not always the result of high temperature, is clear from the following observations:—In relapsing fever the temperature has been known to reach 106° or 107° Fahr., without any cerebral symptoms, and with a favourable result. Of eighty-nine cases of relapsing fever observed by the late Dr. Obermeier, at Berlin, the temperature rose in three to $107\cdot6^{\circ}$ Fahr., in six to $107\cdot7^{\circ}$, and in two to $108\cdot5^{\circ}$. In all these cases no special danger attributable to the high temperature could be discovered, nor even a single circumstance in which they differed from other cases. In seventy cases of relapsing fever at St. Petersburg, Zern found that the temperature in the axilla rose to $108\cdot5^{\circ}$ Fahr. in twelve, of whom only four died. Tennent, at Glasgow, found in many cases of relapsing fever the temperature as high as 107° Fahr., while in two it reached 108° Fahr.; yet these cases of high temperature were in no way different from others. In scarlatina also the temperature may reach 108° Fahr. without a fatal result. On the other hand, most severe cerebral symptoms and a fatal result may occur in typhus, although the temperature has at no time exceeded 103° or 104° Fahr.; while cerebral symptoms, indistinguishable from those of pyrexia, may result from disease of the kidneys, when the temperature is not above the normal standard (*Brit. Med. Journ.*, 4th Jan., 1868).

A distinction must also now be recognised between abnormal temperatures due to specific fevers—febrile pyrexia—and those high temperatures due to lesions of the nerve-centres. In illustration of this, we have—(1.) Sir Benjamin Brodie's case, in which inordinate elevation ensued upon crushing injury to the cervical cord; and, (2.) A case of great interest communicated by Mr. J. W. Teale, of Scarborough, to the Clinical Society of London, on Feb. 28, 1875, in which a temperature ranging from 108° to 122° Fahr., and upwards, had been maintained for a period of nearly nine weeks. The patient had been thrown from her horse, which rolled over her, inflicting serious injuries to the spine (about the region of sixth dorsal vertebra) and ribs, two of which were fractured. About two months

after the injury the temperature began to rise to the excessive heights mentioned above, the pulse at the same time rising to 120, and becoming small, thready, and at times scarcely perceptible. There was intense pain along the spine, relieved by hypodermic injections of morphia. Rapid emaciation ensued, and death from exhaustion seemed imminent. By the skilful use of mercury an improvement was brought about in the general symptoms, but the temperature remained as high as ever. These high readings of the thermometer ceased after a time to cause alarm, seeing that the patient had lived so many weeks with a temperature hitherto supposed to be incompatible with life; and when the temperature still ranged from 110° to 114° Fahr., flesh and power began to be regained rapidly (*Brit. Med. Journ.*, March 6, and July 10, 1875). On the 19th of June, 1878 (three years and nine months after the injury), Mr. J. W. Teale was good enough to write me to say that "his former patient still suffers a great deal of pain in her back at times, when her temperature rises somewhat sharply." On the other hand, there have been as notable cases of depression of temperature, to 81° Fahr. after the same kind of injury (Dr. FARQUHARSON); and in cases of poisoning with alcohol, in which temperatures in man of 76° and 75.5° Fahr., have been recorded (*Brit. Med. Journ.*, April 3, 1875). In the former set of cases with injury to the spinal cord, the explanation may be found in those experiments on animals in which the spinal cord has been divided. In such a condition, compared with that of a normal animal, the former has lost all power of regulating its heat according to external conditions.

In further consideration of the general disturbance of the bodily functions in fever, the following statements may be made relative (a.) to the retention of water in the system; (b.) to the condition of the urine; (c.) to the condition of the blood; and (d.) to the condition of the nervous system.

(a.) *The excessive retention of water in the febrile system* is another remarkable and constant phenomenon. Notwithstanding the large amount of water frequently taken to quench the extreme thirst, the quantity of the urine is lessened, and is even scantiest when the skin is driest. The "concentration of the urine which results, appears to be almost as good an index of the amount of fever as the temperature itself." The excretion of water by the skin is also, as a rule, diminished; and it is a well-known clinical fact that the skin is drier than usual in febrile affections. Very early in the febrile state the buccal mucous membrane also becomes sticky, and the amount of saliva diminishes; and the decrease in the quantity of the gastric fluid during fever has been proved by the well-known experiments of Beaumont on Alexis St. Martin. The intestinal juices, like the gastric, are also probably diminished, for the stimulus of food is taken away, constipation prevails, and the fæces are dry (PARKES). Notwithstanding these well determined clinical facts as regards men, Dr. Senator concludes that the discharge of water is very considerably increased as regards induced fever in animals, which he attributes to excess of urea in the blood acting diuretically (USTIMOVITCH); partly, also, to the state of the circulation, and (during the rigor) to the suppression of the exhalation of water from the skin. This retention of water in the system cannot at present be explained; but Dr. Parkes

suggested that it may possibly be due to the presence in the blood (or tissues generally) of some intermediate waste product of the febrile body, of some substance which (like gelatine) has a powerful attraction for water.

(b.) *The Urine in Fever.*—The general characters of this excretion proper to the febrile state are—deficiency of water, increase of solids, if they are not retained, and especially of the *urea*, the *uric*, the *sulphuric*, the *phosphoric*, and the *hippuric acids*. The *pigments* also are increased, pointing to the increased disintegration of blood-corpuscles, or of muscular tissue, of which it may be a measure of metamorphosis; and the *chloride of sodium* is diminished. The deep colour of febrile urine has usually been attributed to its concentration; but if febrile urine be diluted to the usual amount of fluid contained in healthy urine, it is still darker than normal urine. The colouring matter has been shown by Vogel to be increased sometimes fourfold, and it appears to contain more carbon than usual. This colouring matter in febrile urine is peculiar, and does not give any of the reactions of the bile-pigment. Another important fact connected with the chemistry of the urine in fever is the *augmentation of its free acidity*, as measured by its neutralisation with soda.

(c.) *The Blood in Fever.*—It is important to determine when the blood becomes affected in fever. It has been, and still is, a favourite opinion to refer the *origin* of fever to primary disease of the blood; and in almost all general (specific) diseases a *fever-making cause* appears to enter the blood; at least, writes Dr. Parkes, it may be proved to enter in several cases, and a strong analogical argument can be maintained as to its entrance in others. The fever-making cause also reproduces itself in the blood, or in some organs; and it is now generally admitted that the first action of the fever-producing cause is *on the blood*. The most interesting facts connected with the chemistry and other morbid conditions of the blood in fever are,—(1.) A diminution of the alkaline salts, as shown by Becquerel and Rodier in inflammation; (2.) A diminution of alkalinity of the serum, as shown by Cohn; (3.) A diminution of the albumen after the fever has lasted for some time, with a commensurate increase in the water of the serum; (4.) A diminution in the numbers of the red corpuscles of the blood; (5.) In certain specific fevers the presence of *uric acid* has been detected; for instance, in the fever of rheumatism, by Dr. Garrod; (6.) *Spirilla* were discovered in relapsing fever in 1872, when it appeared in Berlin, by the late Dr. Obermeier, during that epidemic. His first communication was to the Berlin Medical Society, on Feb. 26, 1873, reported in *Berlin Klin. Wochenschrift*, 1873, pp. 152 and 391. After his death from cholera in August of that year, Dr. Engel took up the subject, and several others. An abstract of their work is given by Dr. Sanderson in the *Public Health Reports*, New Series, No. III., for 1874, p. 41. Heydenreich also gives an account of the discovery (see abstract of his paper in *London Medical Record* for October, 1877). And Dr. Vandyke Carter has confirmed the discovery from his observations in India.

(d.) *The Nervous System* seems to play so important a part in fever that Virchow, in his definition, states that the essential phenomena *must have* their immediate cause in changes of the nervous system. It is very

difficult to substantiate this position, but the following general results prove the great influence of the nerves in febrile affections. Taken individually, they, no doubt, will impress different minds with different degrees of force, while, collectively, they cannot fail to furnish an argument in favour of the essential participation of the nervous system in the febrile state:— (1.) There is the generally received physiological law, that nerves regulate the metamorphosis of tissue and the production of heat, both of which are altered in fevers (HELMHOLTZ, LUDWIG, BERNARD). (2.) There are those experiments on the vagus nerve which bring about febrile phenomena, such as increased cardiac action, pulmonary congestion, anorexia, and nausea (BERNARD, PAVY). (3.) There are those arguments derived from the various symptoms which announce, accompany, or terminate fever. (a.) The remarkable depression, apathy, sense of exhaustion and debility, which usher in the febrile state. (b.) The shiverings, the contraction of the superficial vessels and of the skin. (c.) The increased rapidity of the heart's action, and the relaxation of the vessels, which soon follows the stage of contraction just noticed, or occurs without it. (d.) The congestion of the lungs. (e.) The periodicity of some of the phenomena of fever, and the occurrence of death or recovery on so-called critical days. (f.) The abnormal state of the secretions. (4.) The fearfully rapid death, which sometimes ensues in the early stage, from some unknown cause, may with justice be referred to profound nervous lesions; for there is great prostration, a galloping and early-failing pulse, and an excessively rapid respiration. (5.) The effect of certain remedies, such as quinine, upon periodical febrile phenomena.

The Prognosis in any case of pyrexia will of course be greatly influenced by its cause; but, irrespectively of the cause, there are certain conditions which must always be regarded as of serious import. These are summed up by Dr. Murchison as follow:—"1. In grave fevers the prognosis is worse in robust full-blooded persons with abundance of muscle and fat, than in those of spare habit, because in the former there is more material to spare for disintegration during the febrile process. In scarlatina it is a matter of daily observation that the plump and ruddy child succumbs, while the spare and more delicate-looking child pulls through. And in adults it may be said that all fevers are grave in proportion to the development of muscle and fat. 2. The prognosis is always bad when severe fevers attack individuals in whom the defæcation of the blood is naturally imperfect—for example, in persons of a gouty diathesis. 3. The danger in any case of fever is increased by the fact of the patient having a naturally weak heart, not so much from the presence of valvular disease, as from weakness in the contracting power of the muscular walls. Its strength may be sufficient for the ordinary work in health; but when fever occurs, the work is increased while the organ is further weakened, and then it is apt to break down. 4. The danger is always great when there is evidence of antecedent renal disease. To the kidneys we must look for the elimination of the large amount of nitrogenous detritus resulting from the febrile process, and when the eliminative power of these organs is impaired by disease, the peccant material is retained in the system, with the deleterious consequences already described. Here, again, the kidneys may be sufficiently sound for the due performance of their functions in a state of health, although they are quite incompetent to discharge the additional work

thrown upon them in pyrexia. 5. In most fevers the prognosis is bad in proportion to the intensity of the pyrexia, as measured by the thermometer. With few exceptions, a case with a temperature of 105° must be looked on as severe; and one with a temperature of 107° as highly dangerous. 6. In any case of fever the prognosis will be bad in proportion to the intensity of the typhoid state; and when convulsions occur in the typhoid state, recovery is rare. 7. In any case the prognosis will be bad in proportion to the weakness of the heart, and the tendency to stagnation of the blood in the capillaries of the lungs and other parts of the body." Stability of temperature from morning to evening is a good sign; on the other hand, if the temperature remains high from evening till the morning, it is a sure sign that the patient is getting or will get worse.

Pathological Summary.—The abnormal phenomena peculiar to and characteristic of fevers, whatever may be their specific origin, may be summed up under the following heads:—(a.) Increased temperature of the body; (b.) increased disintegration of albuminous substances, and loss of weight; (c.) diminished muscular power; (d.) diminished ingestion of nutriment; (e.) diminished urinary water (retention of water in the system); (f.) diminished powers of the senses and sensorium.

As to the seat of origin of febrile pyrexia, a satisfactory explanation of the nature of fever and of its relation to the febrile process is not yet possible, because we are not as yet in possession of the physiological knowledge necessary for safe conclusions and inferences. Two possibilities, as Dr. Burdon Sanderson has shown, are open for acceptance. One is that fever originates in disorder of the nervous centres, and that by means of its influence on the systemic functions the *liberation* of heat at the surface of the body is controlled or restrained, so that "by retention" the temperature rises, and, finally, that the increased temperature so produced acts on the living substance of the body, so as to disorder its nutrition. The other observation is, that fever originates in the living tissues—that it is from first to last a disorder of protoplasm; and that all the systemic disturbances are secondary. The first of these hypotheses may be definitively rejected in all its forms; for, on the one hand, no disorder of the systemic functions, or of the nervous centres which preside over them, is capable of inducing a state which can be identified with febrile pyrexia; and, on the other hand, it is possible for such a state to originate and persist in the organism after the influence of the central nervous system has been withdrawn from the tissues by the severance of the spinal cord. The tissue origin of fever must, therefore, be adopted as the basis on which eventually must be constructed an explanation of the process; the involved physiological problem being the normal relation between temperature and the generation of heat (thermogenesis).

From what has been written, it may readily be understood how the *conditions which combine to produce the complex phenomena of febrile pyrexia* may be stated to be—(1.) The entrance into the blood or tissue juice of a morbid agent—a material fever-producing cause—either from without (as in small-pox, scarlet-fever, malaria), or generated within the body (gout, rheumatism), or from local injury, such as a wound or surgical operation, in inflammation, and the alteration of the blood to a certain extent under its influence, come first in the order of events. This change begins to

occur during the incubative period, when often there is no rise of temperature, no fever; that is, when no appreciable alteration of the general health can be discovered, the morbid action of the agent on the organism being antecedent to all functional disturbance whatever. The nature of the change in the blood is unknown. (2.) When the change in the blood has reached a certain point, the nervous system, or rather that part especially connected with nutrition and organic contractility (the sympathetic and vagus), begins to suffer changes in composition which probably paralyse, impede, or destroy the normal molecular currents. When this occurs, the nervous symptoms of weakness, depression, rigors, contraction of some parts and vessels, speedily followed by relaxation, mark the stage of invasion. (3.) Various parts, simultaneously, especially the muscles, nitrogenous tissues, and probably some of the organs, deprived in greater or less degree of nervous influence, begin rapidly to disintegrate into substances of similar chemical construction, and by their disintegration preternatural heat is produced. Little or no fresh material is assimilated to compensate for the loss; great muscular prostration ensues; loss of body weight and destruction of tissue are increased by the accelerated action of the heart. (4.) This metamorphosis is aided, in most cases, by the condition of the vagus and vasi-motor nerves, which cause increased action of the heart and dilatation of the vessels, as the earliest results of pyrexia. (5.) The contamination of the blood, already produced by the morbid agent, is increased by the check which the normal extra-vascular currents experience, by the pouring into the blood of the rapidly disintegrating tissues, and by the continued action of the specific disease-poison, which in almost all cases appears to act more rapidly and more powerfully in blood rendered impure in any way, either (as shown by Dr. Carpenter) by retention of excretions, absorption of septic substances, or, as in fever, by the too rapid metamorphosis of tissue. (6.) The various organs suffer (apart altogether from specific changes), and must produce increased deterioration of the blood. Thus the lungs are congested in so many cases that we can scarcely suppose proper aëration to go on; the liver would seem (from Frerich's observations) to be in a most abnormal condition, in some cases at any rate, and to produce compounds unknown in health (such as *leucin*); and the spleen in many fevers, if not in all, enlarges (in persons of a certain age), and is congested, possibly even to extravasation. (7.) Food being almost withdrawn, the various alkaline and neutral salts, so essential to nutrition, no longer pass into the system. (8.) Non-elimination of the products of tissue-metamorphoses may give rise to cerebral symptoms, known generally as *typhoid*, and local inflammation, known as secondary. (9.) On the elimination of the fever poison and of the products of the tissue-metamorphoses, the nerves resume their normal functions, the undue consumption of tissue is checked, and the patient regains strength and weight. (10.) When coma, delirium, or stupor present themselves in the course of fever, it is the custom to refer these symptoms to the action of the fever poison on the brain; but the cerebral functions are more probably deranged, not alone by the fever poison (which was the first and the necessary link in the pathological chain), but by the accumulation in the blood of the products of tissue-metamorphosis, and by the perverted and defective nutrition of the brain itself.

Hence the symptoms in the advanced stage of many fevers are closely assimilated, although the primary poisons have been perfectly distinct (PARKES, MURCHISON).

All these events tend to render the febrile state an extremely complex one, and its investigation difficult. Our science is often spoken of as an exception to the exactness or comparative certainty of the (so-called) physical sciences; but nothing can demonstrate more clearly the claims of the Science of Medicine to exactitude and certainty than the advances made in recent years in our knowledge of the natural history of febrile diseases—their differential diagnosis, their causes, their modes of propagation—their development, natural course, and termination. For many years the German student and physician have been familiar with the use of the thermometer; but, until Dr. Parkes, and the pupils he taught when Clinical Professor in University College Hospital, began to use the thermometer, its usefulness in recognising febrile diseases does not seem to have been hitherto sufficiently appreciated in the medical schools of this country. The author is pleased to learn, from the numerous communications he receives, that this chapter, in previous editions, has led many to use the thermometer invariably in clinical work; and since 1868, the use of the thermometer in practice has steadily increased. (Consult also Dr. H. T. A. GOODRIDGE, *Brit. Med. Journ.*, July 29, 1876.)

III. *Varieties and Types of Fever which are to be clinically recognised.*—These are determined by the course, progress, and severity of the fever, as well as by the special combinations and the succession of the symptoms, and by the probable cause.

The varieties of type are as follow:—

(1.) *Continued*:—Those in which the febrile movement runs a regular course uninterruptedly as to increase of temperature from day to day—the temperature rising continuously up to the maximum, without decided exacerbation or relaxation, till the *acmé* of the fever is reached, after which defervescence sets in—*e.g.*, typhus, small-pox, measles, scarlatina.

(2.) *Remittent*:—When marked abatement, lull, or remission in temperature and other symptoms occurs, lasting for some time, and followed by exacerbations of all the phenomena. This type is seen in fevers of tropical climates and malarial districts, especially in summer and autumn, also in enteric fever and in hectic fever. Enteric fever is sometimes still described as “continued.” It is, in fact, continued and also remittent as to type.

(3.) *Intermittent*:—When periodic intervals and paroxysms of fever occur, each commencing with a chill or cold stage, lasting from half an hour to an hour, and ending by excessive heat of surface and active febrile symptoms, resulting after several hours in a sweating stage, which completes a paroxysm, and the patient appearing then, for the time, quite well; but in from twenty-four to seventy hours the paroxysm repeats itself. This intermittent type includes several forms. When the paroxysm repeats itself in twenty-four hours, the fever is of “*quotidian*” type; in seventy hours, of the “*quartan*” type; when it repeats itself in forty-eight hours, the type is “*tertian*,” when a daily paroxysm, corresponding in time and severity, repeats itself on alternate days, the type is “*double tertian*.” These types are repre-

sented in the different forms of ague. The period between the beginning of two paroxysms is named the "*interval*," and includes one paroxysm with its succeeding *intermission* or *apyrexial* stage.

(4.) *Malarial*:—When fevers are characterised by distinct periodicity as to their phenomena, exhibiting intervals during which the patient is wholly or nearly free from febrile disturbance. Fevers of this type betray a common origin by strong family likenesses. They are alike in occurring in low swampy places—alike in their phenomena, and in the difficulty in eradicating the *malaria* poison from the system—alike in secondary lesions, such as enlargement of spleen and liver, and similar as to alterations of blood which they leave as stamps of their existence.

(5.) *Congestive*:—Some of these malarial fevers are characterised by pernicious, malignant, or destructive characters while at their height, as indicated by intense congestion of one or of several internal organs (cerebral, thoracic, or gastro-enteric), and extreme perversion of innervation.

(6.) *Simple*:—When fever is not severe, nor prolonged, nor combined with any lesion, the type is "*simple fever*."

(7.) *Sthenic or Inflammatory*:—When fever is severe, and febrile reaction is the strongest; the pulse firm and incompressible, full, sharp, and frequent; the skin hot, dry, and retentive of its increased temperature; the thirst intense; urine high coloured, with a thickly furred and moist tongue; loss of appetite and constipation; the face flushed, pains in head and other parts intense; the senses often preternaturally acute; delirium, if present, attended with increased rapidity of thought, and sometimes with violence. The blood readily shows a "*buffy-coat*," indicating increase in fibrine-forming factors. It is a type usually associated with the onset of local and acute inflammations, especially in young and plethoric persons (See p. 75, *ante*).

(8.) *Asthenic or Adynamic*:—When there is extreme debility of the vital actions, and a feeling of great prostration of mind and body, as shown in the voice, attitude, and muscular movements; when the temperature is only three or four degrees above the normal, with heat of surface easily depressed, the pulse soft and easily compressed, such a condition represents what is often called a *low type* of fever.

(9.) *Typhoid or Ataxic*:—When combined with extreme prostration, there is great confusion of thought, impairment of intellect, low muttering, delirium, stupor, more or less unconsciousness, sometimes passing into coma; involuntary voiding of the excretions, frequent muscular tremors, and *subsultus tendinum*, even when no exertion is made; extreme derangement and diminution of the secretions, the tongue dry, brown, and crusted; the secretions of the mouth viscid, dry, and dark-coloured, so that the teeth and gums are covered with "*sordes*;" the surface of the body dry and its heat pungent, but easily reduced; the heart's action greatly impaired, as indicated by its feeble impulse and sounds, and by the rapid, small, and soft pulse, often also irregular and intermittent,—such a combination of symptoms being also often described as "*the typhoid state*." (See p. 78, *ante*.)

(10.) *Malignant, Septic, or Putrid*:—When, in addition to such a combination of symptoms, the state of the blood is markedly altered, so that its putrescency becomes obvious; when petechiæ or vibices, and passive

hæmorrhages occur, or even gangrene, the fever is of the "*malignant*," "*septic*," or "*putrid type*."

(11.) *Hectic*:—When suppuration is prolonged, and the formation of pus continuous, or when there is excessive waste of the bodily substance, as by the discharge of secretions beyond the powers of the constitution to supply; when there is a drain on the system beyond its means, a typical febrile condition is set up, so that a period of remission and a period of exacerbation occur once and sometimes twice in twenty-four hours. Much sweating attends and terminates each paroxysm of this kind, and hence also great exhaustion. This *type* of fever is termed "*hectic*." It begins and creeps on very insidiously—almost imperceptibly at first. The pulse is subject to temporary quick excitement from very slight causes. Heat is especially felt in the palms of the hands and soles of the feet. The excitement of the pulse continues to be daily more and more easily induced, while it daily loses in power, and increases in frequency. It expresses a middle condition between the inflammatory and typhoid type; periodic exacerbation regularly commences towards evening, reaching its height about midnight, and terminating towards morning in most profuse perspiration. This sweating is sometimes called "*colliquative*," because it seems to "melt the patient down," and it may be replaced or accompanied by diarrhœa. When a second paroxysm occurs, it is generally in the morning, after the breakfast period. The pulse at the height of the paroxysm may reach 120—the beat is a jerk—the irritable pulse of a weakened heart. The heat of skin is so distressing that the scantiest covering can scarcely be endured. The respirations are quick and short. The appearance of the face is characterised by a florid, circumscribed suffusion in the form of a bright red, or pink spot, contrasting strongly and often beautifully with the pale cheek, the bright and sparkling eye, the clear, pearly white sclerotic, and commonly known as the "*hectic flush*" of the cheek—so well described by Dickens in the death of "Smike," as related in his novel of *Nicholas Nickleby* (p. 91, *ante*).

IV. *Points to be attended to in the differential diagnosis of fevers, so as to determine their most palpable characteristics and causes.*

(1.) Determine whether the fever is *symptomatic* or *idiopathic*. This is not generally difficult. The history of the case, with absence or presence of definite local pain and disturbance, will generally be sufficient. It must not be forgotten that with the advance of diagnosis many affections have passed from the domain of idiopathic fevers to that of inflammation of certain textures, and therefore to symptomatic fever—*e.g.*, many cases of so-called *gastric fever* are in reality subacute inflammation of the stomach; *brain fevers* turn out to be cases of meningeal inflammations; *lung fevers* turn out to be cases of peri-pneumonia, and hundreds of cases every year are sent to the fever hospital in London, with certificates from medical men that the case is one of idiopathic fever, which afterwards turns out to be erysipelas, pleurisy, pneumonia, or disease of the kidney or liver. (2.) Obtain facts for a natural history of the fever drawn from each case by clinical notes from day to day; determine the day and hour of the commencement of the fever, if possible, and the date of "shivers" or "rigor" which occurred, and the date of exposure to any

cause or source of infection, so as to fix a period of incubation. (3.) Measure the amount of fever heat at least three times daily (in twenty-four hours) by a clinical thermometer, and thus obtain a daily range of temperature, and a curve for the whole disease, showing the degree or amount of fever-heat, as well as the course and mode of progress of the disease. (4.) The duration of the fever in every case is to be determined by the thermometer ceasing to indicate a temperature above the normal standard. (5.) The daily amount and daily range of the excreta are also to be as correctly determined as possible. These are to be judged of in relation to the weight and age of the patient. Measure the whole urine passed in the twenty-four hours, and take its specific gravity, from which a rough estimate may be formed of its solid constituents. (See page 261, *ante*.) The intestinal discharges ought also to be noted, and the activity or torpor of the cutaneous and pulmonary functions taken into account. (6.) The symptoms and phenomena *in sequence* are to be carefully observed; so that the *type of the fever* may be determined, and any skin eruption as to its time of appearance, what part of the body it first appears upon, how it extends, its amount, its exact anatomical characters, elevation or not above the skin, its colour, and any changes it goes through in the course of the disease, the length of time it continues visible, and if it leaves any stain or mark behind it, or persists after death. To mark the same spot and its varying phases before and after death, use the following method of identifying the spots, as devised by Sir WILLIAM JENNER (*Medico-Ch. Trans.*, Vol. XXI., p. 114).

1. When first seen, and when it *disappears on pressure*, surround it with an ink circle, thus—



2. When the spot is seen, and only *fades on pressure*, mark it thus—



3. When the *centre is unaffected by pressure, and the circumference fades*, mark it thus—



4. When the spot is *unaffected by pressure*, mark it thus—



(7.) The correlation of temperature, pulse, respirations, amount of the several excreta, and general symptoms—*i. e.*, 1. The correlation of the temperature and pulse beats; 2. The correlation of the pulse and respirations; 3. The correlation of the amount of excreta, with general or special symptoms, crises, or lesions. (8.) Mode of accession of symptoms, as to being sudden or the reverse; whether the rise of temperature has been rapid or slow; sequence of early symptoms up to highest temperature; any *special symptoms* pointing to disorder of special organs or parts of the body, every individual organ being subjected to daily examination. Thus complications are determined as to their being constant or

accidental in certain fevers, as well as the modes of crises, and progress of defervescence. (9.) In fatal cases the morbid anatomy is to be investigated as regards every organ. (10.) Causes are to be especially investigated, and the modes of propagation—if by contagion alone, or by other modes in addition to contagion. The practical points to be determined with regard to contagion are—1. To find out if the closer and more frequent the contact of attendants with the sick, the greater number attacked by the disease; 2. If the disease is propagated by human intercourse from one locality to another; 3. To determine the limits or area of infection and extent of diffusiveness of the disease-poison—either by air or by water. These are the points for scientific investigation in cases where any fever is met with for the first time, the nature of which is not clearly determined. Some uniform method of inquiry is necessary to bring out characteristic differences amongst idiopathic fevers.

The general symptoms of specific fevers *in sequence* are usually—(1.) *A period of incubation*, more or less definite for each kind of fever, at the end of which certain symptoms, called *premonitory*, set in—namely, chilliness, rigors or shivers, nausea, vomiting, headache, lassitude, pains in the back or limbs, restlessness, sleeplessness, or delirium, when the patient is said to be “*sickening*” for the particular disease. (2.) Increase of temperature, of pulse-rate, and of respirations; great thirst, scanty urine, red in colour, very acid, strong odour, and high specific gravity, containing excess of uric acid and urea; alkaline chlorides usually deficient. The urine also appears thick. (3.) The functions of the alimentary canal from the mouth downwards are greatly deranged and impaired. The secretions are dried up, the tongue dry and furred, and the mouth dry or clammy. There is general thirst, and the bowels are constipated. The desire for food is entirely lost (*anorexia*).

CHAPTER X.

TYPES OF DISEASE AND THEIR TENDENCY TO CHANGE.

Definition.—*The TYPE or FORM of disease is the order of succession observed to obtain among certain morbid phenomena; and admits of modification from various causes, without the intrinsic nature of the phenomena being essentially affected (COPLAND).*

In describing, appreciating, or ascertaining the type of a disease, our attention must be directed to a variety of phenomena and conditions, such as those detailed as regards *fever and its types*. The type of the disease only becomes characteristic and distinctive when some one or other of those conditions becomes predominant, or manifests itself more decidedly than others. The hereditary or natural constitution of the individual may be regarded as an important element in determining and modifying the type of the disease. Nevertheless, it is the rule that diseases preserve

their essential characters and nature from age to age, although the opinions of the profession respecting them and their treatment may change from year to year, or from one period of time to another. It has also been much discussed in the past twenty years as to whether *all* diseases have not undergone a change of type during the past century; and in previous editions I have attempted to state the points in favour of such a belief—held by many—the specific belief being that formerly diseases were *sthenic*, and were to be cured by blood-letting; whereas now they have become *asthenic*, and demand an exactly opposite line of treatment. We have the high authority of Sir Thomas Watson in favour of the belief in such a change of type in disease, given in his usual eloquent language:—"I am firmly persuaded," he says, "by my own observation, and by the records of medicine, that there are waves of time through which the *sthenic* and the *asthenic* characters of diseases prevail in succession; and that we are at present living in one of its adynamic phases" (*Edinburgh Monthly Journal*, June, 1857). On the other hand, as Dr. Bristowe writes, "Many will be disposed to admit that the change of type has been rather in the medical practitioner, than in the disease or in the bodily constitution; and that the gradual change of treatment has been due either to the slow advance of knowledge with respect to the effects of remedies in disease, or to fashion." It must be admitted, however, that much of what has been written regarding changes of type in disease, is based on the uncertain impressions of individual men, sometimes handed down by tradition, rather than based on the results of a deliberative deductive inquiry from statistics applied to the question by men agreed as to the meaning of terms. It is contended that inflammation is the same now as it has ever been—that the analogy sought to be established between it and the varying types of fever is fallacious—that we cannot place reliance on the recorded experience of the past—and that our recent changes in therapeutics are solely due to an advanced knowledge of diagnosis and pathology (Dr. J. H. BENNETT, *Principles and Practice of Medicine*, p. 267). The local phenomena of inflammation are undoubtedly constant; but the question of change of type has reference to the constitution of the individual, and to the constitutional modes by which the inflammatory state is expressed in a given number of persons. There are certainly good grounds for believing that elements of constitutional degeneration exist in great abundance, especially in the communities of large towns; and, that Sir Thomas Watson has, I think, happily expressed the consequence of such deterioration in the sentence just quoted from his writings on the subject.

CHAPTER XI.

MODES BY WHICH DISEASES TERMINATE FATALLY.

OUR knowledge on this subject is derived chiefly from three sources, namely, from the examples and illustrations afforded by the study of—

(1.) Death from old age; (2.) Death from fatal injuries; (3.) The powers and actions of all our best remedies. Such study leads to the important practical conclusion, that the same lesions of important organs may prove fatal in very different ways, both in *acute* and in *chronic* disease; and the fatal event may be averted by very different and very opposite remedies (ALISON). It is also to be observed that in constitutions which are unimpaired, there may be recognised, in every morbid process, a tendency to a spontaneous favourable termination. Death happens either from the decay of life, as in old age; or from rapid sinking of the vital powers in acute diseases, as in *adynamic* fevers. It may happen, also, as an accident caused by some of those untoward lesions or derangements of the vital organs which occur in the course of the various diseases and injuries to which mankind is liable; as, for example, from fatal hæmorrhage before exhaustion has reached its utmost, as in some diseases of the lungs and digestive canal. *Death by extreme old age* may be considered, in many instances, as the desirable end of a long-continued, and perhaps towards its close, a dreary journey. The aged sojourner on the earth appears to fall asleep, as he might do after severe fatigue; and the long and weary pilgrimage of life is thus brought to a close with little apparent derangement of the ordinary mental powers. The final scene is often brief, and the phenomena of dying are almost imperceptible. The senses fail, as at the commencement of sleep; the perceptions become gradually more and more obtuse, and by slow degrees the aged being seems to pass into his final slumber, so that we scarce can tell the precise instant at which the solemn change from life to death has been completed. Sensation fails first, then voluntary motion; but the powers of involuntary muscular contraction, under the excitement of some external stimulus, may continue for some time longer to be feebly expressed. The blood at first ceases to be propelled to the extremities. The pulsations of the heart become less and less efficient. The vital fluid fails to complete its circuit, so that the feet and hands become cold as the blood leaves them, and the decline of temperature gradually advances to the central parts. Thus far the act of dying seems to be as painless as that of falling asleep; and those who have recovered after apparent death from drowning, and after sensation has been totally lost, assert that they have experienced no pain. What has unfortunately been called the *agony of death* may therefore be presumed to be purely automatic, and therefore unfelt. The mind at such a solemn moment may be absorbed with an instantaneous review of those impressions made upon the brain in bygone times, which are said to present themselves with such overwhelming power, vividness, and force, that, in the words of Montaigne, "we appear to lose, with little anxiety, the consciousness of light and of ourselves." At such a time the vivid impressions of a life well spent must constitute that *euthanasia*—that happy death—so much to be desired by all.

The untoward lesions or derangements of vital organs, which occur during the progress of disease, terminate the life of man by various modes of dying. While it is ordained that eventually all must die, yet it is possible sometimes to avert, for a time, the tendency to death. To know by what agents this may be properly accomplished, it is necessary to know the modes in which death may approach in disease. Sir Thomas

Watson has happily observed that life rests upon a tripod, whose three vital supports are, the *heart*, the *brain*, and the *lungs*. Through the impaired functions of some one or more of these organs the tendency to death is expressed. But inasmuch as the functions of these organs are mutually dependent upon each other, so impairment of function in any one of them may ultimately lead to death, while the mode of dying is expressed chiefly through the functions of another. The mode of dying in disease is usually a complex one, for many parts thus mutually dependent on each other are more or less immediately involved. Therefore it is of the greatest practical importance to observe how and when the different functions begin to languish, and how they may be best sustained in their exertions to maintain life.

(1.) *Death by Syncope, or death beginning at the heart.*—Professor Ogston, of Aberdeen, classifies the causes of death by syncope as they act—(a.) *On the circulatory system*, as in all cases of extensive hæmorrhages, whether by wounds of the heart itself or of the larger blood-vessels; escape of blood from other organs, as the uterus, lungs, intestines; excessive discharges which indirectly lessen the quantity of the circulating fluid, such as excessive suppuration and starvation. When a person loses blood to such an extent that he faints, as from a wound, or by hæmorrhage occurring in disease, and if the flow of blood is not arrested the state of *faint* or *syncope* continues, is not recovered from, and the heart's action ceases, not because it is unable to contract, but because its natural stimulus, the blood, is withdrawn from it, or does not arrive in sufficient quantity to be of use. This is sometimes called death by *anæmia*. The symptoms of approaching death by this mode of dying are—paleness of the countenance and lips, cold sweats, dimness of vision, dilated pupils, vertigo, a slow, weak, irregular pulse, and speedy insensibility. If the hæmorrhage has been sudden, in large quantity, as from the uterus in “flooding,” there may be nausea, or even vomiting, restlessness, tossing of the limbs, irregular sighing breathing (anxietas), delirium, and one or two convulsions before death ensues. (b.) *As the causes are referable to certain agencies which depress or exhaust the nervous system, without interfering with the amount of the circulating fluid within the body.* Such are certain poisons, acute peritonitis, low fevers, malignant cholera, severe lesions of the brain, concussions of the spine, severe blows on the *præcordia* or *epigastrium*, or the entrance of air into the veins. This mode of death is therefore more immediately connected with the heart itself, and independent of the supply of blood. In other words, the stimulus from blood may be sufficient, but the contractile power of the organ may fail. Such a mode of death is that by *asthenia*, the “*neuro-paralytic*” death of German pathologists. Many diseases which are due to morbid poisons in the blood tend to prove fatal by this mode of dying, and cases of extensive mortification of parts. The signs of death approaching by *asthenia* consist in the pulse becoming feeble and frequent, and ultimately failing altogether to be perceived. The muscular debility becomes extreme, but the senses remain perfect, often painfully acute, and the intellect clear to the last. Those who die by *anæmia*, or by *asthenia*, are often spoken of as having died in a *faint* or by *syncope*, although the conditions are distinct and the terms not synonymous. *Syncope* results from direct failure of the heart's action;

fainting seems due rather to great loss of nervous (ganglionic) power, consciousness remaining unimpaired. The two conditions may be combined.

Death by Starvation, is a mode of dying intermediate between *anæmia* and *asthenia*. In cases of very gradual starvation, an urgent feeling of hunger is not a prominent symptom; and even when it exists at first, it usually soon diminishes, and is succeeded by a feeling of exhaustion, faintness, and even loathing of food, if abstinence has been long protracted (R. B. HOLLAND). The mental condition connected with poverty may in part account for this deficiency of appetite. A depression produced on the nervous system is very early manifested in the impaired energies of all the vital functions, the weakened conditions of the intellectual faculties and moral feelings, and diminution of the general sensibility. Disturbance of the cerebral functions is at first shown by an unnatural languor, despondency, and listlessness, slowness and hebetude of intellect, with an inability to employ the thoughts steadily on any subject. Notwithstanding all this general languor, however, the patient sometimes manifests a highly nervous state; he is startled by any sudden noise, and flurried by the most trifling occurrences. He is liable to attacks of giddiness, "swimming in the head," staggering dimness of sight, with temporary delirium, and either falls, as in an apoplectic fit, or lapses gradually from a lethargic state into one of stupor, or of complete coma. In many respects the symptoms in these cases have a considerable resemblance to the effects of exposure to cold. In consequence of the torpor of the brain, it is often extremely difficult to obtain the requisite information from patients. Instead of showing any anxiety to communicate the symptoms and cause of their illness, or to relate the privations they have undergone, they have an unwillingness to be questioned, lie in a listless or lethargic state, without taking any notice of what is going on, and seem desirous only not to be disturbed. Such listlessness and torpor of the mental faculties, the tendency to fainting or to perfect syncope, and, finally, a state of cerebral oppression, amounting in some cases to coma, are among the most characteristic symptoms of defective nutrition, and the surest indications of its existence to a serious extent.

In February of 1862, a man, thirty-six years of age, was discovered in a stack, near Morpeth, dying from starvation. All attempts to rally him failed, and he ultimately died. He was an intelligent man, and had been editor and proprietor of a journal. A diary was found in his possession, containing entries of his condition from the 8th to the 25th of February, from which it appeared that during *seventeen* days he had twice tasted a piece of bread, but for the last *thirteen* days he had been entirely without food. During the first *ten* days of the *thirteen* he was able to obtain water, but on the *eleventh* day he found his legs were useless, and he lost all motor power in the lower extremities, so that "one-half of his body appeared to be dead." The case is of interest to the pathologist, as showing the length of time during which existence can be maintained if water alone be taken. Other causes of death by syncope are exemplified in those injuries which destroy life with sudden and extreme violence—such as being crushed by falling buildings, beams, masts, the wheels of machinery, or of railway trains.

A peculiar syncope also follows wounds of the heart itself not immediately fatal.

The *post-mortem* appearances in cases of *death by syncope*, when there has been complete suspension of the action of the heart, are that the lungs, brain, and capillary system generally, are nearly in their natural state. The arteries should contain blood as well as both sides of the heart, where it should be found in nearly equal quantities.

(2.) *Death beginning at the lungs by suffocation*, technically expressed by the term *apnœa*, or privation of breath, may be produced by—(a.) The suspension of the action of the muscles of respiration, due to the *exhaustion* of the muscles themselves from sedative effects on them of cold or debility; to *loss of nervous influence* to them, as from division or compression of the upper part of the spinal cord, or of the phrenic or pneumo-gastric veins; to *mechanical restraint*; or to tonic spasm. Tetanus, and the influence of strychnine, sometimes prove fatal in this way. Forcible pressure upon the chest or abdomen, as sometimes happens in crowds during a continued crush of people, or occurs to workmen who have been buried by falls of earth, sand, or rubbish; in short, whatever causes an immovable condition of the lung-case beyond a period of three minutes and a half will thus produce a fatal result. (b.) *Cessation of the action of the lungs* may be due to morbid states produced by disease, which terminate fatally by *apnœa*, as *œdema* of the glottis; disease, division, or compression of the spinal cord above the origin of the respiratory nerves (phrenic intercostals and spinal accessory); effusion of serum into the pleural cavities; sudden infiltration of the lungs by inflammatory exudation, or collapse of the lung in bronchitis; or to a mechanical obstacle, such as the admission of air or of some of the abdominal viscera into the chest in wounds of the thorax and abdomen. (c.) The partial or complete exclusion of atmospheric air from the lungs may be brought about by its entire absence or complete rarefaction. It may be *mechanically* excluded, as by a foreign body in the larynx, trachea, or upper part of the œsophagus, by submersion, by suffocation, by strangulation, or by suspension; or the place of the atmosphere may be taken by a gas which acts merely by excluding it; or, lastly, irritant gases, by producing spasm of the glottis, may effectually bar the admission to them of the air (Guy's *Manual*, pp. 302, 303). The two former of these sets of causes lead up to the fatal result by suspending the *mechanical actions* of the respiratory organs, the last of them by arresting the *chemical changes* going on in them. Their ultimate effect, however, is the same, the arrest of the *mechanical* speedily involving that of the *chemical functions of the lungs*. The suspension of these, whether directly or indirectly brought about, necessarily involves, in a very short period, the arrest of the actions of the *heart*, and subsequently the functions of the *brain*. The air having ceased to arrive at the lungs, the current of the circulation becomes entirely venous, no longer affording them their necessary stimulus; the heart's action becomes enfeebled; the general circulation is carried on in a languid manner, and, affecting the brain, its functions cease to be performed. The lungs, having now become gorged with blood, the circulation is arrested there, the left cavities of the heart are emptied, and death supervenes. The symptoms of approaching dissolution by this mode of dying are—strong but ineffectual

efforts to contract the respiratory muscles, and struggling efforts to respire, amounting to agony, of short duration, followed by vertigo, loss of consciousness, and convulsions; at last all effort ceases, twitchings or tremors of the limbs alone remain, the muscles relax, and the sphincters yield. The heart, however, still continues to act after all other signs of life are past, and the pulse may be felt. The recent experiments instituted by the Medico-Chirurgical Society of London show that, on an average, the heart's action continues for *three minutes fifteen seconds* after the animal has ceased to make respiratory efforts. On this last circumstance rests our hope of resuscitating persons so suffocated, if artificial respiration be timeously resorted to, and persevered in. This prolonged action of the heart circulates blood, which is dark, venous, and not arterialisied, and accordingly the face, at first flushed, becomes turgid, and then assumes a livid and purple hue; the veins of the head and neck swell, and the eyeballs protrude from their sockets. At length the heart ceases to beat, and life is extinct (WATSON).

The most prominent of the morbid appearances after death by asphyxia will only require notice at present, and these are sufficient to distinguish it from death by syncope. They consist in engorgement, to a greater or less extent, of the right cavities of the heart and of all the more important viscera, as the lungs, liver, spleen, kidneys, and brain, with a comparative vacuity of the left side of the heart and the arterial vessels.

(3.) *Death by Coma, or death beginning at the brain*, occurs when there is a loss of consciousness first, with the appearance of profound sleep, from which the patient may be partially roused. Such primary arrest of the brain functions may result from—(a.) Pressure on the brain, or medulla oblongata, by effused blood, depressed bone, &c.; (b.) the effects of severe blows or falls on the head and other parts, formerly attributed to the mere deficiency of nervous energy of the brain following the concussion or shock, but now to derangements, more or less distinguishable, of the cerebral structure; (c.) the use of certain substances, such as narcotic poisons, may be followed by coma, in consequence of the specific influence which they exert on the brain and nervous system; (d.) lastly, the hæmorrhages or discharges from wounds, when insufficient to induce fatal syncope, may lead to death by coma by their paralysing effects on the nervous centres. When the cause of death is such as to act *primarily upon the brain* so as to suspend its functions, the first effect which succeeds is the interruption of the *mechanical*, and, subsequently, the *chemical processes* which the respiratory organs perform; which next involve the functions of the *heart*, in precisely the same manner as if the cause had acted primarily on the *lungs*. From this it will be gathered that the essential appearances after death in either of these two ways are nearly, if not entirely, alike both in *coma* and *apnœa*—an amount of agreement, however, which mostly occurs in those instances in which *apnœa* has followed *coma*. The symptoms of approaching death by this mode of dying consist in a gradual blunting of sensibility to outward impressions, slowness of respiration—the inspiratory effort being often delayed, and then performed with a sudden noise and jerking effort, technically known as *stertorous* breathing. All voluntary attention to the act of breathing is lost, but the influence of a reflex stimulus to its performance continues. At length this function fails

also. The chest ceases to expand, the blood is no longer aërated, and thenceforward precisely the same changes occur as in *death by apnœa*. Finally, in death beginning *at the brain*, the arteries and the left heart are empty, and this although the suspension of its functions has been the effect of a concussion or otherwise. The right heart, the veins, and the lungs contain, on the contrary, a notable quantity of blood, but much less than when the death has begun at the lungs. There may likewise be congestion or effusion of blood within the head, in consequence less of this mode of death than of cerebral hyperæmia (OGSTON, *Med. Times and Gazette*, 4th March, 1876).

Such are the several modes by which death tends to approach; and "*to obviate the tendency to death*" is a doctrine and a duty which was often and strenuously inculcated by Cullen. After him, no less earnestly was it impressed on many by my respected teacher, the late Professor W. P. Alison, whose interesting *Lectures on Fevers and Inflammation* furnish numerous illustrations of this subject. To his *Outlines of Pathology and Practice of Medicine*, and to the first volume of Dr. Watson's *Lectures on the Principles and Practice of Physic*, the student is referred who would seek further information relative to the modes by which death may approach.

" Many are the ways that lead
To his grim cave, all dismal; yet to sense
More terrible at the entrance than within."

" It is as natural to die as to be born ;"

and thus, in

" Passing through Nature to Eternity,
The sense of death is most in apprehension."

CHAPTER XII.

PRINCIPLES WHICH DICTATE THE TREATMENT OF THE TWO COMPLEX MORBID PROCESSES—FEVER AND INFLAMMATION.

I. *As regards Fevers or the Febrile State.*

To avert the tendency to death in the febrile state, it is necessary to observe how fevers naturally terminate favourably, as described at p. 270, *ante*. When *fever* terminates by any of the modes already described, convalescence commences, normal nutrition is renewed, and the body begins to gain in weight. The blood is poor in albumen and in red particles; and there is a danger that the rapidity of metamorphoses of tissue will exceed the healthy standard, as shown by the great tendency to lose heat during convalescence from fever. The temperature may fall, and the excretions may diminish below their healthy amount. Great care, constant attendance, and watchfulness are therefore required when the

patient begins to convalesce, especially if the fever has been long and severe.

The treatment of the febrile state itself may be thus generally stated as consisting in a combination of measures,—(1.) To reduce excessive heat; (2.) To insure sufficient but not excessive elimination of the excreta; (3.) To act restoratively on the exhausted and semi-paralysed nerves; (4.) To neutralise any specific poison which may have set up the fever, and so to improve the state of the blood; (5.) To relieve distressing symptoms; and, lastly, To obviate and counteract local complications (PARKES, MURCHISON).

(1.) *To reduce excessive heat.*—It is of importance to notice, with the indications of the thermometer, whether the skin of the patient be moist or dry. An equal rise of temperature with a moist skin, indicates a greater increase of heat production than the same rise with a dry skin. In rheumatic fever and cases of puerperal fever, we have often a very high temperature with a skin covered with perspiration. These different states of the skin point to different remedial measures for their relief.

(a.) In pyretic states, *when the skin is dry and burning*, those agents are indicated which depress the circulation, and at the same time act upon the cutaneous vessels. *Antimony* and *aconite* are the types of such apyretic agents which induce perspiration, possessing as they do the double action of lessening the activity of the heart and dilating the cutaneous vessels (the heat-losing area of Rosenthal). They thus increase also the bulk of blood in them, and so tend to dissipate the body heat. By tending also to throw the sweat fluids into action, the further cooling effects of water evaporation are secured. *Antimony* with *opium* is also most useful; and in *chloral-hydrate* we now possess an agent which combines the properties and action secured by the union of *opium* and *antimony*, which exercises at the same time a calmative effect upon the nervous centres. The association of a dry burning skin with a high temperature may be alleviated by the use of the *warm bath*. This will often restore arrested perspiration. The following is an efficient and convenient mode of obtaining such a bath, originally proposed by the late Sir James Y. Simpson:—"It consists of from six to eight soda-water bottles filled with boiling water, and tightly corked; of as many woollen stockings wrung out of hot water, each one being drawn over a soda-water bottle; and the bottles so covered being packed round the patient in bed." The moist stockings so modify the heat, converting it from dry to damp heat; so that between the heat and the moisture the skin is thrown into action, and in from twenty minutes to half an hour free perspiration is induced, and with it increased loss of heat, and reduction of body temperature (*Brit. Med. Journ.*, p. 418, March 27, 1875).

(b.) When an abnormally high temperature (*hyperpyrexia*) is associated with a moist skin, the measures to be employed are such as will *reduce heat production*. Chief of these is the use of *cold* in various ways—as by means of the general cold bath, affusion by cold or tepid water, or by douching when the patient is in a warm bath, packing in a wet sheet, sponging the surface of the body by water, either cold or tepid, and the use of ice applied in bags, or of ice-cold water by enema. Dr. Robert Jackson, "the patriarch of Military Medicine," and after him Dr. Currie, of Liverpool,

in 1797, practised to an extreme degree the application of cold water—a therapeutic agency which is now again challenging attention. Medicine, like history, is thus constantly repeating itself. Jürgensen, Liebermeister, Hagenbach, Kuchenmeister, Wilson Fox, and Hermann Webber, are the most recent advocates and exponents of the application of cold water in the treatment of fevers. In health such an application tends to increase the metamorphoses of tissue (LEHMANN and SANDERSON), and therefore its use in the febrile state requires the greatest care and caution. To be of use, it must be employed very early in the fever—before the third or fourth day. As soon as the temperature rises above 102.5° Fahr., or higher (104° Fahr.), the treatment by cold water is to be commenced, and continued as long as the temperature remains so high. The effect and object of the cold are to lower the temperature—a purely physical phenomenon, and this lowering does not reach its minimum immediately after the use of the cold water, but continues for several hours afterwards, the blood giving up its heat to the cooled surface of the body; and the difference between internal and peripheral temperature, at first great, becomes gradually more and more equalised. The effect of the bath, which may amount to from 2° to 8° Fahr., depends upon its temperature and duration, and also upon the temperature and size of the patient, the period of the disease, and the time of day or night when it is used, according to the period of natural fluctuations of temperature.

To keep the average daily temperature under a certain range is the aim of cold water treatment; and this cannot be carried out safely without the constant use of the thermometer—at least every two or three hours during the day and night. The number of baths, or other methods of using cold water, which thus becomes necessary, varies according to the severity of the fever, the efficiency of the method adopted, and the stage of the disease. In enteric fever, as many as twelve baths may be required in the twenty-four hours in order to keep down the temperature; while in milder cases four or five baths daily may be sufficient; and as fever declines the number of baths becomes less. The contra-indications to the use of cold water are hæmorrhages from the bowels; alarming depression may also follow its use, even when the temperature in the rectum may indicate 101° Fahr. (COLLIE). Symptoms of collapse require to be carefully watched for, with the finger constantly on the pulse at the wrist, so as to administer stimulants, if necessary, while the patient is in the bath. If the bath does good, temperature and pulse are reduced somewhat, the tongue becomes moist and clean, the mind clear, and refreshing sleep follows its use. The result is bad, if restlessness continue, if exhaustion occurs, and sleep does not follow.

“In deciding as to the kind of cases (of *enteric fever*) in which the bath will be beneficial, we must keep in mind one of its most constant consequences, its effect on the heart and circulation. The latter is impeded, internal organs become congested, and the action of the heart itself becomes very feeble, sometimes alarmingly so. From this it would appear to be contra-indicated in cases complicated by internal inflammations; in persons with weak hearts—*e.g.*, children and elderly persons, and in those generally who suffer from any form of heart disease. It would appear, too, to be contra-indicated in cases where the circulation has been weakened by

hæmorrhage; and in the latter half of the disease, when the heart itself has been weakened from overwork, and possibly some kind of specific degeneration. It should not be given when convulsions are present, during menstruation, nor where it continues to alarm the patient. In very young children and in very old people, it should not be employed at all. Generally, it will be found useful in the *earlier* stages of the disease; in young persons with uncomplicated attacks, whose hearts are sound and not acting too feebly, who suffer from continued sleeplessness and delirium, which we prefer as a guide to a fixed line of temperature. We have never given more than three baths in the twenty-four hours; and we should not give in any case more than four, and then only on clear evidence that they were doing good. The duration of the bath should not, as a rule, exceed ten minutes, and in boys and girls of ten not more than seven minutes. Towards the end of the second week I should, in the majority of cases, discontinue the bath, and, if an antipyretic were needed, give quinine, which should be given to children and elderly persons instead of the cold bath" (COLLIE).

It is obvious, therefore, that this method of treatment requires trained attendants and careful supervision by the physician, and must be employed with great discrimination; that other symptoms besides pyrexia must not be neglected, and that very great care is necessary in the selection of the method of cooling to be employed. First, as to *cold affusions*, as used by CURRIE, KUCHENMEISTER, and others. This form of administration is especially useful where cerebral symptoms are severe, with depression of the motor energy of the brain and cord, threatening paralysis of the heart, or severe degrees of bronchial complication with passive collection of large quantities of thick secretion in the tubes. In the unconsciousness of "sunstroke" it is thus useful. If the sitz or shallow bath be used, the patient must have his whole chest, front and back, well rubbed with towels till the skin becomes red, as he sits in the tub. It has been so used with benefit at an *early period* of enteric fever and scarlet fever (*The Practitioner*, July, 1869, p. 45). The proper time for the use of the remedy must not be later than the first few days of the fever; and in scarlet fever, when the skin is hot and the rash bright and red. The patient being stripped, and sitting in an empty tub, or sitz-bath, should have four or five gallons of very cold water (40° to 50° Fahr.) poured over him (affusion); and when the heat of the surface returns, the application may be repeated and renewed again and again. Its good effect is to lower the temperature, to lessen the frequency of the pulse and the respiration, to render the tongue moist and soft, to diminish or remove stupor, to procure sleep, and sometimes it may bring about a perspiration, which brings relief. But if there be much nervous irritability, and especially in delicate females, the *shallow bath*, as less exciting than the *cold affusion*, is to be preferred. The patient then sits or is supported in an open bath, about six feet long, in a depth of water from six to twelve inches, having a temperature from 60° to 80° Fahr. The extremities and trunk must be well rubbed by the assistant, while water of the same temperature as the bath is gently poured over the head. The patient may remain in this shallow bath from five to forty minutes, till the temperature of the body is reduced.

Most patients, however, dislike sudden great differences of temperature;

hence Ziemssen recommends that the bath be begun at a temperature of only ten degrees lower than that of the patient, and then gradually, by adding cold water, reduce the temperature of the bath to about 68° Fahr. Such baths are extremely grateful to the patients. They may be thus laid bodily in a bath of a temperature of about 95° Fahr., which is gradually cooled down to 86° or 77° Fahr.; and as patients get stronger the bath is used colder and colder—77° or 68° Fahr. After the immersion, lasting from three to fifteen minutes, or even an hour, and regulated by effects as indicated by the thermometer, the patient is dried at once, and put to bed and covered as usual; and if the feet are cold, warm bottles, or a hot brick enveloped in flannel, may be applied. In cases of delirium, with a high bodily temperature (104° Fahr.) and prolonged sleeplessness, while the patient is held in a *warm bath* (92° to 98° Fahr.), ten, twenty, thirty, or more bucketfuls of cold water (40° to 60° Fahr.) are to be poured slowly over the head, hot water being constantly added to the immersion bath, so as to maintain its temperature at 92° to 98° Fahr. A refreshing sleep is sometimes the result. By using the *douche*, the cold water is made to impinge on some part of the body (head and shoulders, or individual joints, or any part in succession) with considerable force, and the nervous impression produced is correspondingly great—too great and uncontrollable to admit of its frequent employment in this way. Where *delirium* is furious it may sometimes be so quieted, and the good effects of the *douche* become visible if the pulse and breathing improve, or even continue as they were before commencing it. One good method of applying it is, to place the patient in a warm bath, and then apply the cold *douche* to the head (RINGER).

The relative value of the various methods used for the purpose of cooling the body may be given in the following order (LIEBERMEISTER):—*Cold affusion, cold wet sheet packing, cold bath.* The cold affusion has much less effect in abstracting heat than a full bath of equal temperature and duration. With regard to the cold wet sheet, it has been found that a series of four cold packings, one following upon the other as soon as the sheet becomes warm, has about the same effect as a cold bath of ten minutes' duration, and double the effect of the cold affusion. Since 1870, this method of treating fever has greatly extended over the hospitals of Germany, civil as well as military; and has found its way into private practice. Under its influence the mortality in cases of typhoid fever is said to have diminished by one-half, varying from three to ten per cent. The total duration of the fever, including convalescence, is also thereby shortened. Convalescence is more rapid; and secondary complications, such as pneumonia and bed-sores, occur more rarely; while the strength of the patient, especially the power of the heart, is less impaired. The beneficially calmative influence on the nervous system is also apparent. Most patients treated by cold water-baths sufficiently early retain intelligence unimpaired. Delirium is much rarer, and, if hyperpyrexia is prevented, does not occur; while the earlier the cooling treatment is commenced, the greater the probability is of nervous symptoms remaining altogether in abeyance. Involuntary evacuations from bowel or bladder do not occur; the frequency of the pulse is reduced; the strength of the heart is economised, and thus passive congestion of the lungs is less apt

to happen. The constant use of the bath may thus also lessen the danger of infection from such diseases as typhus and scarlatina.

Quinine and *digitalis* are types of the medicinal agents which best effect reduction of temperature. The use of *sulphurous acid* in drachm doses, or of *quinine* in considerable doses (three, five, to ten or more grains), every two, three, or four hours, all tend to reduce temperature. The effects of a few grains of *quinine* every three or four hours, along with a mineral acid, are often markedly beneficial.

Blood-letting, or hæmorrhage, also tend to reduce temperature. Venesection will lower the temperature of the body 2° or 3° Fahr. ; but general blood-letting can never be tolerated in specific fevers, such as *typhus*, *typhoid*, *scarlatina*, and the like ; and when the febrile condition is associated with a bounding pulse, a strongly acting heart, and a dry skin, *antimony* and *aconite* have now taken the place of blood-letting.

Infusion of digitalis, cautiously used in small doses at long intervals, has been found by Wunderlich and others to have a wonderful influence in reducing and moderating the temperature in many febrile states, such as enteric fever. Its most obvious action in small doses is to depress the force of the heart ; hence the indications for its use are just the opposite of those which obtain in cardiac disease—*i.e.*, *digitalis* is only to be used in those cases of fever in which there is no considerable degree of cardiac weakness (LIEBERMEISTER). The dose should therefore be cautiously regulated : it must not be repeated too soon, nor be increased, if it should not operate at once ; and is usually from eleven to twenty-two grains, extended over a period of thirty-six hours.

Alcohol, too, is useful. The experiments of Professors C. Binz, Parkes, and Assistant-Surgeon the late Count Wolowitz, have shown that alcohol is capable of reducing temperature ; but only in a very unimportant degree, so that its power as an antiphlogistic is very slight ; and such enormous doses must be taken, that harm can only come by any attempt at reduction of temperature from the use of alcohol. Dr. Ringer has made many observations on this point, and is convinced that little can be hoped for from alcohol as a means of diminishing the preternatural heat of fever patients. This much seems certain, however, that its anti-febrile influence is best expressed by the removal of those conditions which induce paralysis of the brain and heart, and when the temperature of the body is high, as indicated by the thermometer. In this respect it approaches quinine in its action, but at the same time possesses in addition its well-known stimulating action on the central nervous system and upon the heart. Depression is generally found associated with a high temperature of the blood, and passes off when it falls. But, in giving alcohol, it must be remembered that two circumstances may contra-indicate its use, namely,—(1.) *Its effect on the pulse* ; (2.) *Its influence on the tone and diameter of the vessels*. It increases the number of the heart's beats, as well as the strength of the contractions of the heart. If such effects are to be feared, of course alcohol is not proper to be employed either in fevers or inflammation. Certain precautions must therefore be observed in its administration, and its effects on the different functions carefully watched, to learn whether we obtain from the employment of alcohol good or harm ; and although the pulse and

heart afford the greatest and most reliable information on this point, yet the influence of the alcohol on the other organs must not be overlooked, as it may happen that while one system is benefited, others are injured, and with some good the alcohol on the whole may do much harm. "In judging also of the influence of the alcohol on the pulse, its compressibility is of more importance than its volume. Under the action of alcohol, a soft and yielding pulse of large volume often becomes much less compressible and smaller, changes which show an increase in the tonicity of the arteries and in the strength of the heart." Other circumstances also afford information as to the employment of alcohol, namely,—“At the two extremes of age, the powers of the body are easily depressed, and hence, with such persons, stimulants are early called for, and must be freely used. In such, and especially the aged, it is of the greatest importance to anticipate prostration by the early employment of alcohol, as when once this occurs the greatest difficulty is experienced in restoring the patient to his former state. Young children, when weak, take stimulants even in large quantities with benefit. And with the stimulant some easily digested food should always be given” (RINGER). Generally, the more the typhoid type of symptoms is expressed (p. 291, *ante*) the greater is the need for alcohol. But it is contra-indicated if delirium increases under its use; also, if delirium be noisy and acute; if there be severe darting and throbbing headache, co-existing with great heat and dry skin, flushing of the face, suffusion of eyes, and no impairment of the cardiac or radial pulse; if there be scanty urine, with a low specific gravity, containing little urea or much albumen; also if there be suppression of urine. On the other hand, the good effects of alcohol are shown by the quick pulse becoming slower; by the tongue becoming clear and moist at the edges; by restlessness and delirium giving place to rest, tranquillity, and perhaps to sleep; by the breathing becoming less difficult, and the skin becoming more comfortably moist.

Spirits, in the form of *brandy* or *whisky*, are the best forms in which to give *alcohol* during the fever movement; *malt liquors* are best during convalescence. The spirits ought always to be diluted to the extent of their volume with water or milk; and if coldness of the extremities or of the skin prevail, the milk or water must be made hot. Such stimulants are to be given in divided doses, frequently repeated—in urgent cases as often as every hour; and in larger doses during the night and towards morning, than during the day. But the nature of the case and the symptoms demanding *alcohol* must regulate its amount. A tablespoonful every hour is a large dose—*i.e.*, *twelve ounces daily*; but whatever may be the amount prescribed (and it ought to be as accurately prescribed as other medicinal substances) the dose ought to be reduced, or given at longer intervals, as soon as the symptoms yield for which it was given.

(2.) *To secure sufficient but not excessive elimination of excretions—the products of metamorphoses*—it is necessary to watch how the functions of the kidneys, liver, intestines, skin, and lungs are carried on, as the remedies must be so arranged as to meet the requirements of each case in these respects. If urinary excreta are deficient, and the symptoms point to retention of urea, *chloride of sodium*, the *alkaline salts of soda*

and *potash*, *citrates of potash and ammonia*, *liquor ammoniac acetatis*, *saline laxatives*, and a free use of *water*, pure and cold, are the most efficient agents for promoting the elimination of urinary excreta; and these may be varied by the use of such diluents as *barley water*, *toast water*, *apple water*, *gruel*, *orangeade*, *lemonade*, *tamarind water*, *currant water*, *raspberry vinegar*, *seltzer* and *soda water*, *tea*, cold, with a slice of lemon in it and no milk or sugar. Each of these may be tried by turns, drinking small quantities at a time and often. Five grains of *nitrate of potash*, or fifteen minims of *spirit of nitrous ether*, every three or six hours, with a table-spoonful of fluid, is a useful diuretic; also *nitre-whey*, prepared by boiling ℥ii. of *nitre* in a pint of milk, and straining; or "imperial drink," which consists of ℥i. to ℥ii. of *bitartrate of potash* to a pint of boiling water, and flavouring with lemon and sugar, to be used as a drink as often as possible. It may be necessary also to stimulate the region of the kidneys by hot fomentations or mustard poultices over the loins and abdomen; or by a poultice composed of an ounce of the fresh leaves of *digitalis*; or by flannel cloths wrung out of the hot infusion and covered with oil silk; or half an ounce to an ounce of the *tincture* may be sprinkled upon previously moistened *spongiopiline*. In any case the application of *digitalis* should not be suffered to remain on for more than eight hours, at the end of which time it ought to be removed, to be replaced at the end of six hours if no effect has been produced. *Purgatives* generally, and especially *salines*—i.e., salts of the alkaline and earthy metals—tend to promote urea elimination by the mucous membrane of the intestines. Purgatives have been recommended to be freely administered to fever patients during the first few days of their illness, and before exhaustion has set in, so as to produce several evacuations in the day (ARMSTRONG). By free purgation in scarlet fever the severe sore throat and swelling of the glands may be prevented, as well as other disagreeable *sequelæ* of this disease, such as discharge from the nose and ears. I have found the following formula of great benefit as a purgative for this purpose:—

R. *Magnesiæ Sulphatis*, ℥vi.; solve in aqua, ℥viii.; adde pulv. Guaiaci, ℥iiss.; Pulv. gum Tragacanth, Co., gr. xl. *Misce bene*. One-sixth part of this mixture given every four hours till the bowels are freely moved, gives great relief to the congested throat and swollen glands.

But in some fevers, as in *typhus*, actual purgation or diaphoresis must be induced with great caution. Excessive elimination by *diarrhœa* (as when stools exceed three to five daily) or sweating, requires to be held in check. So also elimination by the skin, to the extent of *diaphoresis*, is to be dreaded in *typhus* fever.

(3.) To act restoratively on the exhausted semi-paralysed nervous system; to neutralise the effects of any specific poison which may have set up the fever; to improve the state of the blood, to maintain as far as possible the nutrition of the body, and to stimulate, when necessary, the action of the heart by appropriate food, as well as by alcohol and other stimulants. These aims embrace some of the most important and difficult details in the management of fever cases. The restorative agents of the exhausted nervous

centres are, especially, food of a nutritious kind, and of easy digestion, given at frequent and regular intervals of not longer than three or four hours, after the fourth day of a severe fever; and it may be necessary to give food every hour, but not oftener, in small quantities at a time; and the smaller the quantity the more often it must be given, even if it is necessary to waken the patient out of sleep—a step the necessity for which must be carefully considered. Milk, beef-tea, eggs, soups, broths, panada, rice, cream, rice and milk, are the most valuable articles of food. Barley, water, gruel, lemonade, still or effervescing; arrowroot drink; coffee infusion, made nutritious with hot new milk, and with or without an egg beat up in it; or milk with isinglass, and variously flavoured with cinnamon, almonds, and sweetened with sugar, or with fruit, jellies (black currant, pine apple, strawberry, red currants, cherries, or plums) are drinks which may be taken (with ice, if requisite or desired) by the patient. Of all diets, milk is the best, or an oleaginous diet. Mild stimulants with *quinine* are also to be more or less employed. *Infusion of coffee* as a medicine has been given by Dr. Parkes, with the beneficial effect of relieving headache. Böcker and Lehmann have shown that the use of coffee in health delays the metamorphosis of tissue, and excites the nervous system. As a nerve-restorative, *phosphorus* merits some notice. And first, as *iron* is given where the blood requires nourishment and restoration, so *phosphorus* seems to nourish and restore the nervous system, especially in cases of fever, where much *phosphoric acid* has been passing by the urine. The forms in which it is given are—(1.) In pill, $\frac{1}{40}$ or $\frac{1}{70}$ of a grain of finely divided *phosphorus*, melted with fat, and the pill covered with an impermeable coating; (2.) in the form of *hypophosphites of potash, soda, or lime*, given in *camphor water*, to the extent of five grains of the salt, three or four times a day. The *potash salts* seem to have a resolvent and liquefacient action so strongly marked, that mischief may result from its incautious administration to persons affected with tubercular deposit in the lung. For the same reason it is of great value in *chronic bronchitis*, with thick fetid expectoration and congestion of lungs (Dr. THOROWGOOD, in *Practitioner*, July, 1869, pp. 14–20). *Camphor* has been also found of use in the *adynamic* type of fevers. It acts beneficially in strengthening the pulse and reducing its frequency. At the same time it moistens the skin and subdues delirium, especially the low muttering form. *Twenty grains* or more every two or three hours are required for this result, and its effects must be watched (GRAVES). *Counter-irritation by blisters* has been largely employed by Graves and other physicians as stimulants in fever, under the following conditions:—“With acute diseases, such as the idiopathic fevers and inflammations, it not unfrequently happens that persons already weak and much prostrated have their dangers greatly aggravated by the following mental state,—they become apathetic and unobservant, which condition increases till it even reaches partial insensibility or coma, and they can only with difficulty be roused, and then wear a stunned, stupid, and vacant look, and understand very imperfectly what is said to them. The body generally sympathises with this depressed condition of the mind, and its functions are more and more languidly performed, till those necessary

to life altogether cease. It is a condition which may not inaptly be compared to one produced by poisoning with opium, where there is partial coma, which produces a lethargy in the functions of the body, whose activity grows less as the coma continues and deepens. But there is no true and refreshing sleep, while it is a condition in which sleep is most urgently needed. With patients in such a precarious state, it is of all things necessary to rouse them from their state of lethargy, and with the restoration of consciousness and activity of mind, there occurs renewed vigour in the functions of the body, and the patient is removed from a state of imminent danger to one of comparative safety. To accomplish this, blisters of large size, in quick succession, and for a short time, should be applied to different parts of the body; for instance, to the chest, to the abdomen, and to the thighs and calves. I have seen very satisfactory results follow their application to the nape of the neck under such circumstances." But sometimes more good is obtained by an opiate and plenty of stimulants, carefully given, to produce sleep, out of which the patient wakes strengthened and much improved. No fixed rule can be laid down; each case must stand on its merits (RINGER).

The treatment of any special febrile state depends on the disease of which it forms a part, and by which it is more or less modified—forming a special topic for consideration in the part which treats of special diseases. It is above all necessary to guard against the habit of trying always to be doing *something*. As a routine system, nothing can be laid down as a rule, either in the direction of depletion or of evacuants; of stimulation or of restoration. The febrile state is in many diseases part of the essence of the morbid condition, which cannot be cut short nor materially subdued by remedies. There is no specific remedy for the cure of any specific fever. Every disease where fever is present, and every case of specific febrile disease, must be studied so that its management or treatment may be regulated on the merits of the individual case; and must be regulated by the state of each particular function as determined by clinical investigation daily. No remedial agent here mentioned can "cut short" a specific fever. Judiciously employed, they may render them less dangerous, and may in some cases save life. There is no evidence of actual elimination of any specific disease-poison through any one of the excretory channels during specific fevers. Neither diarrhœa, diuresis, nor sweating are shown to carry away the poison of any specific disease. It is, no doubt, through the more or less rapid metamorphoses of tissue which go on, as the patient's system struggles with the fever, that the gradual elimination of deleterious products takes place. Time is an important element in every case; and while food and alcohol, given at the proper time and in the proper way, maintain the system through the struggle for existence, the eliminating organs must not be stimulated to undue secretion, nor congestion be induced, nor increased work thrown, by over-stimulation or over-feeding, on the overtasked glandular organs. If the patient can be kept alive during the febrile movement, the poison and the disorder will together pass away.

(4.) *To relieve distressing symptoms.*—(a.) *Headache* may be relieved by an emetic, or by inducing action of the bowels, or by evaporating lotions to

the forehead, such as *vinegar and water*, with *solution of muriate of ammonia*, or by bladders of ice over the shaven scalp, or by affusion of cold water, or pouring cold water (40° to 50° Fahr.) over the patient's head, as the neck is bent over a basin at the edge of the bed. Sometimes, as in the aged, warm fomentations are best; while in the very young and robust, leeches to the temples may give relief. (b.) *Sleeplessness* may be overcome by *opiates*, combined with *stimulants*, if not otherwise contra-indicated; or by the *hydrate of chloral*, in doses of from fifteen to twenty grains, diluted with simple syrup, provided the heart is not fatty, and there is no atheromatous disease of blood-vessels, nor advanced lung disease, nor instability of the nervous system. (c.) *Tympanites* is best relieved by *turpentine stupes* over the abdomen, or by *enemata of turpentine, assafoetida, and rue*; or *turpentine* may be given internally, combined with *tincture of perchloride of iron*. (d.) *Hiccup* may be relieved by sucking ice, and ice is always most grateful to fever patients, as it helps to allay thirst. Sponging of the body with cold or tepid water is also very comforting to the fevered patient, as it will sometimes bring about relief by perspiration, while it soothes restlessness and favours sleep (RINGER).

(5.) *Hygienic management*.—*Good nursing* (one nurse by night and another by day) is absolutely necessary in the management of all specific fevers. *Fresh air*, and plenty of it, is also an important condition of success—thorough ventilation being secured by open doors, open fire-places, and open windows. No carpets, bed-hangings, nor unnecessary articles of furniture should be in the patient's room, which ought to be large and airy, having two beds in it; so that the patient may be shifted from one to the other as occasion may require. Its temperature ought to average 60° Fahr. The patient ought to lie on a hair-mattress, or on a spring-bed, with a moderate amount of bed-clothes—not weighty—over him. Cleanliness, especially as to the perineum and genitals, must be carefully attended to; and in cases where cerebral oppression exists, attention must be given regularly, at least twice daily, to the condition of the bladder, and the catheter used if necessary. The state of the urine is also of importance to note.

(6.) *Complications and sequelæ must be watched for*.—The thermometer is here of great use, combined with observation of the respiratory functions; for pulmonary complications are those most frequently met with in the form of bronchitis or hypostatic congestion. Diarrhœa and bed-sores may also occur in the course of severe and long-continued fevers. Bed-sores are to be prevented by annular air cushions or water pillows; and as soon as any redness appears on the sacrum or hips, or on any other part subject to pressure, it should be kept dry and painted over twice daily with a mixture of collodion and castor-oil, or with the white of eggs beaten up in an equal volume of rectified spirit, or with a solution of gutta-percha in chloroform (one drachm of sheet gutta-percha to one fluid ounce of chloroform). If sores actually form, stimulating poultices are to be applied till the slough separates. The stimulants to be thus used and covered with the poultice may be—two parts castor-oil, with one part balsam of Peru, spread on pieces of lint; or carbolic oil, or a few drops of carbolic acid, or of turpentine, in the poultice.

(7.) *During convalescence* the diet is to be carefully regulated so as not

to exceed. Ale or porter, rather than wine or spirits, may be given, if necessary; the bowels must be kept open by laxatives or enemata; and *mineral acids* in bitter infusions; *iron, quinine*, or both, are most useful tonics, especially combined with change of residence and open-air exercise.

II.—Treatment as regards Inflammation.

Antiphlogistic treatment is the technical expression which denotes the management of cases of inflammation, and comprehends *rest, regimen, and diet*, with such remedies as have for their object the lowering of the increased vascular action in the inflamed part, and subduing the general or inflammatory fever. As to the febrile state, the principles on which it is to be managed as regards food and drink and hygienic arrangements, have been indicated in the previous pages. But in cases where inflammation affects the organs of respiration, the temperature of the air in which the patient breathes should not be less than 70° to 80° Fahr. and as pure as possible—the air being rendered moist by steam, if necessary.

The antiphlogistic remedies are—*blood-letting, purgatives, diaphoretics, diuretics, antimonial, mercurials, iodide of potassium, opium, aconite, digitalis, alkalies, and salines*.

Blood-letting.—The most important and the most efficient of so-called *antiphlogistic* remedies is undoubtedly *blood-letting*, now become almost a lost art in the practice of medicine, the letting of blood in any way being now one of the rarest of surgical operations; while it must at the same time be remembered that it is not every case of inflammation that requires or warrants the abstraction of blood. It is a spoliative remedy, powerful for good or for evil. There can be no doubt, however, that “whereas general blood-letting was at one time often employed too indiscriminately and in blamable excess, its subsequent almost total disuse was unwise, and in many instances disastrous—a more *serious mistake* than overuse had been” (Sir THOMAS WATSON, in letter to J. T. MITCHELL, Esq., Nov. 6, 1875). In the treatment of inflammation, it has, therefore, been well observed, that “each case requires its special study, speaks its proper language, furnishes its peculiar indications, and reads its own lessons.”

The practice of general blood-letting has thus ceased to be the routine remedy it once was; and there are reasons for believing that venesection is now less valued, because the younger medical men of the day have never, as students, had opportunities of seeing its usefulness. The class of patients in the hospitals of medical schools in large towns is not usually of the plethoric or *sthenic* constitution, which tolerates and requires venesection. It is in country life rather than in town life—amongst the well fed and plethoric, and not amongst the poor, the starved, and anæmic—that cases occur where venesection is required. The younger medical men have been taught to do without its aid, and now declaim against the practice of venesection, as they may have heard their teachers do; and so, because they do not know its merits, they altogether ignore its usefulness. At the present day we undoubtedly overvalue the blood, and estimate too cautiously the loss of it (PAGET). But just as the administration of alcohol in disease has been used to excess, so the practice of venesection has also at times

greatly exceeded its necessity; and as the cases requiring alcohol are now better understood, and certain rules for its administration can be clearly defined (see p. 307, *ante*), so the cases suitable for venesection are also capable of being indicated, and rules laid down for its practice. There are assuredly certain cases in which life may be sacrificed by adopting less active measures than venesection. These are, especially, certain cases of inflammation of the pleuræ, of the peritoneum, and meninges of the nervous system; grave cases of inflammation of the lungs; cases of apoplexy from slow hæmorrhage, with marked plethora; and cases of the so-called ardent fever of the tropics. A veteran surgeon of more than sixty years' experience, writes that for more than thirty years he had filled the office of medical director of one of the largest life insurance companies of the country; that one part of his duty was to record the cases of death and their causes; and from the frequent instances in which death occurred from acute pleuro-pneumonia, peritonitis, and other inflammatory attacks of vital organs, in subjects many of whom were young, and who before their fatal illnesses had enjoyed robust and vigorous health, he came to the conclusion, that the disuse of "general and free blood-letting in the early stages of such affections was the cause of such mortality—a practice which for many years has been most unwisely and unjustly reprobated by teachers and hospital practitioners, and which now is scarcely ever heard of but as one to be utterly condemned" (J. T. MITCHELL, F.R.C.S., *Med. Times and Gazette*, Jan. 15, 1876). The tissue or organ affected, the condition of the patient as to age, constitution, and general health, the nature of the inflammation, and the stage to which the process has reached, are the special points for investigation in deciding as to the propriety of venesection. Every case must be judged upon its merits. The assemblage and succession of symptoms in each case must be taken together; and no single indication is to be relied upon, every one being important in itself, and every one having a relative value. It is necessary clearly to understand and to bear in mind that, in the first instance, it is not the *lesion* which may attend the inflammatory process as a result which is to be attended to; but it is the *diseased action tending to the lesion* which it is the object of the physician to overcome, to subdue, and turn aside; and that the occurrence of any lesion is, if possible, to be prevented. It is the *strictly vital action—the irritation and subsequent excitement of tissue which tend to the organic lesion*,—to which remedies must be applied, in order to avert the lesion, the existence of which is expressed by the symptoms of a local and constitutional kind already referred to.

The carefully recorded facts of well-conducted though empirical observation, since the days of Hippocrates, have attested the immediate sanative influence of blood-letting in *incipient* inflammations; and the most eminent physicians of modern times have recorded, in unmistakable language, how potent is this remedy for good, and the reasons for their belief. Our forefathers well knew when the body suffered from an inflammation in the "*inward parts*;" and in saying this we give them credit for far less scientific knowledge than they really possessed. Against such inflammations, whether in the *head*, the *chest*, or the *abdomen*, they learned, by "*watching* and not by *counting*,"

the sanative efficacy of early venesection; and they obtained most trustworthy evidence and experience of its power to control inflammations. Following such doctrines will be found veteran physicians and surgeons who hold the foremost rank in the science of medicine of the present day in this country. The doctrine hitherto taught, and, till recently, universally acted upon with reference to blood-letting in inflammations is, "*so to bleed as to secure the advantages of the remedy, and to avoid its disadvantages*" (WATSON).

The standard examples of what blood-letting can do soon became apparent to every surgeon's apprentice or hospital pupil of former days—the days of my own apprenticeship, thirty-six years ago—if he did not himself swoon the first time he saw the blood flow from the patient. We saw the apoplectic sufferer roused to consciousness while the blood yet flowed from the vein; and observed also, that the *stounding pains* of the head in *cephalic inflammations* were immediately relieved; that the impatience of light and sound, the frequent, sharp, intermittent pulse, with vomiting or nausea on assuming the erect posture, the tendency to squint,—in short, all the urgent symptoms of incipient encephalitis—at once, or one by one disappeared, as the blood continued to flow. We also noticed in *thoracic inflammation* that the pain, the dyspnœa, the tightness of the chest, all disappeared. Dr. Alison, as regards pleurisy, and Dr. Watson, as regards inflammation of the bowels, have borne personal testimony to the good effects of blood-letting. They experienced its sanative influence in their own persons, and the practice undoubtedly saved their valuable lives from these respective diseases. Sir James Paget has, more recently, written of his experience as an apprentice, that the custom was scarcely without fail to bleed largely all who suffered from acute symptoms, especially from acute pain without the ordinary signs of fever. As soon as the hot skin, and a rapid pulse, and a flushed countenance were found, with local pains, or any other acute symptoms—as delirium, for example—at once bleeding was resorted to; and Sir James expressed his belief that there is at present no remedy employed from which the immediate relief derived is so great, or so complete as it was from bleeding (*Brit. Med. Journ.*, p. 221, 1874.)

The immediate effects of loss of blood in healthy persons up to fainting, and even up to the epileptiform convulsions that come with the loss of blood, is absolutely harmless; and as a remedy in inflammation the effects of venesection are,—(1.) A sedative result on the heart's action, by diminishing the pressure on the vessels, by diminishing the quantity and by altering the quality of the blood. Thus it weakens the power of the heart, and thereby prevents it from sending the blood with the same force and velocity into the suffering structures. The withdrawal of a considerable amount of stimulus from the central organ has thus a depressing effect, greater in proportion to the sudden loss of blood—the excitability of the nervous system being also reduced. (2.) It unlocks all the emunctories, and thus promotes secretion and elimination. (3.) The loss of blood generally has a derivative influence upon the blood in the part about to become the seat of effusion. This has been seen to occur in experiments upon the transparent parts of animals. (4.) In accomplishing these two results general blood-letting diminishes the quantity of living matter in which the peculiar changes comprised

under inflammation are going on. (5.) By disgorging the vessels at the seat of the disease, blood-letting restores the circulation, and places the absorbent vessels in a better condition for the removal of effused matter; and thus also favours, probably, by promoting absorption, the action of *purgatives, diaphoretics, diuretics, and anodynes*. (6.) While the effect of blood-letting is well known, by the observations of Louis and Alison, not always to check the extension of the sphere of inflammation, *yet it essentially modifies* its character, sometimes (a.) by limitation of its sphere to a certain extent, (b.) by diminishing the general plethora of blood from which the inflamed part is nourished, and (c.) by rendering the fibrinous exudation more liable to re-absorption. (7.) It lowers temperature from 1° to 5° or 6° Fahr. in a few hours, diminishing the arterial tension by facilitating the peripheric circulation. (8.) It reduces the pulse from ten to twenty beats *per minute*.

Of late years many men of large and varied experience in the medical profession have proclaimed, with no uncertain sound, as to the good effects of blood-letting in the treatment of certain inflammations. And one of our chief inquiries is to know what are the kind of cases which suffered from venesection, and what the kind of cases that were really benefited by the treatment which we now neglect. It ought to be well known that no remedy demands a more careful study of its application, or a more delicate adjustment of its powers.

“It is an important practical error to fix the attention, particularly of students of the profession, too much on those characters of disease which are drawn from changes of structure already effected, and to trust too exclusively to these as the diagnostics of different diseases; because, in many instances, these characters are not clearly perceptible until the latest and least remediable stage of diseases. The very object of the most important practice, moreover, in many cases, *is to prevent the occurrence of the changes on which these lesions depend*. After these lesions are once established, the cases are very often hopeless, or admit only of palliative treatment. In those diseases in which most can be done by art, our practice must always be guided in part by conjecture, because, if we wait for certainty, we very often wait until the time for successful practice is past; and therefore, although an accurate knowledge of the whole history of each disease is essential to its proper treatment, yet, in a practical view, the most important part of its history is *the assemblage and succession of symptoms*, by which its nature at least, if not its precise seat, may often be known *before any decided lesion of structure has occurred*.” (W. P. ALISON).

(1.) Blood-letting must not be indiscriminate, but must be done for a reason, early and freely, to save tissue and promote resolution. The suitable cases are those of acute inflammation of any of the solid organs or serous membranes in young plethoric persons otherwise healthy, in whom fever is severe and of the inflammatory or sthenic type (See pp. 75 and 291, *ante*). (2.) We ought not to bleed when the inflammation depends on, or is associated with, the action of a morbid poison, as in specific fevers, *septicæmia, pyæmia, puerperal fevers*, and the like. (3.) It is necessary to observe carefully, and to watch the *combination and succession* of the

constitutional and local symptoms from the commencement of the febrile attack, and so to judge as to the proper time, as well as the propriety of blood-letting. (4.) In order to obtain the good effects of the remedy by a full bleeding, it must be done *prior to fibrinous effusion or new growth*; seeing that it is *the diseased action which tends towards the lesion*—namely, effusion or growth of new material—which the physician desires to control and prevent by this remedy. (5.) When the symptoms of *inflammatory fever* are little complicated and seen early, in persons previously healthy, *the more violent the symptoms are, the more intense and rapid the constitutional reaction* (if it does not indicate exhaustion), and *the more decided the change on the function of the part affected*, the more confidently we may depend on the effect of full blood-letting in relieving them. (6.) When the symptoms of inflammatory fever have been uncertain and insidious in the beginning, so that the early stage has passed over unchecked, or modified by previously existing constitutional disease, or complicated with organic local disease, or when they denote debility, exhaustion, or the so-called typhoid state, such cases are improper cases for blood-letting, even when seen within the first few days. (7.) Generally, it may be stated, that when the fever is high (above 104° Fahr.), when we may be sure that over a part of the inflamed organ there is congestion, stagnation of blood, distension of vessels, commencing extravasation, and change of the constitution of the blood—but these latter changes still partial and not far advanced—the power of blood-letting to control such disease has been clearly established. It will lessen hyperæmia and engorgements of the venous system—the result of impeded circulation through the lungs and the left side of the heart. A feeble arterial pulse will not, in such cases, contra-indicate blood-letting; and stimulants are not incompatible with its practice. (8.) The nature of the membrane or organ affected must always be considered in estimating the propriety of bleeding. If a *serous membrane*, for instance, be actually inflamed, the patient, *if otherwise healthy*, for the most part bears bleeding well, and is usually greatly relieved by it. With respect to organs, it is found that *inflammation of the brain* is less influenced by bleeding than *inflammation of the liver*, and *inflammation of the liver* less than *inflammation of the lungs*. The gist of the evidence brought forward in Dr. Markham's very able and interesting "Gulstonian Lectures" establishes the special efficacy of venesection in those inflammations which are accompanied with *obstructions of the cardiac and pulmonary functions*; but do not prove it to be useless nor injurious in the cure of acute inflammations generally, if its remedial functions are properly used, and the advantage gained be duly followed up by appropriate remedies, such as are to be presently noticed. The symptoms which demand a full blood-letting in *pneumonia* are also those which indicate the greatest danger—namely, violent *pyrexia*, usually beginning suddenly, with full, strong, hard, and quick pulse—urgent *dyspnoea*, even *orthopnoea*—*swelling and flushing of the face*, frequency and violence of cough, with scanty or truly *pneumonic expectoration*, aggravating the pain which extends through the chest. *When such symptoms are seen within THREE days of their commencement, especially in those of robust and full habit in the prime of life, blood-letting is the remedy to be used,—everything else is trifling, and it is not safe to dispense with it.* The nearer a case answers this description, the more sure we may be that the

effect of blood-letting will be satisfactory, and its repetition, if the symptoms shall recur, will be well borne (ALISON). Mr. J. T. Mitchell, F.R.C.S., writing on this subject, after an extensive experience of more than sixty years, gives the following graphic account of a case in point (*Med. Times and Gazette*, Jan. 15, 1876):—

“In the year 1847, during the notorious epidemic of influenza then extensively prevailing throughout this country, very many cases of acute pleuro-pneumonia came under my treatment, which gave me a most extensive opportunity for observing the effect of general bleeding in acute disease. At that time I had a very intelligent assistant, who had been for some time a pupil at one of our largest hospitals, where for twelve months he had been observing the practice of the physicians of the institution; and I shall never forget the astonishment which he expressed on seeing my treatment in a case of pleuro-pneumonia that occurred in a young and previously robust and healthy man. One morning, whilst he and I were engaged among a crowd of waiting people, a young woman, in a most excited state, rushed unceremoniously into the surgery, pushing the people aside, and with great importunity came up to me exclaiming, ‘Oh, sir; do come as soon as possible to see my husband, for he is dying!’ I asked her from what he was suffering, his age, and his business, when she answered, ‘He is a carter, and about twenty-six years of age; he was quite well the day before yesterday until night, when he was seized with difficulty in breathing, a dreadful cough, and agonising pain in the side; his face is now perfectly blue, and his hands and feet are as cold as ice. As her importunity was so great, I said to my assistant, ‘This poor fellow evidently is suffering from acute pleuro-pneumonia; go down, and immediately take from his arm twelve or sixteen ounces of blood.’ Upon which he said, ‘I never bled in my life, and I have not a lancet.’ I then gave him a lancet and a short lecture on bleeding, and sent him off with the poor woman; very soon after which he returned, and told me that ‘the poor man was dying, and nothing would save him—indeed, he was pulseless and cold.’ As soon as we had dismissed the cases surrounding us, we proceeded together to this patient’s house, where I found him suffering in the manner described by his wife and by the assistant. I had seen cases much in the same state, but perhaps never under the same extremely alarming circumstances. His wife now repeated what she had told me before of his previous condition, adding that he had always been a most temperate man, and had never been ill before. Well, what was to be done to give him any chance of relief? I said to the assistant, ‘I shall at once bleed him.’ This evidently excited his ridicule. ‘What!’ said he, ‘bleed a pulseless man!’ ‘Yes,’ said I; ‘wait and see the effect of my attempt.’ I first procured two large pails, and got them filled with water about 100°. Having placed them at the side of the bed, I cautiously raised him from the recumbent to the sitting position on the edge of the bed, and put each foot and leg into one of the pails. I then had two wash-hand basins nearly filled with water of the same temperature, and placed his hands and arms as deeply as I could into them. I then tied up his right arm, for the purpose of ‘raising a vein.’ At first, pulseless as he was at the wrist, no vein would rise, but after a minute or two a vein became sufficiently prominent to enable me to make a free incision into it. The first effect of this was that blood flowed only drop by drop, but in a short time a small continuous stream followed, until enough blood had passed to relieve the stagnant circulation, when the stream

increased, and at last it flowed *pleno vivo*,—upon which my young friend's formerly sceptical countenance changed, and began to brighten with evident astonishment, and he expressed his wonderment. By this time the pulse at the wrist had become restored to considerable power, the venous livid congestion of the face had greatly lessened, and very soon it entirely passed away. I now requested the man to inspire as deeply as he could, upon which he said the pain in the chest and side was greatly lessened. I still allowed the blood to flow, until sixteen ounces had been collected in the basin, at which time he said he had no more pain, but he felt extremely faint; upon which, having secured the vein, I removed him from a sitting to a recumbent position, and gave him two grains of opium; after which, having darkened the room by drawing down the blind, we left him, having directed the wife to give him nothing but warm milk, and as much as he might be disposed to take; and if he should fall asleep, by all means to prevent his being awake. All this took place about mid-day, and at six in the evening we went again to see him, when we found him with a countenance bearing a natural aspect, pulse distinct and of moderate power, and about 100 in the minute; his breathing very much relieved, but still more frequent than natural; but the pain in the side had returned to a slight extent, upon which I again tied up his arm, and, from the same orifice previously made in the vein, drew off, in a good stream, six ounces more blood: this entirely relieved him. I then repeated the dose of two grains of opium, and left him, having reiterated the instructions given in the morning. From this time, by implicit rest, sedative diaphoretic medicine, counter-irritation by mustard-plasters on the chest, and light nutritious diet—chiefly milk—he day by day rapidly improved, so as to be able to return to his work after a fortnight's interval. On observing the conspicuously sudden and unmistakable result which followed the bleeding, my young friend declared, as we walked from the house, that he had learnt more of practical pathology, therapeutics, and physiology relating to the functions of the heart and lungs from this case and treatment, than he had gained by all his previous studies and observations made during the time which he had spent at the hospital, and in the course of his four years' previous apprenticeship, which he had passed in a large dispensary in a populous town in the West of England."

(9.) It is found in practice that this most powerful of therapeutic agents in the cure of inflammation requires the greatest caution in its repetition, for there is a line beyond which bleeding becomes destructive instead of remedial. Certain indications are of great use in determining as to the amount to be taken, and as to the propriety of a second blood-letting. As to the amount to be taken, it must depend upon the impression produced. When the face becomes pale, the pulse soft, and the pain and sense of distress are alleviated, enough has been drawn. Next, the position is of importance, as Marshall Hall showed—namely, to bleed quickly from a large opening in a good-sized vein, and to avoid the recumbent posture—thus the maximum of good at the least cost is attained. As to the propriety of repeating the operation, every case must make its own rule; and the following indications may be of use as a guide:—(a.) *As to how the first bleeding is borne*—a test first suggested by the late Dr. Marshall Hall. If much blood flow from an ordinary-sized opening, before any tendency to syncope manifests itself, venesection is then con-

sidered to be well borne; if, on the contrary, the patient soon faints after a vein is opened, further depletion must be abstained from. The urgent symptoms, being thus relieved for the moment, may again return, after a longer or shorter interval, and thus demand a repetition of the remedy; the propriety of which is to be now judged of—(b.) by the reaction of the system generally, as indicated by the state of the local symptoms, their urgency for relief, the character of the pulse, and the appearance of the blood first drawn. The reaction may be of such a kind that a *sthenic* state of *inflammatory fever* still continues, or returns after temporary subsidence. The inflammatory process having been interrupted, so far modified, but not arrested, the remission proves transient, and the re-accession may be more fierce than the onset. A repetition of blood-letting is demanded so soon as such reaction has declared itself. On the contrary, the reaction may be *asthenic*, or of nervous character, the pulse being rapid, soft, and jerking, the breathing oppressed, headache and *tinnitus aurium* present, with general nervous excitement; bleeding, under such circumstances, *is not to be repeated*. A full opiate will allay the nervous excitement, and furnish the most effectual means of obviating reaction. (10.) It is an object to effect the sanative result with as little expenditure of blood as possible; but the amount to be taken can only be judged of by the effects produced. The patient should be bled, if possible, in the upright position, and a full stream of blood allowed to flow from a sufficiently large orifice in a vein. To accomplish this fully, it may sometimes be necessary to open a vein in each arm, so that the flow may be from both at the same time.

Blood-letting may be employed either generally or locally. General bleeding is best adapted to subdue acute inflammation of visceral organs, because it makes a more decided and rapid impression upon the system. Local blood-letting, by leeching, scarification, or cupping, is more useful in chronic inflammation and in inflammations affecting membranes, such as the pleura, peritoneum, meninges, and articular membranes, by diverting the blood through the superficial vessels from the deeper arteries which supply the inflamed parts (see the observations of STRUTHERS and TURNER in *Edinburgh Monthly Journal*). It is sometimes advantageous to combine the two methods of taking blood. In the case of external visible inflammation, the direct abstraction of blood from the inflamed part during the congestive period of the inflammatory process, is *seen to* exercise a benign influence over the progress of the inflammation.

Purgatives.—The next most important class of antiphlogistic agents in the treatment of inflammation consists of *purgatives*, especially the *resinous cathartics*, like *jalap*, *scammony*, and *gamboge*. (1.) They free the stomach and intestines from accumulated food and fæces, or other irritating and acrid matters. (2.) They subdue the inflammatory tendency by the discharge of a large quantity of serous fluid, charged with albumen, from a large extent of mucous membrane. Thus they tend to check effusions and diminish the force of the heart's action. Their use is especially indicated in *encephalic* inflammations and *hepatic* congestions; but they are less efficient in subduing *thoracic* inflammation; while in the *enteric* inflammations they ought not to be pushed beyond merely unloading the alimentary canal. Combined with diaphoretics, they promote the

elimination of morbid material through the glands. Next to these come *salines* and *alteratives*, in all acute and inflammatory affections, and in *acute intestinal inflammation*, maintaining a gentle, non-irritant, evacuant action, by large serous discharges. By such evacuant action the blood pressure is diminished, when a considerable quantity of fluid is withdrawn from the intestinal vessels (BRUNTON). Of saline remedies, the simplest and best are—*Bicarbonates of potass* and of *soda*, *sulphate of soda*, *bitartrate of potass*, *tartrate of potass and soda* (Rochelle salts), *sulphate of magnesia*, and the various natural purgative waters, such as Pulna, Friedrickshall and Hunjadi-Janos. Drachm doses of *sulphate of magnesia*, with a grain of *quinine*, dissolved in *dilute* or *aromatic sulphuric acid*, and an ounce of infusion of *gentian*, taken every three or four hours, is a very efficient antiphlogistic purge.

Mercury varies in its influence with its mode of administration and the constitution of the patient. It is followed, in large doses, by increased flow of watery evacuations from the bowels, and an increased flow of saliva. As a purgative combined with *compound jalap powder*, it is most efficient in causing a copious discharge from the mucous glands of the intestines, promoting (*somehow?*) a flow of bile, and if not, the purgative action is of a distinctive and peculiar quality. The stools are different from those caused by other purgatives, and the therapeutical effects are *sui generis*, as amply attested by a vast clinical experience. Mercurials, in all forms, greatly increase the elimination of the products of waste, by the intestinal glandular apparatus, and tend to lower temperature. If the use of the remedy is continued, especially in small and repeated doses combined with opium, so that it is not passed off by the bowels, this mineral induces *salivation*—that is, saliva flows profusely, the gums become tender, red, swollen, and ulcerated on the margins in contact with the teeth. The patient gets rapidly thin during its use. The blood is decomposed, the red corpuscles are rapidly destroyed, to the extent of *one-sixth* or more; the fibrine is diminished by *one-third* of its amount, the albumen by *one-seventh*; and, at the same time, it may become loaded with a fetid matter, the product of decomposition (WRIGHT). "Thus it is an agent of terrible activity, and we may well be cautious how to handle it" (HEADLAND). Its sanative power is believed to consist in controlling or preventing the coagulation of lymph; and for this purpose it is used as an auxiliary to blood-letting, and next to that as a remedy, superior to purgation. It is only useful in the *sthenic* forms of inflammatory action. *Bichloride of mercury* is certainly of great use in *iritis*, and in inflammations of the deeper seated parts of the eye. It is also of service in the inflammations of serous membranes, in which it appears not only to subdue the inflammation, but to promote the absorption of the effused products of the disease. Wherever coagulable lymph is effused, wherever adhesive inflammation exists, gluing parts together, and "spoiling the texture of organs," there mercury is of service notwithstanding the recent fashion to discredit it. The system should then be brought as speedily as possible under its specific influence—a result which is known by its effects on the gums, and on the breath of the patient. The gums grow red and spongy (then said to be "touched"), and the patient complains of their being sore, and that he has a metallic

taste—a taste like copper—in the mouth. At the same time, an unpleasant and very peculiar fetor is to be smelt in the breath; which, when once experienced, is easy of recognition again. Beyond this physiological point the effects of mercury ought not to be allowed to advance; salivation especially ought to be avoided. The specific action of mercury is best obtained by giving small equal and repeated doses of *calomel*, combined with just as much *opium* as will prevent its passing off by the bowels. Two or three grains of *calomel*, with a third or a quarter of a grain of *opium*, given every three, four, or six hours, according to the necessity of the case, will effect the results indicated in the course of thirty-six or forty-eight hours. Mercury thus employed sufficiently early, tends to prevent effusion, as well as to favour absorption of effused products. It is advantageously employed in membranous inflammations, and such as go on slowly. It is of special value in subduing the specific inflammations and products of syphilis when indurations exist. Its administration ought always, then, to be combined with nourishing diet and tonic remedies, especially when its use is continued for a lengthened period. Previous blood-letting renders the body more readily susceptible to the influence of mercury, and the two remedies will accomplish, by their joint power, what neither of them might be able to accomplish singly. If the inflammation has been slow and chronic, the influence of mercury must be also slowly brought about, as the lymph can only be slowly and gradually taken up again by the specific action of the drug sustained for a considerable length of time (WATSON, SIMON). Mercury is decidedly hurtful in cases of *erysipelas* disposed to gangrene, in *scrofulous states of the system*, in *debility*, in *disease of the kidneys*, such as *Bright's disease*, and in cases where the nervous system is in an irritable condition, and the condition of the patient tending to the so-called typhoid state.

Iodide of Potassium, by its administration, in addition to *mercury*, removes more quickly the inflammatory effusions, such as occur in pleurisy, and in the inflammatory thickening of organs, and in granular inflammations. It may be combined with *carbonate of ammonia*. It promotes the elimination of *mercury* and *lead*.

Antimony, in the form of *tartar emetic*, has long had the reputation of being a most valuable antiphlogistic. It tends to increase the secretions from the gastric and intestinal glandular apparatus, but particularly those from the skin and lungs. It is especially useful in those sthenic inflammations which are rapid, in which a sudden and powerful action is desired; and where the direct sanative influence of blood-letting is to be maintained. It is thus indispensable in *croup*, extremely efficacious in *sthenic pneumonia*, and highly useful in *acute bronchitis*. It is most efficient when the mucous membrane is dry and swollen, $\frac{1}{20}$ to $\frac{1}{12}$ of a grain every three or four hours. As an agent to keep up the sanative influence obtained by blood-letting, the action of *antimony* is invaluable; for when blood-letting has weakened the force of the heart, by diminishing the pressure on the vessels, then *antimony* maintains this diminished pressure in proportion as it maintains the diminished force of the heart's action. A perseverance in its use produces a watery condition of the blood, diminishing especially the amount of

fibrine. The production of nausea is an indication that it has taken sufficient effect. Much can be done with antimony according to its dose. A *sixth* or an *eighth* of a grain in any watery solution, repeated every *two* or *three* hours, will generally induce diaphoresis, thereby tending to subdue the intense heat of skin so characteristic of inflammatory fever. A quarter of a grain similarly repeated will add nausea to the diaphoresis, and thereby increase the sedative effect on the circulation.

Opium is one of the most important remedies we possess in the treatment of various inflammations. It relieves pain, quiets restlessness, and subdues the irritable state of the nerves of the part. It is especially useful in inflammations of the serous membranes; and if administered less in affections of the pulmonary mucous membrane, it is only because there is a fear that by lowering the respiratory process, it may tend to prevent expectoration. Sore throats, bad colds, and ulcers heal rapidly under its influence. As soon as a cause of inflammation begins to operate, treatment by *opium* ought to be commenced—a grain or two, or even three grains, or these equivalents in *morphia*, every three, four, or six hours, according to the indications, so as to keep the patient under its narcotic influence by renewed doses of the drug. Where pain has to be relieved and vascular excitement subdued, it ought to be combined with *antimony* or *ipecacuanha*, or both.

Aconite is also proven to be a powerful agent in counteracting and in subduing inflammations, producing manifest effects in small doses, easily disguised. Its tendency seems rather to arrest, or *cut short*, the disease, than to remove the products of inflammation when they are formed. It requires, therefore, to be used very early; and so long as the inflammation is extending, *aconite* will do good. Acute inflammation of any of the tissues may be subdued by the early administration of the *tincture of aconite* (two to five drops every three hours, according to age). In the inflamed parts, like the *tonsils* or the mucous membrane of the throat, the good effects of *aconite* may be *seen*; and its good results are most apparent when the inflammation to be subdued is not extensive or severe, as in the catarrh of children, tonsillitis, or acute sore throat. Under its influence the large, livid, dry, and glazed tonsils will have their appearance completely altered in a day, so that the mucous membrane is moist and bathed with mucus or pus, after which a little *glycerine of tannin*, applied to the part, will complete the cure. *Aconite* ought to be given at the earliest stage, when the chill is yet upon the patient. "In a few hours the skin, which before was dry, hot, and burning, becomes comfortably moist, and in a little time longer it is bathed in profuse perspiration, which may be so great that drops of sweat run down the face and chest. With this appearance of sweat, many of the distressing sensations, such as the restlessness, the chilliness, or heat and dryness of the skin, are removed. At the same time the quickened pulse is much reduced in frequency, and, in a period of twenty-four or forty-eight hours, it and the temperature have reached their natural state. It is rare that a quinsy or sore throat, if caught at the commencement, cannot be disposed of in twenty-four or forty-eight hours by its use. The sweating may continue for a few days after the decline of the fever, on slight provocation, but it then ceases" (RINGER). *Aconite* has had for a long time a reputation as a depressant of the heart's action, and capable as

such of lowering the force, or slowing the circulation. It lessens the pulse rate, lowers arterial tension, and diminishes abnormal heat. The admirable account of the physiological properties of *aconite* given in the inaugural dissertation of the late Dr. Fleming, of Birmingham, brought its usefulness more to the notice of the profession; but still it has been long in coming into general use, chiefly on account of its highly poisonous nature, and from the want of precise rules as to the class of cases demanding its administration. For a long time several eminent members of the profession have attributed the very highest value to *aconite* as an antiphlogistic, and Mr. Liston highly advocated its use in the treatment of *erysipelas*. Dr. Wilks, of Guy's Hospital, has been in the habit of using it for many years, when favourable cases occurred—that is, cases seen at the very commencement of an inflammatory process. He has used it in *pneumonia*, *acute rheumatism*, *whooping-cough*, and *chronic catarrh*, in doses varying from three to five drops of the tincture of *aconite* every four hours (*The Practitioner*, Vol. I, p. 334). Dr. Ringer has used it with success in cases of *catarrhal croup*, *severe colds*, *tonsillitis*, *pharyngitis*, and in *pericarditis*. When the heart throbs violently, thus producing extreme pain, *aconite* will quiet the heart and ease the pain. Dr. Ringer gives of the tincture *half a drop*, or *one drop*, in a teaspoonful of water every ten minutes or a quarter of an hour for two hours, after which the dose is to be continued hourly. It is contra-indicated in inflammatory states of the gastrointestinal mucous membrane; but is very serviceable in acute hepatic congestion and hepatitis.

Alkalies dissolve the fibrine of the blood and retard its formation. The ingredients of the urine produced out of the destruction of the albuminous compounds of the body are increased largely in amount by the administration of alkalies. They generally pass out of the body as salts, having combined with acids in the system; and they tend to leave behind them an excess of alkali in the blood.

All treatment of inflammations ought to be judiciously regulated by the knowledge of the tendency of the disease to a spontaneous favourable termination—the accidental symptoms of urgency requiring treatment and control in many cases, rather than the disease itself. In every case, the details of treatment must be well considered and fixed upon to have any practical value. It is not possible to lay down one rule based on general principles applicable to every case; for each case must be studied and managed in accordance with its individual requirements; and it is especially the duty of the clinical teacher to instruct the student in this kind of knowledge, because it is a kind of instruction which must be seen to be appreciated. To teach the art of applying remedies is the duty of the clinical instructor; and any text-book of practical medicine can only faintly indicate the *principles* by which treatment ought to be guided.

When individual diseases are considered in this Text-book, whether involving *fevers* of a specific kind or *inflammations*, the details of treatment applicable to each will be set forth in as much detail as possible, but always requiring that the principles here stated should be constantly referred to; and, therefore, they will not be again specified in each instance of *fever* or *inflammation*.

THE SCIENCE AND PRACTICE OF MEDICINE.

PART II.

METHODICAL NOSOLOGY—SYSTEMATIC MEDICINE; OR THE DISTINCTIONS, DEFINITIONS, NOMENCLATURE, AND CLASSIFICATION OF DISEASES.

CHAPTER I.

THE AIM AND OBJECTS OF NOSOLOGY.

NOSOLOGY, regarded as a distinct department of the Science of Medicine, embraces three separate topics for consideration—namely, *First*, The DISTINCTION and DEFINITION of particular diseases, or of genera and species of diseases; *Secondly*, The NOMENCLATURE OF DISEASES, or the assignment to them of names by which they are to be designated, so that each disease may be known by an appropriate name; and, *Thirdly*, The ARRANGEMENT or CLASSIFICATION of diseases in some methodical and convenient order, by which they may be distributed into groups or classes, orders, genera, or species. These three divisions of Nosology are respectively known as the DEFINITIONS, the NOMENCLATURE, and the CLASSIFICATION of diseases. Of these in their order,—

I. The Definitions of Diseases.—The first object of Nosology is to obtain such brief enumerations of the peculiar characters of diseases as are sufficient, provisionally, to define them. In modern times the great advantages that have arisen from establishing definitions in natural history upon fixed principles (not only of its various objects individually, but also of the groups under which it was found possible to arrange them) suggested to medical men the idea that much advantage might also result to the Science of Medicine from defining diseases, and grouping them under general terms or common names, based also upon some fixed principles. Sydenham recommended that definitions or brief descriptions of diseases should be framed after the model of those given of plants; and he lays down various rules for the proper execution of this object in the preface to his work *On Acute Diseases*, first published in 1675. The

precepts of Sydenham were never reduced to practice in his day; but about fifty-seven years after his work *On Acute Diseases* was published, the idea was taken up and acted upon by Franciscus Boissier de Sauvages, a distinguished physician and eminent professor of medicine at Montpellier. He attempted to arrange diseases, as botanists have arranged plants, into classes, orders, and genera. He endeavoured to lay down the characteristic phenomena of each, and to enumerate their principal varieties. The outlines of his nosological system were first published in 1732, and followed, thirty years afterwards, by his *Nosologia Methodica*—a work which marks an important era in the history of Medicine, as having led to much greater accuracy in the distinction of diseases than was previously observed.

At present the only useful method of defining diseases seems to be an artificial one. It is assumed by nosologists that the proper foundation for the distinction of particular diseases is the occurrence of constant and uniform combinations of morbid phenomena or symptoms, presenting themselves in concurrence or in succession. Thus some of the essentials of a definition are obtained, and each disease may thus be marked out by such a brief enumeration of its leading characters as serves to distinguish it from every other. A series of nosological definitions, more or less correct, may be thus provisionally established, so that the same things are designated by the same terms. Objections have been urged to methods of this kind, on the ground that diseases are unsteady and variable in their character; but the aids to science are now so numerous that physicians are becoming more and more able to distinguish diseases from one another, and to tell by what marks, or upon what grounds, they do so distinguish them. As often as we attempt to establish a distinction among diseases, either the deficiencies or the errors of our definitions will be the more easily perceived; and the attempt will lead to a more accurate consideration of observations previously made, as well as to a greater degree of accuracy in the future. Definitions of diseases are therefore not only of much service to methodical nosology, but they help to render the diagnosis of diseases more perfect.

Pathologists, however, are not agreed as to whether the definitions of diseases should be derived from the external phenomena that present themselves in their course, or from the internal pathological conditions on which these phenomena are supposed to depend; and particularly such of these conditions as consist in lesions or structural alterations discoverable after death, or a combination of all. Cullen was in favour of definitions derived from symptoms of diseases; and he believed that the information derived from pathological anatomy might guide to more correct and definite distinctions among them. Defining diseases by their supposed proximate causes may lead to error, inasmuch as in many cases these causes are still disputable. Whatever principle of defining diseases be adopted, it is absolutely necessary that it should be independent of every theoretical view; for any theory employed, however specious, however much we may be persuaded of its truth, may not appear in the same light to others, and may, therefore, occasion endless confusion (CULLEN). If no uniform principle can be laid down for arriving at precise definitions of disease, we must be content with such methods of definition as will

serve the main purpose of connecting intelligible general notions regarding the disease with given modes of expression. For example, although we cannot give such a definition of many a disease as will embrace even all the leading phenomena of every case, we may still give such a definition as shall apply, with reasonable accuracy, to the disease we intend to designate, so that no one may suppose we mean thereby either *small-pox* or the *gout* when we mean *typhus fever* or *dysentery*; and, in a progressive science like medicine, definitions must always be provisional.

II. The Nomenclature of Diseases.—This, the second object of Nosology, has given rise to many disputes, and has furnished much scope for the display of classical erudition. From the earliest periods of medicine, the names imposed upon diseases have been derived from several different sources; but the following considerations have generally regulated the naming of a disease:—*First*, Some names have been taken from the part affected—*e.g.*, *peripneumonia*, *podagra*, *ophthalmia*, *dysentery*. *Secondly*, The most characteristic symptoms have furnished the name—*e.g.*, *ileus*, *tenesmus*, *paralysis*, *diarrhœa*, *dyspnœa*, *coma*. *Thirdly*, Some names have been taken from these two circumstances combined—*e.g.*, *cephalgia*, *otalgia*, *cardialgia*, *odontalgia*, *hysteralgia*. *Fourthly*, An alteration of tissue upon which subsequent changes depend being recognised as the essential element of the disease, it is named accordingly—*e.g.*, *pleuritis*, *peritonitis*. *Fifthly*, Such alteration not being discovered, the first tangible link in the chain of causation has been used instead—*e.g.*, *melancholia*, *cholera*, *typhus*. *Sixthly*, When a lesion tending to sudden death at once follows the application of a cause, that cause may name the disease—*e.g.*, *lightning*, *prussic acid*, *arsenic*, *burn*, *scald*, *sunstroke*, *cut*, *stab*, *frost-bite*, &c. *Seventhly*, A considerable number of names of diseases have been derived from some imaginary resemblance to external objects—*e.g.*, *elephantiasis*, *cancer*, *polypus*, *anthrax*, &c. *Lastly*, There are still many names the origin of which it is not now easy to trace—*e.g.*, *sypilis*. It is obvious, from these statements, that the names of diseases must change as our knowledge changes and becomes more precise; and many diseases which were once named after their symptoms, are now named according to the lesion from which most of those symptoms proceed. An apt illustration of this is to be found in *paralysis*, which is no longer regarded as a disease *per se*, but is merely a symptom of several structural alterations of the brain and spinal marrow.

By some it is maintained that "the name of each disease or species should be so characteristic and significant that a person slightly acquainted with the language and the subject should, on hearing it, immediately understand what is the nature of the disease it designates" (PLOCQUET). In this respect the name ought to be composed out of the same elements as the definition of the disease; in fact, it ought to be the definition converted into a name, and derived either from the symptoms of the disease, or from the supposed proximate cause. But a name which is expressive only of the nature, seat, or proximate cause of a disease may be erroneous in respect of each of these facts singly, or of all of them together. The history of the nomenclature of fevers, especially *enteric*, would amply illustrate these statements—*e.g.*, *putrid fever*, *adynamic fever*, *bilious fever*, *pythogenic fever*, *typhoid*, *meningogastric fever*, *nervous fever*, *gastric fever*, are instances of its confusing nomenclature. So also *lardaceous disease* (see

that article, p. 129, *ante*) may furnish another illustration. All these are mild examples of nomenclature and of confusion, which ought to make a man pause before he attempts to construct a new name. Naming diseases, such as fevers, from the names of places (for example, *Crimean fever*, *Bulam fever*, *Mediterranean fever*), is also extremely objectionable (p. 269, *ante*). It is inexpedient, also, to abandon (except when unavoidable) the names of distinct diseases received and recognised by our forefathers; or to substitute new ones in their place, without an extreme necessity. Sauvages insisted much on this point, and Cullen entertained the same opinion. "Words," says the former, "are good only in respect of their signification." In dealing, therefore, with ancient nomenclature, which, for the time being, may appear objectionable, it is better to extend, if possible, the signification of the word, name, or term, than to alter it. At the same time it must always be permitted to give new names to new diseases, and to select the best out of those which are in use, when a great number have been used to designate one and the same thing. There are some principles, therefore, which it is well to recognise as influencing the judicious choice of a name. Such names, for example, as involve or attempt to indicate a proximate cause, are more liable to lead to error than those which are derived from leading symptoms. If names were to be based on supposed causes, new names of diseases would be required whenever a new hypothesis is started. Look, for example, at the names of *enteric fever*, already mentioned, and the systems of Linnæus, Vogel, Pinel, and even Mason Good, will show that medical nomenclature has been repeatedly changed without any urgent necessity; and great inconvenience has especially resulted from incorporating particular and often peculiar pathological doctrines with the language and nomenclature of diseases. So much has this been the case that the language of medical science has been in danger of becoming "a curious mosaic of the chief speculations of ancient and modern times." The passion for inventing new terms retards also, in a wonderful degree, the progress of the student of medicine, and tends to involve him in difficulty and doubt. It will, therefore, be well for him and for our science, if teachers at the various schools will absolutely adhere to the definite form of nomenclature as agreed to by the London College of Physicians, and published ten years ago by the College.

III. The Classification of Diseases.—From time to time physicians have considered it advisable or advantageous to arrange, under more or less comprehensive groups, the whole of the diseases they are able to define and to name. A consideration of the different plans which may be pursued in such arrangements, and of the advantages to be derived from them, forms the third object of Nosology.

It is obvious that any single character, or combination of characters, in respect of which diseases agree with or differ from each other, may be made the basis of methodical arrangement, under a larger or smaller number of divisions, or of higher or lower genera (language of logicians), or of classes, orders, and genera (language of naturalists). By ingenious devices of the mind, the physician or the statist may classify and arrange his knowledge so as to bring it all more readily within his reach for any special purpose,—so as to make it, in fact, more at his disposal—to facilitate and pave the way for further investigation. Such are the

legitimate objects and the results of all methodical arrangements. Classification, therefore, being only a method of generalisation, there are, of course, several classifications of disease which may be used with advantage for special purposes. The physician, the pathologist, the jurist, the hospital statist, the army or navy medical officer, may each legitimately classify diseases from his own point of view, and for his own purposes, in the way that he thinks the best adapted to facilitate his inquiries, and to yield him general results. The medical practitioner may found his main divisions of diseases on their treatment, as medical or surgical; the pathologist, on the nature of the morbid action or product; the anatomist or the physiologist, on the tissues and organs involved; the medical jurist, on the suddenness, slowness, violent, or unnatural mode of the death; the hospital statist, on the kind of diseases which are treated in its wards; and all of these points of view may give useful and interesting results (FARR). There is thus no question on which more diversified opinions may be legitimately entertained than on that of classification. Although it is the aim of all systematic writers and observers to arrange the objects of study in the most natural order possible, and although diseases are named as if they were individual entities, yet they present so great varieties that they will not admit of that definite and, in many respects, natural species of classification which can be made with objects of natural history. Manifest reasons of convenience and facility for work can therefore be assigned as the great incentive to classification; and numerous reasons exist for classifying diseases in various ways:—(1.) Men differ in their estimation of the characters on which different arrangements may be founded. (2.) The facts and phenomena of diseases on which classifications may be made are not all regarded from the same point of view. Most systems are avowedly *artificial*, being arranged with the view to elucidate or support a theory, or otherwise to effect a definite end. For example, by classifying diseases and recording the causes of death, the most valuable information is obtained relative to the health of the people, or of the unwholesomeness and pestilential agencies which surround them. “We can take this or that disease, and measure not only its destructiveness, but its favourite time of visitation; we can identify its haunts and classify its victims” (FARR). We are able to trace diseases also as they perceptibly get weaker and weaker. We thus learn that certain diseases are decreasing, or growing less and less destructive; that certain other diseases have ceased in some measure; while other severe diseases have exhibited a tendency to increase. The advantages, therefore, of adopting some system of classifying diseases, which can be put to such useful practical purposes, must be obvious to every one. (See *Returns of Registrar-General*, prepared by Dr. FARR.) To some extent other systems are *natural* in their arrangement, in so far as they attempt to express or exhibit some of the natural relations which subsist among diseases; but the mere expression of one man’s interpretation of peculiarities of disease of the same species, and the elevation of such diseases in a classification as specifically distinct, are apt to be based on insufficient evidence as regards natural relations.

Principles of Classification.—Many systems of Nosology have been adopted from time to time; and as valuable general principles have

been adduced from some, the grounds on which diseases have been classified may be briefly and usefully described under the following ten divisions, namely :—

I. *The nature of the ascertained causes of disease.*—On this principle two classes of diseases are recognised, namely,—(1.) Diseases arising from general causes; (2.) Diseases arising from specific causes.

II. *The pathological states or conditions which attend diseases.*—The principle of this classification consists in determining alterations of the structure or the chemical composition of parts, from which names are given to the disease—*e.g.*, *pleuritis*, *pneumonia*. The distinctions of Sauvages were generally derived from symptomatic and pathological characters, or external symptoms alone; Cullen, following (1792), adopted similar grounds of classification; but, with much more comprehensive views than Sauvages, a more lucid order, and a happier simplicity, he excelled in accuracy of definitions all who had gone before him. His descriptions of disease received no colouring from his theories. They are faithful to nature, consistent with the knowledge of his day; and, greatly in advance of his time, his original and inventive mind dwelt much on the causes of disease in all his reasonings and explanations upon medical subjects. Aware, however, of the imperfections of the Art of Medicine, he did not attempt to arrange diseases according to their proximate causes, but according to a method founded partly on their symptoms, partly on their causes, and partly on their seats (CURRIE). A methodical arrangement of this kind has generally been considered the most desirable, as being likely to bring together diseases corresponding not only in some very important relations as regards their symptoms, but also in the indications and means of treatment which they suggest and require. But it is obvious that such an arrangement must vary according to the progress of knowledge and of opinion; for a disease which may at present be supposed to depend upon one pathological condition, may be found at a future time to proceed from another. Besides, the arrangement involves a principle which tends to separate diseases bearing a striking resemblance to one another in their external phenomena, though depending on different pathological conditions; for example, different species of *apoplexy* and *epilepsy*. It is an arrangement, also, bringing together diseases which, though belonging to the same natural family, may be respectively characterised by groups of symptoms that do not bear any very obvious resemblance. Thus the hæmorrhages at once bring together *apoplexy* and *hæmoptysis* in this classification.

III. *The properties, powers, or functions of an organ or system of organs being deranged*, dictates a classification in which the most prominent effects or phenomena of morbid states are considered as the disease—*e.g.*, *palpitation*, *diarrhœa*. It is an arrangement which brings diseases into approximation with one another according to the part of the body principally affected and the function principally disturbed. When disease consists in perverted powers or functions, it is then denominated a *dynamic* affection or disorder. When it depends on change of structure, it is termed an *organic lesion* or disease. This third basis of classification is Physiological, and was adopted by Drs. Young and Mason Good, in imitation of Ploucquet of Tubingen. It has been the most popular

arrangement of diseases, and perhaps the best adapted for lectures, or for treatises on the practice of Medicine, because it brings together the different diseases of the same organ, and of those organs most intimately related to one another; but, to profit by the arrangement, the student must be previously instructed in the general doctrines of disease.

IV. The diseases comprehended under the two latter principles of classification are sometimes inaccurately and loosely brought together under the heads of *Structural* and *Functional* diseases (See p. 255, *ante*). The diseases of function, for instance, are made to embrace the *neuroses*, *hæmorrhages*, and *dropsies*; while *inflammation*, *tubercle*, *cancer*, *melanosis*, *hypertrophy*, and *atrophy* are subordinate classes of the diseases of structure. The diseases of function embrace all those in which the action, the secretion, or the sensation of a part is impaired, without any primary alteration of structure of the organ or tissue affected, so far as our imperfect means of research can ascertain. Thus, *mania*, *cataplexy*, *neuralgia*, are *neuroses* of the brain or other portions of the nervous system. *Colic*, *vomiting*, *diarrhœa*, and *constipation* are *neuroses* of the alimentary canal; and so on of other parts. *Hæmorrhage*, or the effusion of blood, and *dropsies*, or an effusion of water into the shut cavities of the body, as that of the head, chest, or abdomen, are also instances of functional disease. Such are the grounds of classification adopted by the late Dr. Robert Williams, of St. Thomas's Hospital, London.

V. A basis of classification has been adopted, founded on the *pathological nature of the different morbid processes*, but the arrangement of the orders and subdivisions is determined by the anatomical arrangement of the textures and organs of the animal body, as originally developed by Bichat. Such is the principle and mode of classification adopted by Dr. Craigie (1836).

VI. A ground of classification exists, having reference to the *general nature and localisation of the morbid states*. It comprehends three classes,—(1.) Diseases which occupy the whole system at the same time, and in which all the functions are simultaneously deranged. These have been named general diseases, such as *fevers*. (2.) Constitutional affections, meaning thereby diseases which display themselves in local lesions in any part, or in several parts of the system, but not in all parts at the same time—*e.g.*, *rheumatism*, *gout*. (3.) Local morbid processes. Such is the classification adopted by Dr. Wood, of Pennsylvania (1847).

VII. Applying the principles of a *purely humoral pathology*, we have a classification consisting of—(a.) *Fevers*. (b.) *Dyscrasiæ*—*e.g.*, *tubercle*, *chlorosis*, *scorbutus*, *dropsy*, *diabetes*, *pyæmia*, *tuberculosis*, *carcinoma*. (c.) Constitutional diseases, induced by—(1.) Specific agents; (2.) Vegetable substances. Such is Wunderlich's arrangement of diseases (1852).

VIII. M. de Savignac, Professor of Clinical Medicine at the Naval School of Toulon, propounded (1861) a nosological arrangement founded on what he believed to be the “elements” of disease. To each of the classes he so defined, the question would at once suggest itself, and require solution, as to what the “element” may be on which the particular class is made to stand alone. He merely subjoined the word “element” to an adjective formed from the name of each class of diseases. Thus the class *Neuroses* is distinguished as the *neurotic element*; the class *Rheumatism*, as the *rheumatic element*; the class *Phlegmasiæ*, as the *phlegmatic element*; and so on.

as the *rheumatic element*; and so on to the number of fourteen classes. In the formation of orders, genera, or groups of diseases under this classification, no fixed principle can be recognised.

IX. Dr. Stark, of Edinburgh, proposed (1864) an arrangement embracing sixteen classes, namely:—(1.) Fevers; (2.) Diseases of the brain, &c.; (3.) Diseases of the heart and organs of circulation; (4.) Diseases of organs of respiration; (5.) Diseases of organs of digestion; (6.) Diseases of urinary organs; (7.) Diseases of organs of generation; (8.) Diseases of organs of locomotion; (9.) Diseases of skin and cellular tissue; (10.) Diseases of uncertain seat; (11.) Malformation; (12.) Debility at birth, and premature birth; (13.) Old age; (14.) Sudden deaths; (15.) Violent or unnatural deaths; (16.) Causes not specified.

X. Upwards of thirty-six years ago a classification was proposed by Dr. William Farr, of the Registrar-General's Office, which recognised the following five large classes of diseases, namely:—(1.) *Epidemic, endemic, and contagious diseases*. (2.) *Sporadic diseases of uncertain or variable seat*. (3.) *Sporadic disease of special systems and organs*. (4.) *External causes, poisoning, asphyxia, injuries*. (5.) *Developmental diseases*. To the first of these classes he assigned the name of *Zymotic diseases*, embodying a theory that a *materies morbi* was introduced into the body, which remained for a time unperceived in the system, and that, after an indefinite period of incubation, leavened the whole mass of the circulating fluid—a pathological series of events, however, which cannot be asserted positively of each form of disease comprehended in the class. Moreover, the larger number of diseases strictly preventible by efficient sanitary arrangements are comprehended in this class, so that the so-called *zymotic diseases* came to be synonymous with *preventible diseases*, and for statistical purposes it has become common to point to a large number of deaths from *zymotic diseases*, or *sickness* in any locality, as an index of faulty sanitary arrangements. In this class are comprehended *typhus, thrush, diarrhœa, rheumatism, syphilis*, a group of diseases each one of which is now known to be composed of very different pathological elements; and, as a group, impossible to represent the existence of any definite or even similar cause. The class *zymotic* was thus made to include diseases of which the origin and mode of propagation are wholly dissimilar; and the group was subdivided into "*miasmatic, enthetic, dietic, and parasitic diseases*," a subdivision involving no common principle. Even the *miasmatic* groups, implying diseases due to *miasm* (on which many medical men are at one as to the main features) is not a congruous group. Thus, *quinsy*, a simple inflammation of the fauces, is placed next to *scarlatina*; *erythema*, simple redness of the skin, stands as a subordinate variety of *erysipelas*, which it is not. *Diarrhœa* stands between *dysentery* and *cholera*, and has no place among the diseases of the intestinal canal, and the order concludes with *ague* and *rheumatism*, ignoring alike the malarious origin of the one and the constitutional origin of the other—*gout* standing at the head of *constitutional diseases*. As to *parasitic diseases* being *zymotic*, it is getting more and more difficult to understand even the analogy to such a pathological process (*Med.-Chir. Review*, October, 1869).

None of these methods leads to a perfectly philosophical or purely natural classification, because diseases are not yet sufficiently understood

to permit us to see clearly their mutual relations; and the best recommendation of any one of them would be a negative one—namely, that of doing the least possible violence to our very imperfect knowledge regarding the natural affinities or alliances of diseases, of which we have at present only a sort of instinctive recognition. In the present imperfect state of our knowledge, therefore, diseases cannot be philosophically classified, nor arranged according to natural or true pathological relations, dependencies, or alliances. Nevertheless, a great advantage inevitably results from the institution of nosological classification, on account of the necessity which every such attempt imposes on those who engage in it, of marking very accurately the characteristic phenomena of particular diseases; and every one acquainted with the progress of natural history, must know that the study of details, and the repeated attempts to systematise them, have mutually promoted and supported each other. It is the same with regard to diseases; and if a Methodical Nosology cannot be rendered perfect, it is a certain proof that, for the time being, the details of which it must be composed are neither accurate nor complete, and are not likely to be so till attempts to observe, investigate, and systematise have made further progress. Every attempt to reduce to system tends to enlarge our stock of facts; and though we may fail to obtain a perfectly philosophical arrangement, yet the very attempt to attain it must be of advantage, by leading to useful discussions regarding the Pathology and History of diseases (CULLEN). No one could be more convinced than Cullen was that “perfect division and definition is the summit of human knowledge in every department of science, and requires not only the clearest, but the most comprehensive views, such as (with respect to diseases) we can arrive at only by often-repeated attempts and much study.” A no less distinguished pathologist—M. Bayle—in discussing the difficulties connected with classification, recommends us “to follow the plan which presents fewest imperfections, remembering that the determination of specific characters is what is most essential in Nosology, arrangement being the least important; for each arrangement will have its defects, will present its deficiencies, and exhibit some forced approximations.” Every plan of arrangement ought, therefore, to be accepted for what it is worth, and appreciated at its true value—namely, as to how far it fulfils the object for which it was mainly devised. Cullen, also, in his lectures and in his writings on this subject, everywhere speaks with the utmost modesty and diffidence, and endeavours at all times to impress upon the mind the fact that Nosology, like other branches of medical science, must necessarily be progressive in its advancement; and that it is only by frequent and multiplied trials that it can be brought to any degree of perfection. A perfectly philosophical or natural system of classification aims at having the details of its plan to agree in every respect with all the facts as they exist in nature. To effect this end, arrangements, as they *naturally* exist, require to be traced out, not devised. The tracks in which such a pursuit must be followed up, and in which our knowledge is as yet deficient, may be shortly indicated under the following heads, namely:—(1.) The affinities or alliances of diseases with each other. (2.) The minute textural morbid anatomy of diseased parts. (3.) The communication, propagation, inoculation, generation, development,

course, and spontaneous natural termination of diseases. (4.) The connection of the phenomena recognised during life with the facts of morbid anatomy. (5.) The geographical distribution of diseases. (6.) The succession of diseases, so far as they can be traced through past ages; the peculiarities they have exhibited at different periods in the world's history, or within comparatively recent cycles of years. But the time has not yet come for a classification on a basis so comprehensive—simply because the material does not yet exist; and attempts to make so-called *natural* systems of arrangement must end in disappointment, on account of the uncertain and fluctuating data on which they must be based.

Present State and Aim of Nosology.—The most distinguished Physicians and Statists recently lent their aid to obtain a nomenclature and classification of diseases which could be applied to the wants of the civil and military population in every country. Dr. William Farr devised a system of Nosology which was discussed at several meetings of the Statistical Congress of the Great Powers of Europe, convened for the purpose, amongst other business, of devising and adopting a uniform system of nomenclature for recording diseases and the causes of death from them. The Congress met in Paris on the 10th of September, 1855, when a nomenclature of the causes of death was agreed upon, essentially the same as that used in England and Geneva. At a third Conference, held at Vienna, in 1857, a *nomenclature* substantially uniform was agreed upon for adoption in all the States of Europe; and fatal cases were to be registered on a uniform plan. Dr. Farr's system of nomenclature was in use for many years by the Registrar-General of this country, and by the Army Medical Department. It was also adopted in the first four editions of this Text-book; and although it had its imperfections and defects, it was still practically the most useful and authoritative Nosology; and "in the English list of names," proposed and adopted ten years ago (*i.e.*, 1869) by the College of Physicians, "it seemed desirable that as little deviation as possible should be made from those employed by the Registrar-General of England; otherwise his settled plans, and his forms of returns, which have been followed for years, would require to be re-modelled; the comparison of future years with past returns would be made difficult and perplexing, if not impossible; and a damaging break would be caused in evidence which becomes more and more trustworthy and valuable in proportion as it is prolonged and continuous." Accordingly, the important task of devising a "Provisional Nomenclature and Definition of Diseases," consistent with the progress of medical science, engaged the attention of a Committee of the Royal College of Physicians of London, during the ten years from 1857 to 1867; and after many interruptions and much consideration, the Committee at last completed their work, and submitted a Provisional Nomenclature to the College, on the 3rd of August, and again on the 28th of November, 1867, when it was unanimously adopted, and published. This was a great achievement. The Registrars-General of England, Scotland, and Ireland, the chiefs of the Medical Department of the Army, Navy, and British troops in India, all concurred with others in framing the Nomenclature; and therefore it was not unreasonably expected that greater accuracy, certainty, and uniformity, for comparison, than hereto-

fore would characterise the statistical records of disease, alike in civil life and in the public services. To facilitate the work becoming *international*, the College translated its nomenclature into Latin, French, German, and Italian equivalents.

The methods of gradually improving the Nomenclature of Diseases in Medicine have some analogy to the methods of gradually improving Representative Reform in Politics. A nomenclature of diseases and a policy of parliamentary representation, judicious and proper a quarter of a century ago, must each eventually give place to the influence of progressive knowledge and power, which invariably come with the rapid movements of the age in which we live. But reform, whether of political representation or of medical nomenclature, to be generally acceptable, cannot be the work of one man, nor the accomplishment of a limited period of time. To be on a broad basis and free of prejudice, each work ought to be the combined result of the best men of the time—each man being willing to yield, adapt, and mould his convictions on entering into a mutual arrangement to achieve a common end. Men thus brought together, who differ very seriously as to certain points, may yet, by mutual discussion and concession, come to a definite and reasonable agreement for practical purposes—the terms of the compromise being settled, doubtless, at the expense of some personal bias, which often has a firmer hold the more imperfect the information of the holder, but which mutual deliberation clears away. Knowing how biassed in opinion individual members of professional and political bodies are apt to be, the unanimous adoption of a Provisional Nomenclature by the London College of Physicians was undoubtedly a great and a bold achievement; and although essentially a compromise of conflicting, and sometimes even of opposing views, the result is a work beyond all praise. It is thus, and thus only, that, in an uncertain science like medicine, any approach to truth can be attained.

The plan of the "Nomenclature" is, first, "*To give an English name to the disease, employing the terms in popular use whenever they are not absolutely inaccurate; and to use only one word, or as few words as possible, in NAMING a disease.*" Definitions have been attached to the English names in some instances only, as where there might be some ambiguity as to the signification which the College desires to attach to them. Thus the definitions have been framed for the purpose of identification only, not as explanations of the phenomena of disease. Secondly, *The classification adopted is based upon anatomical considerations, GENERAL DISEASES, or such as affect the whole frame (subdivided into Sections A and B), and LOCAL DISEASES comprehend the classification.* "Section A comprehends those disorders which appear to involve a morbid condition of the blood, and which present for the most part, but not all of them, the following characters:—*They run a definite course, are attended with fever, and frequently with eruptions on the skin, are more or less readily communicable from person to person, and possess the singular and important property of generally protecting those who suffer them from a second attack. They are apt to occur epidemically.*" Some of the so-called Zymotic diseases of Dr. Farr are comprehended in this classification. "Section B comprises, for the most part, *disorders which are apt to invade different parts of the same*

body simultaneously or in succession. These are sometimes spoken of as constitutional diseases, and they often manifest a tendency to transmission by inheritance." Some of the *Constitutional diseases* of Dr. Farr are comprehended in this classification.

Many physicians may regret the absence of those classes of disease familiar to them ever since they commenced the study of medicine, and which they learned from Cullen's *Nosology*, then the safest guide to classification which the student could follow. But the conflict of opinion rendered it impossible (we are told) for the Committee of the College to subdivide into smaller groups the larger divisions of diseases which have been adopted. To each individual, therefore, and especially to each teacher of medicine, must be left the formation of such groups as may facilitate to himself and to his pupils the study of the science from that particular standpoint which forms the groundwork of his study or his teaching (*Med.-Chir. Rev.*, October, 1869). No fact could be predicated of any number of diseases which was not either too wide in its comprehensiveness, or too narrow in its exclusiveness, or which did not imply a theory which might have been found to be true of certain members of a group, but could only be applied theoretically to the remainder. Therefore the Committee abandoned all attempts to subdivide the diseases into groups, beyond arranging under the two Sections A and B all that ought strictly to be called *general* diseases. To give any definition of either section was also found impossible, because no definition would absolutely embrace the whole of its members, and yet seem to exclude those belonging to the other division. A conception of the dominant character of each section would be more readily found by a simple enumeration of its constituent members, than by any exposition of it in the set terms of a definition. Thus the general diseases of the College embrace the original classes (1) and (2) of Dr. Farr's classification. The local diseases follow nearly the same order as Class (3) of Dr. Farr (p. 332, *ante*). The primary distinction between the *general* and *local* diseases is recognised in this, that the first comprehends those which more or less pervade the whole system, and in which any local affection, whether essential or accidental, is due to the previous existence of some general malady; while the second, or local diseases, are those which affect certain organs, and in which, when any other part of the body is involved, it is so only as a consequence of the primary disorder or local lesion.

In studying the Science of Medicine systematically, a *METHODICAL NOSOLOGY* ought to be regarded as a table of reference to aid the student in naming diseases, and so preserving uniformity in his records and diagnoses, and a system to guide him generally in acquiring a knowledge of his profession, especially with reference to the practical questions of the day. The nosology of the Royal College of Physicians of London ought, therefore, to be accepted simply as a contrivance to aid us in giving the same name to similar conditions of disease, and "for perfecting the statistical registration of diseases, with a view to the discovery of statistical truths concerning their history, nature, and phenomena." *The nomenclature* takes no note of causation, it deals only with the pathological fact, and the arrangement is really, in its main features, anatomical—the organs are viewed as portions of tissue, which have deviated from the

healthy standard. Such a nomenclature of diseases as that which the College recommended for general adoption, is a great boon alike to the medical profession and to the public; and as it still enables future registration to be compared with the past registration of the Registrar-General, it may gradually and eventually supersede that and other systems for the registration of disease in Civil Hospitals, as it has already done in the Public Services. Numbers are introduced with each disease for the purpose of easy reference in double entries. But practically there is sometimes a difficulty here, as shown by my friend Dr. Steele, in the application of the nomenclature and classification to the registration of diseases in hospitals such as Guy's. In framing the report of that hospital for 1868, and in the attempt to adhere to the *numerical* arrangement in the construction of the statistical tables, Dr. Steele found himself frequently at a loss for a numerical indicator corresponding to the particular complaint. This arose from two causes,—the first depending on the exclusively anatomical character of the terminology, which does not provide for diseases of a duplicate or multiple character, such as *broncho-pneumonia*, *pneumonia-typhus*, *pleuro-pneumonia*, *disease of joint*, &c.; and the other attributable to the too rigid definitions which, in many cases, and then not always, could only hope to be revealed by *post-mortem* examination. Many obscure affections of the *heart* and *brain* can only be entered as "*disease of heart, or brain*," as the case may be, without corresponding numbers, to escape the possibility of "reckless conjecture" in the absence of a *post-mortem examination*. In the registration of disease in an hospital, a column should be added to the disease table, to be filled in from the *post-mortem* register (as has been done by Dr. Steele), indicating the more immediate cause of death. Such is especially useful where a difficulty has been experienced during life in selecting, fixing upon, or specifying any one particular form of disease, where two or more have contributed to the fatal issue. Dr. Steele's statistical tables, which have been annually published, for the past twenty-six years, relative first to the Glasgow Infirmary, and subsequently to Guy's Hospital, London, are models of the construction of such tables for hospital registration, and of the resulting information to be obtained from them.

The Science of Pathology, we know, is yet too young to base a scientific classification upon it; but as Pathology and the Art of Practical Medicine advance, so must Nosology. There are many nice questions which always will arise, relative to the nature of diseases, on which it is in vain to expect physicians and statisticians to agree unanimously; and therefore no system even of naming, far less of classifying, the diseases of mankind can we hope to see otherwise than as a *provisional* one, ready to assimilate itself to the progressive advance of the Science of Medicine—a plant of slow but of sure growth. There is every reason to hope, however, that, by the numerous inquisitive researches of the day, Pathology and Nosology will grow even more rapidly than hitherto. The mere enumeration of diseases has almost doubled since Cullen's Nosology was written; while our knowledge of facts relating to disease has greatly more than doubled. Cullen's Nosology became effete and useless at last, under the pressure of increasing

knowledge acquired and effected with resources very inferior to those we now possess, and far less extensive. The nomenclature and classification promulgated ten years ago by the College of Physicians, was therefore intended to be *strictly provisional*, and "*subject to decennial revision*," as stated on the title-page. It will be well, then, if the Colleges of Physicians and Surgeons in Scotland and in Ireland (who unfortunately do not seem to have taken part in the preparation of the nomenclature) would unite with that of London in this eminently practical work, and appoint committees to communicate with each other in revising and re-adjusting such nomenclature at the end of every ten years; and so stamp with their united authority the progressive improvements in the Science of Medicine which are capable of being indicated or expressed in the Nomenclature and Definitions of diseases. Such systematic arrangements, if consistent with existing knowledge, never cramp or hamper a man in carrying out scientific investigations; on the contrary, they enable him to see more clearly in what direction his labour must be advanced, and demonstrate more forcibly than otherwise the deficiencies of his knowledge. The time has now come again (1879) for a revision of the Nomenclature.

The "Provisional Nomenclature" of the Royal College of Physicians of London, comprehended in the following list, has therefore been again adopted, in the text of this the *seventh*, as originally given in the two previous editions; while the synonyms, equivalents, and definitions have also been incorporated at the places where the diseases are described in the text throughout both volumes.

CHAPTER II.

"THE NOMENCLATURE OF DISEASES" DRAWN UP BY A JOINT COMMITTEE
APPOINTED BY THE ROYAL COLLEGE OF PHYSICIANS OF LONDON
(1869).

GENERAL DISEASES.

SECTION A.

- | | | |
|--|---|---|
| <ol style="list-style-type: none"> 1. Small-pox.
<i>Group A</i> (unmodified).
<i>Group B</i> (modified).
<i>Varieties</i>, applicable to both groups:
<i>a.</i> Confluent.
<i>b.</i> Semi-confluent.
<i>c.</i> Distinct. <i>Syn.</i>, Discrete.
<i>d.</i> Abortive. <i>Syn.</i>, Varicelloid.
<i>Subordinate Varieties:</i>
<i>e.</i> Potechial.
<i>f.</i> Hæmorrhagic.
<i>g.</i> Corymbosæ. 2. Cow-pox; 3. Chicken-pox. 4. Measles. 5. Scarlet fever. <i>Syn.</i>, Scarlatina.
<i>Varieties:</i>
<i>a.</i> Simple.
<i>b.</i> Anginose.
<i>c.</i> Malignant. 6. Dengue. 7. Typhus fever. 8. Cerebro-spinal fever. <i>Syn.</i>, Malignant purpuric fever; | <ol style="list-style-type: none"> Epidemic cerebro-spinal meningitis. 9. Enteric fever. <i>Syn.</i>, Typhoid fever; and in children often named Infantile remittent fever. 10. Relapsing fever. 11. Simple continued fever. 12. Febricula. 13. Yellow fever. 14. Plague. 15. Ague. <i>Syn.</i>, Intermittent fever.
<i>Varieties:</i>
<i>a.</i> Quotidian.
<i>b.</i> Tertian.
<i>Sub-variety:</i>
Double tertian.
<i>c.</i> Quartan.
<i>Sub-variety:</i>
Double quartan.
<i>d.</i> Irregular.
(10b.) <i>Brow Ague.</i>¹ 16. Remittent fever. 17. Simple cholera. 18. Malignant cholera. <i>Syn.</i>, Ser- | <ol style="list-style-type: none"> ous cholera, Spasmodic cholera, Asiatic cholera. <i>a.</i> Choleraic diarrhoea. 19. Diphtheria.
<i>a.</i> Diphtheritic paralysis. 20. Hooping-cough. 21. Mumps. 22. Influenza. 23. Glanders. 24. Farcy. 25. Equinia mitis. <i>Syn.</i>, Grease. 26. Malignant pustule. 27. Phagedæna. 28. Sloughing phagedæna. 29. Hospital gangrene. 30. Erysipelas.
<i>Varieties:</i>
<i>a.</i> Simple. <i>Syn.</i>, Cutaneous.
<i>b.</i> Phlegmonous. <i>Syn.</i>, Cellulo-cutaneous.
<i>c.</i> Diffuse inflammation (of cellular tissue). 31. Pyæmia. 32. Puerperal fever. 33. Puerperal ephemera. <i>Syn.</i>, Weed. |
|--|---|---|

SECTION B.

- | | | |
|--|---|---|
| <ol style="list-style-type: none"> 31. Acute rheumatism. <i>Syn.</i>, Rheumatic fever.
(<i>a.</i>) Sub-acute rheumatism. 35. Gonorrhœal rheumatism. 36. Synovial rheumatism. 37. Muscular rheumatism.
<i>Local varieties:</i>
<i>a.</i> Lumbago.
<i>b.</i> Stiff neck. 38. Chronic rheumatism. 39. Acute gout. 40. Chronic gout. 41. Gouty synovitis. 42. Chronic osteo-arthritis. <i>Syn.</i>, Chronic rheumatic arthritis. 43. Syphilis.
<i>A.</i> Primary syphilis.
<i>Varieties:</i>
Hard chancre.
Indurated bubo.
Soft chancre.
Suppurating bubo.
Phagedænic sore.
Sloughing sore.
<i>b.</i> Secondary syphilis.
<i>c.</i> Hereditary syphilis. 1. Local syphilitic affections. 44. Cancer. <i>Syn.</i>, Malignant disease.
<i>Varieties:</i>
<i>a.</i> Scirrhus. <i>Syn.</i>, Hard cancer. | <ol style="list-style-type: none"> <i>b.</i> Medullary cancer. <i>Syn.</i>, Soft cancer. <i>c.</i> Epithelial cancer. <i>Syn.</i>, Canceroid epithelioma. <i>d.</i> Melanotic cancer. <i>Syn.</i>, Melanosis. <i>e.</i> Osteoid cancer. 1. Local cancer. 45. Colloid. <i>Syn.</i>, Colloid cancer; Alveolar cancer. 1. Local colloid. <p>ARRANGEMENT OF NON-MALIGNANT TUMORS and CYSTS.</p> <p>Fibrous tumour.
Fibro-cellular tumors.
Fibro-nucleated tumor.
Fibro-plastic tumor.
Myeloid tumor.
Fatty tumor. <i>Syn.</i>, Lipoma.
Osseous tumor.
<i>a.</i> Of bone. <i>Syn.</i>, Exostosis.
<i>Varieties:</i>
1. Ivory.
2. Cancellated.
3. Diffused.
<i>b.</i> Of the soft parts.
Cartilaginous tumor. <i>Syn.</i>, Enchondroma.
Fibro-cartilaginous tumor. </p> | <p>Glandular tumor. <i>Syn.</i>, Adenocoele.
Vascular tumor.
Nævus.
Sebaceous tumor.
Cholesteatoma.
Molluscum.
Warty tumor and warts.
Condyloma.
Cheloid.
Villous tumor.
Simple or barren cysts.
<i>a.</i> Serous.
<i>b.</i> Synovial. <i>Syn.</i>, Bursal.
<i>c.</i> Mucous.
<i>d.</i> Suppurating.
<i>e.</i> Sanguineous.
<i>f.</i> Hæmorrhagic.
<i>g.</i> Aneurismal.
<i>h.</i> Oily.
<i>i.</i> Colloid or gelatinous.
<i>j.</i> Seminal.
Compound or proliferous cysts.
<i>a.</i> Complex cystic tumor. <i>Syn.</i>, Cysto-sarcoma.
1. With intracystic growths.
<i>b.</i> Cutaneous or piliferous cyst. <i>Syn.</i>, Dermoid.
<i>c.</i> Dentigerous cyst. 46. Lupus. </p> |
|--|---|---|

¹ The diseases printed in italics are to be returned, not among the local diseases, but under the headings referred to by number within brackets.

- Varieties:*
a. Chronic lupus.
b. Lupus exedens.
47. Rodent ulcer.
48. True leprosy. *Syn.*, Elephantiasis Græcorum.
49. Scrofula.
Varieties:
a. Scrofula with tubercle.
b. Scrofula without tubercle.
1. Local scrofulous affections.
 Tubercular meningitis.
 Scrofulous ophthalmia.
- Tubercular pericarditis.
 Scrofulous disease of glands.
 Phthisis pulmonalis.
 Hæmoptysis.
 Acute miliary tuberculosis.
 Tabes mesenterica.
 Tubercular peritonitis.
50. Rickets.
51. Cretinism.
Varieties:
a. Complete cretinism. *Syn.*, Incurable cretinism.
b. Incomplete cretinism. *Syn.*, Curable cretinism.
52. Diabetes. *Syn.*, Diabetes mellitus.
 (935a.) *Ergotism.*
53. Purpura.
Varieties:
a. Simple.
b. Hæmorrhagic.
54. Scurvy.
55. Anæmia.
56. Chlorosis. *Syn.*, Green sickness.
57. General dropsy.
58. Beri-Beri.

LOCAL DISEASES.¹ARRANGEMENT OF LOCAL DISEASES.²

- | | | |
|--------------------------------|-----------------------------------|-----------------------------|
| Catarrh. | Dropsy. | (43.) Syphilitic disease. |
| Inflammation. | Fibrinous deposit. | (44.) Cancer. |
| Ulcerative inflammation. | Alteration of dimensions. | (45.) Colloid. |
| Suppurative inflammation. | Dilatation. | Non-malignant tumors. |
| Plastic inflammation on. | Contraction. | Cyst. |
| (51.) Pyæmic inflammation. | Hypertrophy. | (49.) Scrofula. |
| Rheumatic inflammation. | Atrophy. | (49a.) a. With tubercle. |
| Gouty inflammation. | Degeneration. | (49b.) b. Without tubercle. |
| (43.) Syphilitic inflammation. | Fatty and calcareous. | Parasitic disease. |
| (49.) Scrofulous inflammation. | <i>Syn.</i> , Atheroma. | Calculus and concretion. |
| Gonorrhœal inflammation. | Ossification. | Malformation. |
| Gangrene. | Fibroid. | (992, &c.) Injury. |
| Passive congestion. | Lardaceous disease. <i>Syn.</i> , | 1014, &c.) Foreign body. |
| Extravasation of blood. | Amyloid disease, Waxy disease. | Functional diseases. |
| Hæmorrhage. | | |

DISEASES OF THE NERVOUS SYSTEM.¹

- | | | |
|--|--|---|
| DISEASES OF THE BRAIN AND ITS MEMBRANES. | 74. Diseases of the cerebral arteries. | 85. 2. Hemiplegia. |
| 59. Encephalitis. | <i>a.</i> Fatty and calcareous degeneration. <i>Syn.</i> , Atheroma, Ossification. | 86. 3. Paraplegia. |
| 60. Meningitis. | <i>b.</i> Aneurism. | 87. 4. Locomotor ataxy. |
| 1. Inflammation of the dura mater. | <i>c.</i> Impaction of coagula. | (797.) 5. Progressive muscular atrophy. |
| 2. Inflammation of the pia mater and arachnoid. | 1. Thrombosis (Local coagulation). | 88. 6. Infantile paralysis. |
| 49.) 3. Tubercular meningitis. <i>Syn.</i> , Acute hydrocephalus. | 2. Embolism (Coagula conveyed from a distance) | 89. 7. Local paralysis. |
| (8.) Cerebro-spinal fever. | | <i>a.</i> Facial paralysis. |
| 61. Inflammation of the brain. | DISEASES OF THE SPINAL CORD AND ITS MEMBRANES. | <i>b.</i> Scrivener's palsy. |
| 62. Red softening (of the brain). | 75. Inflammation. | (19a.) 8. Diphtheritic paralysis. |
| 63. Yellow softening (of the brain). | <i>Varieties:</i> | (908b.) 9. Lead palsy. |
| 64. Abscess (of the brain). | <i>a.</i> Spinal meningitis. | (966a.) 10. Paralysis from Lathyrus. |
| 65. Apoplexy. | <i>b.</i> Myelitis. | |
| <i>Varieties:</i> | 76. Hæmorrhage (Spinal). <i>Syn.</i> , Spinal apoplexy. | FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM. |
| <i>a.</i> Congestive. | 77. Atrophy (Spinal). <i>Syn.</i> , Tabes dorsalis. | 90. Tetanus. |
| <i>b.</i> Sanguineous. <i>Syn.</i> , Cerebral hæmorrhage. | 78. White softening (of the Spinal cord). | 91. Hydrophobia. |
| 66. Sunstroke. | (44.) Cancer. | 92. Infantile convulsions. |
| 67. Chronic hydrocephalus. | 79. Non-malignant tumors. | 93. Epilepsy. |
| 68. Hypertrophy (of the brain). | <i>Return such tumors here according to the list at p. 339.</i> | <i>a.</i> Epileptic vertigo. <i>Syn.</i> , Petit mal. |
| 69. Atrophy (of the brain). | 80. Malformations. | 94. Convulsions. |
| 70. White softening (of the brain). <i>Syn.</i> , Atrophic softening. | <i>Return such cases here according to the list at pp. 252 to 254.</i> | 95. Spasm of muscle. |
| (43.) Syphilitic disease. | <i>a.</i> Spina bifida. | 96. Laryngismus stridulus. <i>Syn.</i> , Spasm of the glottis, Spasmodic croup, Child-crowing. |
| (44.) Cancer. | DISEASES OF THE NERVES. | 97. Shaking palsy. |
| 71. Fibrous tumor. | 81. Inflammation. | (907a.) <i>Mercurial tremor.</i> |
| 72. Osseous tumor. | 82. Atrophy. | 98. Chorea. <i>Syn.</i> , St. Vitus's dance. |
| (49.) Tubercular deposit. | (44.) Cancer. | <i>a.</i> Acute. |
| <i>a.</i> Military or granular tubercle. | 83. Neuroma. | <i>b.</i> Chronic. |
| <i>b.</i> Yellow tubercle. | 84. Paralysis. | 99. Hysteria. |
| 73. Parasitic disease. | (108.) 1. Paralysis of the insane. <i>Syn.</i> , General Paralysis. | 100. Catalepsy. |
| <i>Return cases of this class according to the list at pp. 148 to 150.</i> | | (243.) Syncope. |
| 73*. Malformations. | | 101. Neuralgia. |
| <i>Return such cases here according to the list at pp. 252 to 254.</i> | | <i>Principal Varieties:</i>
<i>a.</i> Facial. <i>Syn.</i> , Tic douloureux.
<i>b.</i> Brow-ague. <i>Syn.</i> , Hemispheralgia.
<i>c.</i> Sciatica. |

¹ Register the diseases printed in italics, not under this heading, but at the place referred to in each instance, by number within brackets.

² The Local Diseases have been drawn up in accordance with the above arrangement, and the nature of their lesions has been described in the first part of this Text-book, from p. 51 to the end of Part I., as "Topics relative to Pathology."

- d. Pleurodynia.
e. Irritable stump.
102. Hyperæsthesia.
103. Anæsthesia.
(938a.) *Delirium tremens.*
104. Hypochondriasis.

- DISORDERS OF THE INTELLECT.
105. Mania.
a. Acute mania.
b. Chronic mania.
106. Melancholia.
107. Dementia.

- a. Acute dementia.
b. Chronic dementia.
108. Paralysis of the insane.
Syn., General paralysis.
109. Idiocy (Congenital).
110. Imbecility (Congenital).

DISEASES OF THE EYE.¹

DISEASES OF THE CONJUNCTIVA.

111. Conjunctivitis. *Syn.*, Ophthalmia.
112. Catarrhal ophthalmia.
113. Pustular ophthalmia.
114. Purulent ophthalmia.
115. Purulent ophthalmia of infants. *Syn.*, Ophthalmia neonatorum.
(49.) *Scrofulous ophthalmia.* *Syn.*, *Strumous ophthalmia.*
116. Exanthematous ophthalmia.
117. Gonorrhœal ophthalmia.
118. Chronic ophthalmia.
119. Œdema of the sub-conjunctival tissue. *Syn.*, Chemosis.
120. Pinguecula.
121. Pterygium.
122. Fatty tumor.
123. Parasitic disease.
Return cases of this class according to the list at p. 148.
124. Metallic stains.
a. From nitrate of silver.
b. From lead.

DISEASES OF THE CORNEA.

125. Keratitis.
126. Chronic interstitial keratitis.
127. Keratitis with suppuration.
Syn., Onyx.
128. Ulcer.
129. Opacity. *Syn.*, Leucoma.
130. Conical cornea.
131. Arcus senilis.
132. Staphyloma.
133. Parasitic disease in the anterior chamber.
Return cases of this class according to the list at p. 148.

DISEASES OF THE SCLEROTIC.

134. Sclerotitis.
135. Staphyloma.

DISEASES OF THE IRIS.

136. Iritis.
137. Traumatic iritis.
138. Rheumatic iritis.
139. Arthritic iritis.
(43.) *Syphilitic iritis.*
(49.) *Scrofulous iritis.*
140. Gonorrhœal iritis.

141. Sequelæ of iritis.
142. Malformations.
Return such cases here according to the list at p. 252.

DISEASES OF THE CHOROID AND RETINA.

143. Choroiditis.
144. Retinitis.
145. Choroidal apoplexy.
146. Amaurosis.
147. Impaired vision.
148. Muscæ volitantes.
149. Albinism.

DISEASES OF THE VITREOUS BODY.

150. Synchysis.
151. Various morbid deposits.

DISEASES OF THE LENS AND ITS CAPSULE.

152. Cataract.
Varieties:
a. Hard.
b. Soft.
c. Fluid.
153. Parasitic disease.
Return cases of this class according to the list at p. 148.
154. Malformations.
Return such cases here according to the list at p. 252.
a. Congenital cataract.
155. Traumatic cataract.

GENERAL AFFECTIONS OF THE EYE.

156. Glaucoma.
157. Hydrophthalmia.
(44.) *Cancer.*
(49.) *Scrofulous deposit within the eyeball.*
158. Total disorganisation of the eye from injury.
158*. Malformations.
Return such cases here according to the list at p. 252.

VARIOUS DEFECTS OF SIGHT.

159. Short sight.
160. Long sight.
161. Faulty perception of colours.

Syn., Colour blindness.

162. Hemeralopia.
163. Nyctalopia.
164. Astigmatism.

DISEASES OF THE LACHRYMAL APPARATUS.

165. Lachrymal obstruction.
166. Abscess and fistula.
167. Dacryolith.
168. Diseases of the lachrymal gland and its ducts.

DISEASES OF THE EYELIDS.

169. Inflammation.
170. Hordeolum.
171. Abscess in the Meibomian glands.
172. Epicanthis.
173. Entropium.
174. Ectropium.
175. Trichiasis.
176. Madarosis. *Syn.*, Loss of the eyelashes.
177. Tarsal ophthalmia.
178. Blepharospasmus.
(44.) *Cancer.*
179. Cyst of the lids.
(895.) *Phthiriasis.*
179*. Malformations.
Return such cases here according to the list at p. 252.

DISEASES WITHIN THE ORBITS.

180. Abscess in the orbit.
181. Strabismus.
182. Protrusion of the eyeball.
Syn., Proptosis.
(282.) *Exophthalmic bronchocele.*
(250.) *Orbital aneurism.*
(44.) *Cancer.*
183. Non-malignant tumors.
Return such tumors here according to the list at p. 339.
184. Parasitic disease.
Return cases of this class according to the list at p. 148.
185. Affections of the orbital nerves.
Injuries of the Eye are given at p. 352.

DISEASES OF THE EAR.¹

DISEASES OF THE AURICLE.

186. Gouty and other deposits.
187. Hematoma auris.
(44.) *Cancer.*
188. Non-malignant tumors.
Return such tumors here according to the list at p. 339.
(827. &c.) *Cutaneous affections.*
189. Malformations.
Return such cases here according to the list at p. 252.
(1012.) *Injuries.*

DISEASES OF THE EXTERNAL MEATUS.

190. Inflammation.
a. Acute.
b. Chronic.
191. Abscess.
192. Accumulation of wax.
193. Polypus.
194. Sebaceous tumor. *Syn.*, Molluscous tumor.
195. Osseous tumor of bone. *Syn.*, Exostosis.
195*. Malformations.

- Return such cases here according to the list at p. 252.*
(1014.) *Foreign bodies.*

DISEASES OF THE MEMBRANA TYMPANI.

196. Inflammation.
197. Ulceration.
198. Perforation.
(1012.) *Injuries.*

DISEASE OF THE EUSTACHIAN TUBE.

199. Obstruction.

¹ Register the diseases printed here in *Italics*, not under this heading, but at the place referred to in each instance by number within brackets.

DISEASES OF THE TYMPANUM.

200. Disease of the mucous membrane.
201. Disease of the ossicles.
202. Disease of the mastoid cells.

DISEASES OF THE INTERNAL EAR.

203. Organic disease.
204. Necrosis of the petrous bone.
205. Deafness.
Varieties:
a. Functional or nervous.

- b. From disease.
c. Deaf-dumbness.

- (44.) *Cancer.*
205^b. Malformations.
Return such cases according to the list at p. 252.

DISEASES OF THE NOSE.¹

206. Hypertrophy. *Syn.*, Lipoma.
207. Wart.
208. Sebaceous cyst.
(44.) *Cancer of the skin.*
(46.) *Lupus.*
209. Ozena.
210. Ulceration of the pituitary membrane.
211. Abscess of the septum.
212. Perforation of the septum.
213. Epistaxis.

214. Hypertrophy of the pituitary membrane.
(44.) *Cancer.* *Syn.*, *Malignant polypus.*
215. Polypus nasi.
Varieties:
a. Gelatinous.
b. Fibrous.
1. Naso-pharyngeal polypus.

216. Non-malignant tumors of the septum.
217. Rhinoliths.
217*. Malformations.
Return such cases here according to the list at pp. 252 to 254.
(1015.) *Foreign bodies.*
218. Loss or perversion of the sense of smell.

DISEASES OF THE CIRCULATORY SYSTEM.¹

DISEASES OF THE HEART AND ITS MEMBRANES.

DISEASES OF THE PERICARDIUM.

219. Pericarditis.
220. Suppurative pericarditis.
(49.) *Tubercular pericarditis.*
221. Adherent pericardium.
222. Dropsy.
(44.) *Cancer.*
223. Malformations.
Return such cases here according to the list at p. 252.
(1056.) *Injuries.*

DISEASES OF THE ENDOCARDIUM.

224. Endocarditis.
225. Valve-disease.
1. Aortic.
2. Mitral.
3. Pulmonic.
4. Tricuspid.
Varieties:
a. Vegetations.
b. Fibroid thickening.
c. Fatty and calcareous degeneration.
Syn., Atheroma, Ossification.
d. Aneurism.
e. Laceration.
f. Simple dilatation of orifice.
g. Malformations.
Return such cases here according to the list at p. 252.
Obstruction to the circulation and Regurgitation should be specially noted when they accompany the valve-disease.
226. Fibrinous concretions in the cavities of the heart.

DISEASES OF THE MUSCULAR STRUCTURE OF THE HEART.

227. Myocarditis.
228. Abscess.
229. Hypertrophy.
a. Of left side.
b. Of right side.

230. Dilatation.
a. Of left side.
b. Of right side.
231. Atrophy.
232. Excess of fat
233. Fatty degeneration.
234. Fibroid degeneration.
235. Aneurism.
236. Acute aneurism.
237. Rupture.
(44.) *Cancer.*
238. Parasitic disease.
Return cases of this class according to the list at p. 148
239. Disease of the coronary arteries.
240. Malformations.
Return such cases here according to the list at p. 252.
241. Cyanosis.
(1056-58.) *Injuries of the heart.*
242. Angina pectoris.
243. Syncope. *Syn.*, Fainting fit.
244. Palpitation and irregularity of the action of the heart.

DISEASES OF THE BLOOD-VESSELS.

(a.) DISEASES OF THE ARTERIES.

245. Arteritis.
246. Fatty and calcareous degeneration. *Syn.*, Atheroma, Ossification.
247. Narrowing and obliteration.
248. Occlusion.
a. From compression.
b. From impaction of coagula.
1. Thrombosis (local coagulation).
2. Embolism (coagula conveyed from a distance).
249. Dilatation.
250. Aneurism.
In returning such cases, state whether the aneurism be—
a. Fusiform,
b. Saccular, or
c. Diffused (sac formed by the surrounding tissues)
251. Rupture of artery.

- a. From disease of artery.
b. From disease external to artery.
252. Partial rupture of artery.
Syn., Dissecting aneurism.
253. Traumatic aneurism.
254. Arterio-venous aneurism.
255. Aneurismal varix.
Varieties:
a. Traumatic.
b. Spontaneous.
256. Varicose aneurism.
Varieties:
a. Traumatic.
b. Spontaneous.
257. Cirroid aneurism. *Syn.*, Arterial varix.
258. Aneurism by anastomosis.
259. Malformations.
Return other cases of this class here according to the list at p. 252.
a. Commencement of the descending aorta (contracted or obliterated).
(1009, &c.) *Injuries of arteries.*
Contusion.
Laceration.
a. Of the whole vessel.
b. Of the outer coat.
c. Of the inner coat.
Wound.

(b.) DISEASES OF THE VEINS.

260. Phlebitis.
Varieties:
a. Adhesive.
b. Suppurative.
261. Phlegmasia dolens.
262. Fibrinous concretions in the veins.
263. Obstruction.
264. Obliteration.
265. Phleboliths.
266. Varicose veins.
267. Nævus vascularis.
268. Parasitic disease.
Return cases of this class according to the list at p. 149, Nos. 28 and 30.
(1009, &c.) *Injuries of veins.*
Rupture, without external wound.
Wound of vein, with entrance of air.

¹ Register the diseases printed here in *Italics*, not under this heading, but at the place referred to in each instance by number within brackets.

DISEASES OF THE ABSORBENT SYSTEM.¹

269. Inflammation of lymphatics.
 270. Suppuration of lymphatics.
 271. Inflammation of glands.
 272. Suppuration of glands.
 273. Hypertrophy of glands.
 a. Chronic enlargement of glands.
 274. Atrophy of glands.
 275. Lymphatic fistula.
 (1142.) Foreign bodies and concretions.

276. Obstruction of the thoracic duct.
 277. Obstruction, obliteration, and varicosity of lymphatics.
 278. Bursting of lymphatics.
 (43.) Syphilitic bubo.
 (43.) Syphilitic inflammation of glands.
 (41.) Cancer.
 (49.) Scrofulous disease of glands.
 (49.) Suppuration.

- (1143.) Wound of lymphatics.

DISEASES OF THE BRONCHIAL GLANDS.

- (340.) Inflammation.
 (341.) Abscess.
 (342.) Enlargement.
 (44.) Cancer.
 (343.) Non-malignant tumors.
 (49.) Tubercle.

DISEASES OF THE DUCTLESS GLANDS.¹

DISEASES OF THE THYROID GLAND.

279. Inflammation.
 a. Acute.
 b. Chronic.
 280. Goitre.
 281. Cyst.
 282. Exophthalmic bronchocele.
 283. Pulsating bronchocele.

- (44.) Cancer.

DISEASES OF THE THYMUS GLAND.

284. Hypertrophy.
 (44.) Cancer.
 285. Non-malignant tumors.
 Return such tumors here according to the list at p. 339.

DISEASES OF THE SUPRA-RENAL CAPSULES.

- (44.) Cancer.
 (49.) Tubercular degeneration.
 286. Addison's disease, *Syn.*,
 Bronzed skin, Melasma
 Addisoni.

DISEASES OF THE RESPIRATORY SYSTEM.¹

DISEASES OF THE RESPIRATORY SYSTEM NOT STRICTLY LOCAL.

287. Hay asthma.
 (22.) Influenza.
 (20.) Hooping-cough.
 288. Croup.
 (19.) Diphtheria.
 (995.) Asphyxia.

DISEASE OF THE NOSTRILS.

289. Coryza. *Syn.*, Nasal catarrh.

DISEASES OF THE LARYNX.

290. Inflammation of the epiglottis.
 291. Ulceration of the epiglottis.
 292. Laryngeal catarrh.
 293. Laryngitis.
 a. Acute.
 b. Chronic.
 294. Ulcer.
 295. Abscess.
 296. (Edema of the glottis.
 297. Necrosis of cartilage.
 298. Contraction.
 (44c.) Epithelial cancer.
 299. Warty growth.
 300. Polypus.
 301. Cyst.
 301.* Malformations.
 Return such cases here according to the list at p. 252.
 (592-1039.) Injuries.
 (1044.) Foreign bodies in the larynx.
 302. Aphonia.
 303. Paralysis of the glottis.
 304. Spasm of the glottis.
 (96.) Laryngismus stridulus.

DISEASES OF THE TRACHEA AND BRONCHI.

305. Bronchial catarrh.
 306. Bronchitis.
 a. Acute.
 b. Chronic.

307. Ulcer.
 308. Casts of the bronchial tubes.
 309. Necrosis of the cartilages of the trachea.
 310. Dilatation.
 311. Contraction.
 (44.) Cancer.
 312. Non-malignant tumors.
 Return such tumors here according to the list at p. 339.

- (49.) Tubercle.
 313. Parasitic disease.
 Return cases of this class according to the list at p. 149.
 313. Malformations.
 Return such cases here according to the list at p. 252.
 (1044.) Foreign bodies in the bronchi.
 314. Asthma.

DISEASES OF THE LUNG.

315. Pneumonia.
 Variety:
 a. Lobular.
 316. Abscess.
 (31.) Pyemic inflammation and abscess.
 317. Gangrene.
 318. Passive congestion.
 a. Hæmoptysis.
 319. Pulmonary extravasation.
 Syn., Pulmonary apoplexy.
 a. Hæmoptysis.
 320. (Edema.
 321. Cirrhosis.
 322. Emphysema.
 a. Vesicular.
 b. Interlobular.
 323. Atelectasis.
 324. Collapse.
 (43.) Syphilitic deposit.
 (44.) Cancer.
 (49.) Phthisis.
 (49.) Acute miliary tuberculosis.
 325. Acute pneumonic phthisis.
 326. Chronic pneumonic phthisis.
 327. Parasitic disease.

Return cases of this class according to the list at p. 148.

327. Malformations.
 Return such cases here according to the list at pp. 252 to 254.

- (1054-1058.) Injuries.
 (1044.) Foreign bodies.
 328. Millstone-makers' phthisis.
 329. Grinders' asthma.
 330. Miners' asthma.

DISEASES OF THE PLEURA.

331. Pleurisy.
 332. Chronic pleurisy.
 333. Empyema.
 334. Adhesions, including thickening and ossification.
 335. Hydrothorax.
 336. Pneumothorax.
 (41.) Cancer.
 337. Non-malignant tumors.
 Return such tumors here according to the list at p. 339.
 (49.) Tubercular pleurisy.
 (1053-1054.) Injuries.

DISEASES OF THE MEDIASTINUM.

338. Abscess.
 (44.) Cancer.
 339. Non-malignant tumors.
 Return such tumors here according to the list at p. 339.
 (284-285.) Diseases of the thymus gland.

DISEASES OF THE BRONCHIAL GLANDS.

340. Inflammation.
 341. Abscess.
 342. Enlargement.
 (44.) Cancer.
 343. Non-malignant tumors.
 Return such tumors here according to the list at p. 339.
 (39.) Tubercle.

DISEASES OF THE DIGESTIVE SYSTEM.¹

DISEASES OF THE LIPS.

- The affected lip ought to be specified.*
 344. Ulcer.

- (43.) Syphilitic ulcer.
 345. Fissures.
 (44.) Cancer.
 (59.) Scrofulous hypertrophy.
 346. Cyst.

347. Malformations.
 Return such cases here according to the list at p. 252.
 a. Hare-lip.

¹ Register the diseases printed here in *Italics*, not under this heading, but at the place referred to in each instance by number within brackets.

DISEASES OF THE MOUTH.

348. Stomatitis.
 349. Ulcerative stomatitis.
 350. Thrush. *Syn.*, Aphtha, Vesicular stomatitis.
 351. Abscess of the cheek.
 353. Cancrum oris. *Syn.*, Gangrenous stomatitis.
 354. Cyst of the cheek.
 355. Ranula.
 (44.) *Cancer*.
 356. Parasitic disease.
 a. Parasitic thrush. *Syn.*, Parasitic aphthæ.
Return cases of this class according to the list at p. 148.

DISEASES OF THE JAWS, INCLUDING THE ANTRUM.

357. Adhesion of the jaws by cicatrix.
 358. Abscess of the antrum.
 (44.) *Cancer*.
 359. Fibrous tumor.
 360. Myeloid tumor.
 361. Osseous tumor.
 a. Hypertrophy of the bones of the face.
 362. Cartilaginous tumor.
 363. Vascular tumor.
 364. Cyst.
 (1016.) *Foreign bodies in the antrum.*

DISEASES, MALFORMATIONS, AND INJURIES OF THE TEETH, GUMS, AND ALVEOLI.

365. Teething.

DISEASES OF THE DENTAL TISSUE.

366. Caries.
 367. Necrosis.
 368. Exostosis.
 369. Absorption.

DISEASES OF THE DENTAL PULP.

370. Irritation.
 371. Inflammation.
 372. Ulceration.
 373. Gangrene.

DISEASES OF THE DENTAL PERIOSTEUM.

374. Granulation or polypus.
 375. Calcification.
 376. Inflammation.
 377. Gum-boil.
 378. Chronic thickening.
 379. Rheumatic inflammation.

DISEASES OF THE GUMS.

380. Inflammation.
 381. Ulceration.
 382. Hypertrophy.
 383. Atrophy.
 384. Induration (in infancy).
 (44.) *Cancer*.
 385. Non-malignant tumors.
Return such tumors here according to the list at p. 339.
 a. Polypus.
 b. Cartilaginous tumor.
 c. Vascular tumor.
 386. Epulis.

DISEASES OF THE ALVEOLI.

387. Inflammation.
 388. Necrosis.
 389. Caries.

390. Exostosis.
 391. Dentigerous cyst.
 392. Absorption.

SPECIFIC DISEASES AFFECTING THE DENTAL PERIOSTEUM, GUMS, OR ALVEOLI.

393. Mercurial inflammation.
 394. Phosphoric inflammation and necrosis.
 (908c.) *Blue gum from lead.*
 (54.) *Scurvy.*

IRREGULAR DENTITION.

- Irregularity in the time of eruption of the—
 395. Temporary teeth.
 396. Permanent teeth.
 Irregularity in the position of the—
 397. Temporary teeth.
 398. Permanent teeth.
 Irregularity of the number of the—
 399. Temporary teeth.
 400. Permanent teeth.
 Irregularity in the form of the—
 401. Temporary teeth.
 402. Permanent teeth.
 Abnormal development of the—
 403. Dental tissue.
 404. Enamel.
 405. Dentine.
 406. Cementum.
 407. Alveolar portions of the jaws, in size.
 408. Alveolar portions of the jaws, in form.
 409. Defective growth of lower jaw.
 410. Mechanical injuries of the alveoli and dental periosteum.
 a. Hemorrhage.
 b. Fracture.
 411. Mechanical injuries of the teeth.
 a. Fracture.
 b. Dilaceration.
 c. Dislocation.
 d. Friction.

DISEASES OF THE TONGUE.

412. Glossitis.
 413. Ulcer.
 414. Aphthous ulcer.
 415. Abscess.
 416. Hypertrophy.
 (43A.) *Primary syphilis.*
 (43B.) *Secondary syphilis.*
 (44.) *Cancer.*
 417. Vascular tumor.
 418. Tongue-tie.
 (89.) *Paralysis.*

DISEASES OF THE FAUCES AND PALATE.

419. Sore throat.
 420. Relaxed throat.
 421. Ulcerated throat.
 422. Quinsy. *Syn.*, Cynanche tonsillar.
 423. Tonsillitis.
 424. Sloughing sore throat. *Syn.*, Putrid sore throat, Cynanche maligna.
 (19.) *Diphtheria.*
 425. Enlarged tonsils.
 (44.) *Cancer of the tonsils.*
 (49.) *Scrofulous disease of the tonsils.*
 426. Elongated uvula.
 427. Perforation of the palate.
 428. Stricture of the fauces.

- (43.) *Syphilitic affection of the fauces and tonsils.*

- (44.) *Cancer.*

429. Non-malignant tumor.
Return such tumors here according to the list at p. 339.
 a. Fibro-cellular tumor.
 b. Fibro-cystic tumor.

430. Malformations.
Return such cases here according to the list at p. 252.
 a. Cleft palate.

DISEASES OF THE PHARYNX.

431. Pharyngitis.
 432. Ulcer.
 a. Superficial ulcer.
 b. Perforating ulcer.
 433. Abscess.
 434. Sloughing.
 435. Adhesion of the soft palate.
 436. Dilatation.
 (43.) *Syphilitic affection.*
 (44.) *Cancer.*
 (1047.) *Injury by corrosive substances.*
 (1045.) *Foreign bodies.*
 (89.) *Paralysis.*

DISEASES OF THE SALIVARY GLANDS.

437. Inflammation.
 438. Salivation. *Syn.*, Ptyalism.
 439. Abscess.
 440. Salivary fistula.
 (21.) *Mumps.*
 (44.) *Cancer.*
 441. Non-malignant tumors.
Return such tumors here according to the list at p. 339.
 442. Salivary calculus.

DISEASES OF THE ESOPHAGUS.

443. Esophagitis.
 444. Ulceration.
 445. Perforation.
 446. Stricture.
 (44.) *Cancer.*
 (1046.) *Foreign bodies.*
 447. Malformations.
Return such cases here according to the list at p. 252.
 (1047.) *Injury by corrosive substances.*
 (89.) *Paralysis.*
 448. Dysphagia.

DISEASES OF THE STOMACH.

449. Gastritis.
 (976, &c.) *a.* From irritant poisons.
(For the list of poisons, see p. 351.)
 450. Chronic ulcer.
 451. Hematemesis.
 452. Perforation.
 453. Dilatation.
 454. Stricture.
 455. Gastric fistula.
 456. Hernia.
 (44.) *Cancer.*
 (45.) *Colloid.*
 457. Non-malignant tumors.
Return such tumors here according to the list at p. 339.
 458. Parasitic disease.
Return cases of this class according to the list at p. 150.
Nos. 46, 47.
 (1066-1071.) *Injuries.*
 (1074.) *Foreign bodies.*
 459. Spontaneous laceration.
 460. Dyspepsia.
 461. Gastrodynia.
 462. Pyrosis.

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463. Vomiting.

DISEASES OF THE INTESTINES.

464. Enteritis.
 465. Typhlitis.
 466. Dysentery.
 467. Ulceration.
 468. Perforation.
 469. Abscess in the sub-peritoneal tissue.
 470. Fæcal abscess.
 471. Fistula.
 a. Fæcal fistula. *Syn.*, Artificial anus.
 (561.) *Vesico-intestinal fistula.*
 472. Hemorrhage.
 473. Melæna.
 474. Dilatation.
 475. Tympanites.
 476. Obstruction.
 477. Stricture.
 478. Intussusception.
 479. Internal strangulation.
 a. Mesenteric.
 b. Mesocolic.
 480. Hernia.
 a. Reducible.
 b. Irreducible.
 c. Obstructed.
 d. Inflamed.
 e. Strangulated.
 1. Diaphragmatic.
 2. Epigastric.
 3. Ventral.
 4. Umbilical.
 5. Lumbar.
 6. Inguinal.
 a. Oblique.
 b. Direct.
 c. Incomplete.
 d. Scrotal.
 e. Congenital.
 f. Infantile.
 7. Femoral.
 8. Obturator.
 9. Perineal.
 10. Pudendal.
 11. Vaginal.
 12. Ischiatic.
 481. Diseases of hernial sacs.
 a. Inflammation.
 b. Fibrinous effusion with closure.
 c. Suppuration.
 d. Dropsy.
 e. Movable bodies.
 f. Laceration.
 (44.) *Cancer.*
 (45.) *Colloid.*
 482. Non-malignant tumors.
 Return such tumors here according to the list at p. 339.
 a. Polypus.
 483. Parasitic disease.
 Return cases of this class according to the list at p. 149, Nos. 1-3, 8-13, 15-20, 24, 25, 27, 32, 34, 35.
 (1075.) *Concretions.*
 483. Malformations.
 With the exception of hernia, which will appear under 480, return such cases here according to the list at p. 252.
 (1075.) *Foreign bodies.*
 (1066-1071.) *Injuries.*
 484. Diarrhœa.
 (17.) *Simple cholera.*
 (18.) *Malignant cholera.*
 a. *Choleraic diarrhœa.*
 485. Paralysis.
 486. Colic.
 (908*a.*) *Lead colic.*
 487. Constipation.

DISEASES OF THE RECTUM AND ANUS.

488. Ulceration.
 489. Abscess.
 490. Fistula in ano.
 (562.) *Recto-vesical fistula.*
 (600.) *Recto-urethral fistula.*
 (676.) *Recto-vaginal fistula.*
 491. Hemorrhoids.
 a. Internal.
 b. External.
 492. Hemorrhage from the rectum.
 493. Fissure of the anus.
 494. Prolapsus.
 495. Stricture.
 (43.) *Syphilis of the rectum.*
 496. Condyloma of the anus.
 (44.) *Cancer of the rectum.*
 (44.) *Cancer of the anus.*
 497. Non-malignant tumors of the rectum.
 Return cases of this class here according to the list at p. 339.
 497. Parasitic disease.
 Return cases of this class here according to the list at p. 149, see No. 10.
 497. Malformations.
 Return such cases here according to the list at p. 252.
 (1081-1082.) *Injuries.*
 (1089.) *Foreign bodies in the rectum.*
 498. Neuralgia.
 499. Spasm of the sphincter ani.
 500. Pruritus ani.

DISEASES OF THE LIVER.

501. Hepatitis.
 502. Abscess.
 (31.) *Pyæmic inflammation and abscess.*
 503. Acute atrophy.
 504. Simple enlargement. *Syn.*, Congestion of the liver.
 505. Thickening of the capsule.
 506. Cirrhosis.
 507. Fatty liver.
 508. Fibroid deposit.
 509. Lardaceous liver. *Syn.*, Amyloid disease of the liver.
 Waxy liver.
 (43.) *Syphilitic deposit.*
 (44.) *Cancer.*
 (45.) *Colloid.*
 510. Non-malignant tumors.
 Return such tumors here according to the list at p. 339.
 511. Cyst.
 (49.) *Tubercle.*
 512. Parasitic disease.
 Return cases of this class according to the list at p. 149, Nos. 14, 21-23, 25, 28-34, 35.
 512. Malformations.
 Return such cases here according to the list at p. 252.
 (1066-1071.) *Injuries.*
 513. Jaundice. *Syn.*, cterus.
 514. Obstruction of the vena porta.

DISEASES OF THE HEPATIC DUCTS AND GALL BLADDER.

515. Inflammation.
 516. Ulcer.
 517. Perforation.
 a. Biliary fistula.

518. Obstruction.

- (44.) *Cancer.*
 519. Parasitic disease.
 Return cases of this class according to the list at p. 149, No. 25.
 520. Gallstones.
 a. Passage of gallstones through the duct.
 520.* Malformations.
 Return such cases here according to the list at p. 252.
 (1066-1071.) *Injuries.*

DISEASES OF THE PANCREAS.

521. Abscess.
 522. Obstruction of the duct.
 (44.) *Cancer.*
 (45.) *Colloid.*
 523. Calculi.

DISEASES OF THE SPLEEN.

524. Splenitis.
 525. Abscess.
 (31.) *Pyæmic inflammation and abscess.*
 526. Congestion. *Syn.*, Ague cake.
 527. Fibrinous deposit.
 528. Hypertrophy.
 a. Leucocythæmia.
 529. Lardaceous spleen. *Syn.*, Amyloid disease, Waxy spleen.
 (44.) *Cancer.*
 (45.) *Colloid.*
 (49.) *Tubercle.*
 530. Parasitic diseases.
 Return cases of this class according to the list at p. 149, No. 22.
 (1066.) *Rupture.*

DISEASES OF THE PERITONEUM.

531. Peritonitis.
 (719.) *a. Metro-peritonitis. Syn., Puerperal peritonitis.*
 b. Chronic peritonitis.
 c. Suppurative peritonitis.
 (49.) *d. Tubercular peritonitis.*
 e. Adhesions of the peritoneum.
 532. Ascites.
 532.* Non-malignant tumors.
 Return such tumors here according to the list at p. 339.
 (44.) *Cancer.*
 (45.) *Colloid.*
 533. Parasitic disease.
 Return cases of this class according to the list at p. 149, Nos. 4, 14, 22.
 (1067-1070.) *Injuries.*

DISEASES OF THE MESENTERIC GLANDS.

534. Inflammation.
 535. Abscess.
 536. Enlargement.
 (44.) *Cancer.*
 537. Non-malignant tumors.
 Return such tumors here according to the list at p. 339.
 (49.) *Tubercle.*
 (49.) *Tabes mesenterica.*

DISEASES OF THE URINARY SYSTEM.¹

- DISEASES OF THE KIDNEY.**
538. Bright's disease. *Syn.*, Albuminuria.
1. Acute Bright's disease. *Syn.*, Acute albuminuria, Acute desquamative nephritis, Acute renal dropsy.
 2. Chronic Bright's disease. *Syn.*, Chronic albuminuria.
- Subdivisions:*
- a. Granular kidney. *Syn.*, Contracted granular kidney, Chronic desquamative nephritis, Gouty kidney.
 - b. Fatty kidney.
 - c. Lardaceous kidney. *Syn.*, Amyloid disease, Waxy kidney.
539. Suppurative nephritis.
540. Abscess.
541. Pyelitis.
542. Fibrinous deposit.
543. Hydronephrosis.
544. Hypertrophy.
545. Atrophy.
- (44.) *Cancer.*
546. Non-malignant tumors.
- Return such tumors here according to the list at p. 339.*
547. Simple cyst.
548. Urinary cyst (from injury).
- (49.) *Tubercle.*
549. Parasitic disease.
- Return cases of this class according to the list at p. 148.*
550. Calculus.
551. Calculus in the ureter.
552. Malformations.
- Return cases of this class according to the list at p. 252.*
- (1066-1071.) *Injuries.*
553. Hematuria renalis.
554. Suppression of urine. *Syn.*, Ischuria renalis.
- (52.) *Diabetes.* *Syn.*, *Diabetes mellitus.*
555. Diuresis.
556. Movable kidney.
- DISEASES OF THE BLADDER.**
557. Cystitis. *Syn.*, Catarrh of the bladder.
- a. Acute.
 - b. Chronic.
558. Ulceration.
559. Suppuration.
560. Sloughing.
561. Vesico-intestinal fistula.
562. Recto-vesical fistula.
- (660.) *Utero-vesical fistula.*
- (675.) *Vesico-vaginal fistula.*
563. Hypertrophy.
564. Distension.
- a. Sacculated bladder.
 - b. Rupture.
565. Inversion.
566. Extroversion.
567. Hernia.
- (44.) *Cancer.*
568. Fibrous tumor.
569. Vileous tumor.
570. Calculus.
- a. Uric acid.
 - b. Urate of ammonia.
 - c. Uric oxide. *Syn.*, Xanthic oxide.
 - d. Oxalate of lime.
 - e. Cystic oxide.
 - f. Phosphate of lime.
 - g. Triple phosphate.
 - h. Fusible.
 - i. Carbonate of lime.
 - k. Fibrinous.
 - l. Urostealith.
 - m. Blood calculus.
- Foreign bodies.
571. Hematuria (Vesical).
571. Malformations.
- Return such cases according to the list at pp. 252 to 254.*
- (1083, 1091.) *Injuries.*
572. Paralysis.
573. Irritability.
574. Spasm.
575. Neuralgia.
576. Incontinence of urine.
577. Retention of urine.
- DISEASES OF THE PROSTATE GLAND.**
578. Inflammation.
- a. Acute.
 - b. Chronic.
579. Ulceration.
580. Abscess.
581. Atrophy.
- (44.) *Cancer.*
582. Non-malignant tumor. *Syn.*, Enlarged lobe of the prostate.
582. Chronic enlargement.
583. Cyst.
- (49.) *Tubercle.*
584. Calculi.
- GONORRHEA AND ITS COMPLICATIONS.**
585. Gonorrhœa.
- a. In the male.
 - b. In the female.
586. Balanitis.
- (341.) *Herpes preputialis.*
587. Phimosis.
588. Paraphimosis.
589. Bubo.
590. Lacunar abscess.
- (580.) *Prostatic abscess.*
591. Epididymitis. *Syn.*, Gonorrheal orchitis.
- a. Abscess.
592. Abscess of the spermatic cord.
593. Condyloma.
- a. In the male.
 - b. In the female.
594. Gleet.
- (631.) *Inflammation of ovary.*
595. Abscess of the vulva.
- (117.) *Gonorrhœal ophthalmia.*
- (140.) *Gonorrhœal iritis.*
- (35.) *Gonorrhœal rheumatism.*
- DISEASES OF THE MALE URETHRA.**
595. Urethritis.
596. Stricture.
- a. Organic.
 - b. Traumatic.
 - c. Spasmodic.
 - d. Inflammatory.
597. Ulcer.
598. Urinary abscess.
599. Urinary fistula.
600. Recto-urethral fistula.
601. Extravasation of urine.
601. Impacted calculus.
- a. Foreign bodies.
601. Malformations.
- Return such cases according to the list at p. 252.*
- (1078-1091.) *Injuries.*

DISEASES OF THE GENERATIVE SYSTEM.¹

DISEASES OF THE MALE ORGANS OF GENERATION.

- DISEASES OF THE PENIS.**
602. Inflammation.
603. Abscess.
- (505a.) *Gonorrhœa.*
- (593a.) *Condyloma.*
604. Gangrene.
605. Priapism.
- (43.) *Syphilis.*
- (44.) *Cancer.*
- a. Of the prepuce.
 - b. Of the body.
606. Non-malignant tumors.
- Return such tumors here according to the list at p. 339.*
- (1078.) *Injuries.*
607. Malformations.
- Return such cases according to the list at p. 252.*
- a. Phimosis—congenital.
- DISEASES OF THE SCROTUM.**
608. Sloughing.
609. Edema.
610. Elephantiasis.
- (834.) *Prurigo.*
- (43.) *Syphilis.*
- (44.) *Cancer.*
- (44c.) *Epithelial cancer.* *Syn.*, Chimney-sweeper's cancer.
611. Non-malignant tumors.
- Return such tumors here according to the list at p. 339.*
611. Malformations.
- Return such cases here according to the list at p. 252.*
- DISEASES OF THE CORD.**
612. Hydrocele.
- Varieties:**
- a. Encysted.
 - b. Diffused.
613. Varicocele.
614. Non-malignant tumors.
- Return such tumors here according to the list at p. 339.*
615. Neuralgia.
- DISEASES OF THE TUNICA VAGINALIS.**
616. Inflammation.
617. Hydrocele.
- Varieties:**
- a. Congenital.
 - b. Infantile.
 - c. Encysted.
618. Hematocele.
619. Loose bodies.

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DISEASES OF THE TESTICLE.

620. Orchitis.
a. Acute.
b. Chronic.
 620. Epididymitis.
 621. Abscess.
 622. Protrusion of tubuli. *Syn.*
Hernia testis, Fungus testis.
623. Atrophy.
 (43.) *Syphilitic disease.*
 (44.) *Cancer.*
 624. Non-malignant tumors.
Return such tumors here according to the list at p. 339.
 625. Cystic disease.
 (49.) *Tubercle.*
 (1078.) *Injuries.*

626. Malformations.
Return such cases according to the list at pp. 252 to 254.
a. Fœtal remains in the testicle.
b. Malposition.
 627. Spermatorrhœa.
 628. Impotence.
 629. Neuralgia.

DISEASES OF THE FEMALE ORGANS OF GENERATION IN THE UNIMPREGNATED STATE.

DISEASES OF THE OVARY.

630. Inflammation.
 631. Abscess.
 632. Hæmorrhage.
 633. Atrophy.
 634. Hypertrophy.
 (44.) *Cancer.*
 635. Fibrous tumor.
 636. Encysted dropsy.
 637. Complex cystic tumor. *Syn.*
Alveolar, gelatinous, and colloid tumor. Cystosarcoma.
a. With intracystic growths.
 638. Cyst, containing tegumentary structures.
a. Cutaneous or piliferous cyst. Syn., Dermoid cyst.
b. Dentigerous cyst.
 (49.) *Tubercle.*
 639. Parasitic disease.
Return cases of this class according to the list at p. 148.
 640. Dislocation.
a. Transplantation.
 641. Hernia.
 642. Malformations.
Return such cases according to the list at p. 252.

DISEASES OF THE FALLOPIAN TUBE.

643. Abscess.
 644. Dropsy.
 645. Stricture.
 646. Occlusion.
 (44.) *Cancer.*
 647. Cyst.
 (49.) *Tubercle.*
 648. Dislocation.
 649. Hernia.

DISEASES OF THE BROAD LIGAMENT.

650. Inflammation.
a. Pelvic peritonitis.
b. Pelvic cellulitis.
 651. Abscess.
 652. Cyst.
 653. Peritertiary or pelvic hæmatocele.

DISEASES OF THE UTERUS, INCLUDING THE CERVIX.

654. Catarrh. *Syn., Leucorrhœa.*
a. Hydrorrhœa.
 655. Inflammation.
 656. Granular inflammation.
 657. Abrasion.
 658. Ulcer.
 658. Rodent ulcer.
 659. Abscess.
 660. Utero-vesical fistula.
 661. Stricture of the orifice.
 662. Stricture of the canal.
 663. Occlusion of the orifice.
 664. Occlusion of the canal.
 665. Hypertrophy.
a. Elongation of the cervix.
 666. Atrophy.
 (44.) *Cancer.*
a. Scirrhus.
b. Medullary cancer.
c. Epithelial cancer.
 667. Non-malignant tumor.
a. Fibrous tumor.
b. Polypus.
 (49.) *Tubercle.*
 668. Displacements and distortions.
a. Anteversion.
b. Retroversion.
c. Antelexion.
d. Retroflexion.
e. Inversion.
f. Prolapsus.
1. Procentia.
g. Hernia.
 669. Malformations.
Return such cases according to the list at pp. 252 to 254.

DISEASES OF THE VAGINA.

670. Catarrh. *Syn., Leucorrhœa.*
 671. Inflammation.
 672. Abscess.
 (585b.) *Gonorrhœa.*
 673. Cicatrix or band.
 674. Vaginal fistula.
 675. Vesico-vaginal fistula.
 676. Recto-vaginal fistula.
 677. Hernia.
a. Cystocele.
b. Rectocele.
 (44.) *Cancer.*
 678. Non-malignant tumors.

a. Polypus.

679. Laceration.
 679. Malformations.
Return such cases here according to the list at pp. 252 to 254.

DISEASES OF THE VULVA.

680. Inflammation of the labia.
 681. Pruritus.
 (843.) *Eczema of the labia.*
 682. Edema of the labia.
 683. Abscess.
 684. Gangrene.
 685. Hypertrophy.
 686. Occlusion.
 687. Imperforate hymen.
 (266.) *Varicose veins.*
 (43.) *Syphilis.*
 (44.) *Cancer.*
 688. Vascular tumor of the meatus urinarius.
 689. Mucous cyst.
 (593b.) *Condyloma.*
 689. Malformations.
Return such cases here according to the list at p. 252.

FUNCTIONAL DISEASES OF THE FEMALE ORGANS OF GENERATION.

690. Amenorrhœa. *Syn., Absent menstruation.*
Varieties:
a. From original defective formation.
b. From want of development at the time of puberty.
c. From mechanical obstruction.
d. From temporary suppression.
 691. Scanty menstruation. *Syn., Deficient menstruation.*
 692. Vicarious menstruation.
 693. Dysmenorrhœa. *Syn., Painful menstruation.*
 694. Menorrhagia. *Syn., Excessive menstruation.*
 694. Hæmorrhage.
 (56.) *Chlorosis. Syn., Green sickness.*

AFFECTIONS CONNECTED WITH PREGNANCY.

DISORDERS OF THE NERVOUS SYSTEM.

- Neuralgia.
Varieties:
a. Odontalgia.
b. Cephalalgia.
c. Mastodynia.
 Choreæ.
 Convulsions.
 Hypochondriasis.
 Mania.

DISEASES OF THE CIRCULATORY SYSTEM.

- Varicose veins—
a. Of the lower extremities.
b. Of the labia.
c. Of the rectum. Hæmorrhoids.
 Serous exudation.
Varieties:
a. Ascites.
b. Edema of the labia.

c. Edema of the lower extremities.

- Syncope.
 Palpitation.

DISORDERS OF THE RESPIRATORY SYSTEM.

- Dyspnoea.
 Orthopnoea.
 Cough.

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DISORDERS OF THE DIGESTIVE SYSTEM.

Salivation.
Depraved and capricious appetite.
Nausea and vomiting.
Cardialgia or Heartburn.
Pyrosis.
Intestinal cramp—colic.
Constipation.
Diarrhœa.
Jaundice.

DISORDERS OF THE URINARY SYSTEM.
Albuminuria.

Dysuria.
Incontinence of urine.
Retention of urine.

DISORDERS OF THE GENERATIVE SYSTEM.

695. Metritis. *Syn.*, Hysteritis.
696. Discharge of watery fluid from the uterus. Hydrorrhœa.
697. Rheumatism of the uterus.
698. Hysteralgia.
699. Spurious pains and cramps.

(670.) *Catarrh of the vagina.* *Syn.*, *Leucorrhœa.*
700. Sanguineous discharge. *Syn.*, Menstruation.
701. Hæmorrhage.
702. Displacements of the uterus.
Varieties:
a. Prolapsus.
b. Hernia.
c. Retroversion.
(681.) *Pruritus of the vulva.*
703. Abortion.
704. Premature labour.
705. Extra-uterine gestation.

AFFECTIONS CONNECTED WITH PARTURITION.

706. Atony of the uterus.
707. Over-distension of the uterus.
a. From excess of liquor amnii.
b. From twins, triplets, &c.
708. Mechanical obstacle to the action of the uterus.
a. From occlusion of the os uteri.
b. From rigidity.
(1.) Of the os uteri.
(2.) Of the vagina.
(3.) Of the perineum.
c. From cancer of the cervix uteri.
d. From narrowness of the vagina.
e. From cicatrix or band in the vagina.
f. From vaginal cyst.
g. From prolapsus of the bladder.
h. From stone in the bladder.
i. From distended rectum.
k. From prolapsus of the rectum.
l. From tumor.

Varieties:
1. Uterine.
2. Ovarian.
3. Pelvic.
4. Of external parts.
m. From polypus.
n. From fractured pelvis.
o. From exostosis.
p. From distorted or contracted pelvis.
q. From dislocated lumbar vertebrae into pelvis. *Syn.*, Spondylo listhesis.
r. From ankylosed coccyx.
s. From diminutive pelvis.
t. From extreme anteversion of the uterus (with pendulous abdomen).
u. From excessive size of the fœtus.
v. From malposition of the fœtus.
w. From malformation of the fœtus.
x. From enlargement of the fœtus from disease.
y. From unusual thickness

of the fetal membranes.
z. From unusual shortness of the funis.
709. Hæmorrhage.
a. From placenta prævia. *Syn.*, Unavoidable hæmorrhage.
b. From accidental detachment of the placenta. *Syn.*, Accidental hæmorrhage.
c. From thrombus of the cervix uteri or labium.
710. Rupture or laceration of the uterus.
711. vagina.
712. urinary bladder.
713. perineum.
714. Retention of the placenta.
a. From atony of the uterus.
b. From irregular or hour-glass contraction.
c. From preternatural adhesions.
715. Inversion of the uterus.
716. Convulsions.

AFFECTIONS CONSEQUENT ON PARTURITION.

717. Post-partum hæmorrhage.
(33.) *Puerperal ephemera.*
718. Milk fever.
(32.) *Puerperal fever.*
719. Metro-peritonitis. *Syn.*, Puerperal peritonitis.
a. Metritis.
(531.) b. *Peritonitis.*
(260.) *Phlebitis.*
(261.) *Phlegmasia dolens.*
(650b.) *Pelvic cellulitis.*
720. Iliac and pelvic abscesses.
721. Sloughing of the cervix uteri.
722. " " vagina.
723. " " perineum.

724. Sloughing of the bladder.
725. " " rectum.
(660.) *Utero-vesical fistula.*
(675.) *Vesico-vaginal fistula.*
(676.) *Recto-vaginal fistula.*
(729.) *Inflammation of the female breast.*
(730.) *Abscess of the female breast.*
726. Puerperal mania.
a. Connected with parturition.
b. Connected with lactation.
727. Puerperal convulsions. *Syn.*, Eclampsia.

728. Sudden death after delivery.
a. From shock or nervous exhaustion.
b. From impaction of coaguli in the heart and pulmonary artery.
1. Thrombosis.
2. Embolism.
c. From entrance of air into veins (from separation of the placenta).
(902.) *Still-born.*
(903.) *Premature birth.*

DISEASES OF THE FEMALE BREAST.¹

729. Inflammation.
a. Acute.
b. Chronic.
730. Abscess.
731. Sinus.
732. Galactorrhœa.
733. Deficiency of milk.
734. Hypertrophy.
735. Atrophy.
736. Depressed nipple.
737. Chapped nipple.
738. Ulcerated nipple.
(44.) *Cancer.*

a. *Cirrhous.*
b. *Medullary cancer.*
c. *Epithelial cancer.*
(45.) *Colloid.*
739. Non-malignant tumors.
a. Fibrous tumor. *Syn.*, Painful subcutaneous tumor.
b. Fibro-plastic tumor.
c. Fatty tumor.
d. Osseous tumor.
e. Cartilaginous tumor. *Syn.*, Enchondroma.

f. Chronic mammary tumor. *Syn.*, Adenoid tumor.
g. Vascular tumor.
746. Cyst.
747. Complex cystic tumor. *Syn.*, Cysto-sarcoma.
748. Parasitic disease.
Return cases of this class according to the list at p. 148.
749. Hyperæsthesia.
750. Mastodynia. *Syn.*, Neuralgia.

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DISEASES OF THE MALE MAMMILLA.¹

751. Inflammation.
752. Hypertrophy.
(44.) *Cancer.*

753. Non-malignant tumors.
*Return such cases according
to the list at p. 339.*

754. Cyst.

DISEASES OF THE ORGANS OF LOCOMOTION.¹

DISEASES OF BONES.

755. Ostitis.
a. Periostitis.
1. Nodes.
756. Diffuse periostitis. *Syn.*,
Acute periosteal abscess.
a. Acute necrosis.
757. Osteo-mylitis.
758. Chronic abscess.
759. Caries.
760. Necrosis.
761. Mollities ossium.
762. Hypertrophy.
763. Atrophy.
764. Spontaneous fracture (The
cause, if known, should be
stated).
(43.) *Syphilitic disease.*
(44.) *Cancer.*
765. Non-malignant tumors.
a. Fibrous and fibro-cystic.
b. Myeloid.
c. Cartilaginous. *Syn.*, En-
chondroma.
d. Osseous tumor. *Syn.*,
Exostosis.
Varieties:
1. Ivory.
2. Cancellated.
3. Diffused.
766. Cyst.
(50.) *Rickets.*
(49.) *Scrofulous disease.*
767. Parasitic disease.
*Return cases of this class
according to the list at
p. 148.*
767. Malformations.
*Return such cases here ac-
cording to the list at pp.
252 to 254.*

DISEASES OF JOINTS.

768. Acute synovitis.
769. Chronic synovitis.
a. Pulpary degeneration of
synovial membrane.
(49.) b. *Scrofulous disease of the
joints.*
(49.) 1. *Morbus coxæ.*
770. Ulceration of cartilage.
771. Abscess.
(31a.) *Pyæmic abscess.*
772. Ankylosis.
a. Deformity from anky-
losis.
773. Dropsy of joints.
(35.) *Gonorrhæal rheumatism.*
(36.) *Synovial rheumatism.*
(41.) *Gouty synovitis.*

(42.) *Chronic osteo-arthritis. Syn.*,
Chronic rheumatic arthritis.
774. Degeneration of cartilage,
and of the articular sur-
faces of bones.
775. Perforation of joints.
776. Loose cartilage. *Syn.*, Loose
body.
777. Relaxation of ligament.
778. Displacement of articular
cartilage.
779. Knock-knee.
780. Bow-leg, or out-knee.
(44.) *Cancer.*
781. Non-malignant tumors.
*Return such cases here ac-
cording to the list at p.
339.*
782. Neuralgia of joints.

DISEASES OF THE SPINE.

783. Ulceration of ligaments and
cartilages.
784. Caries and necrosis.
a. Spontaneous fracture of
the odontoid process.
785. Psoas, lumbar, and other
abscesses.
786. Angular deformity. *Syn.*,
Kyphosis.
787. Lateral curvature. *Syn.*,
Skoliosis.
788. Anterior curvature. *Syn.*,
Lordosis.
(50.) *Rickety curvature.*
789. Ankylosis.
(42.) *Chronic osteo-arthritis.*
790. Non-malignant tumors.
*Return such cases here ac-
cording to the list at p.
339.*
(44.) *Cancer.*
791. Parasitic disease.
*Return cases of this class
according to the list at
p. 148.*
792. Malformations.
*Return such cases here ac-
cording to the list at p.
252.*
a. Deformity from malfor-
mation.
(80a.) b. *Spina bifida.*

DISEASES OF THE MUSCULAR
SYSTEM.

DISEASES OF THE MUSCLES.

793. Inflammation.

794. Abscess.
795. Gangrene.
796. Atrophy.
797. Progressive muscular atro-
phy.
798. Fatty degeneration.
799. Ossification.
(43.) *Syphilitic deposit.*
(44.) *Cancer.*
(45.) *Colloid.*
800. Non-malignant tumor.
a. Erectile tumor.
801. Cyst.
(114.) *Rupture.*
(85.) *Infantile paralysis.*
802. Parasitic disease.
*Return such cases here ac-
cording to the list at p.
149, No. 4.*
a. Trichinosis.
(95.) *Spasm.*
803. Exhaustion.
(89b.) *Sirivener's palsy.*
(19a.) *Diphtheritic paralysis.*

DISEASES OF TENDONS

804. Inflammation.
(865a) *Thecal abscess.*
805. Adhesion of tendons.
(44.) *Cancer.*
806. Non-malignant tumors.
807. Contraction of tendons, fas-
cia, or muscles.
808. Club-foot.
a. Talipes varus.
b. " valgus.
c. " equinus.
d. " calcaneus.
e. " calcaneo-varus.
f. " equino-vulgus.
Syn., Flat-foot.
809. Club-hand.
810. Contracted palmar fascia.
811. Wry-neck.
(1145.) *Rupture.*

DISEASES OF THE APPENDAGES OF
THE MUSCULAR SYSTEM.

812. Enlarged bursa patellæ. *Syn.*,
Housemaid's knee.
813. Enlargement of other bursæ
(specify which).
814. Bursal tumor.
815. Bursal abscess.
816. Bunion.
817. Ganglion.
a. Diffused palmar gan-
gion.

DISEASES OF THE CELLULAR TISSUE.¹

818. Inflammation.
819. Abscess.
820. Inflammatory induration in
the newly born.
821. Slough.
(30b.) *Phlegmonous erysipelas.*
(862.) *Carbuncle. Syn., Anthrax.*

822. Obesity.
823. Hæmorrhage.
(653.) a. *Pelvic hæmatocele.*
824. Non-malignant tumors.
*Return such cases according
to the list at p. 339.*
(44.) *Cancer.*

825. Parasitic disease.
*Return cases of this class
according to the list at p.
148.*
(1146.) *Foreign substances.*
826. Emphysema.

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DISEASES OF THE CUTANEOUS SYSTEM.¹

- (30.) *Erysipelas*.
 827. Erythema. (This term includes
 1. Erythema leve.
 2. Erythema fugax. *Syn.*,
 E. volaticum.
 3. Erythema marginatum.
 4. " papulatum.
 5. " tuberculatum.
 6. " nodosum.)
 828. Intertrigo.
 829. Roseola. (This term includes
 1. Roseola astiva.
 2. " autumnalis.
 3. " symptomatica.
 4. " annulata.)
 830. Urticaria. *Syn.*, Nettle-rash.
 a. Urticaria acuta.
 b. " chronica.
 (Under one or other of these
 heads are included
 1. Urticaria febrilis.
 2. " evanida.
 3. " perstans.
 4. " conferta.
 5. " subcutanea.
 6. " tuberculata.)
 831. Pellagra.
 832. Acrodynia.
 833. Asturian rose.
 834. Prurigo.
 835. Lichen. (This term includes
 1. Lichen simplex.
 2. " pilaris.
 3. " circumscriptus.
 4. " agrius.
 5. " tropicus. *Syn.*,
 'Prickly heat'.
 (The so-called Lichen lividus
 is really a form of Purpura.)
 836. Strophulus. *Syn.*, Red gum.
 Tooth rash. (This term
 includes
 1. Strophulus intertrinctus.
 2. " confertus.
 3. " candidus.)
 (Strophulus albidus is referred
 to Acne.
 " volaticus to Ery-
 thema.)
 837. Pityriasis. (This term in-
 cludes Pityriasis capitis.
 Syn., Dandruff.)
 (Pityriasis versicolor is re-
 ferred to Parasitic affec-
 tions as a *Synonym* of Tinea
 versicolor.)
 838. Psoriasis. (This term in-
 cludes Lepra.)
 a. Psoriasis vulgaris. *Syn.*,
 Lepra vulgaris.
 b. Psoriasis guttata.
 c. " diffusa.
 d. " gyrata.
 e. " inveterata.
 840. Miliaria.
 a. Sudamina.
 841. Herpes.
 a. Herpes phlyctenodes.
 b. " circinatus.
 c. " iris.
 d. " zoster. *Syn.*,
 Shingles.
 842. Pemphigus. *Syn.*, Pompho-
 lyx.
 a. Pemphigus acutus.
 b. " chronicus.
 c. " solitarius.
 843. Eczema.
 a. Eczema simplex.
 b. " rubrum.
 c. " impetiginodes.
 d. " chronicum.
 844. Impetigo.
 a. Impetigo sparsa.
 b. " confluens.
 1. Figurata.
 2. Larvalis. *Syn.*, Por-
 rigo larvalis.
 845. Rupia.
 a. Rupia simplex.
 b. Rupia prominens.
 c. Rupia escharotica. *Syn.*,
 Pemphigus gangre-
 nosus.
 846. Ecthyma.
 847. Acne.
 a. Acne punctata.
 b. " indurata.
 c. " rosacea.
 d. " strophulosa.
 Syn., Strophulus
 albidus.
 848. Sycosis. *Syn.*, Mentagra.
 849. Stearrhoea.
 a. Stearrhoea simplex.
 b. " nigricans.
 850. Ichthyosis.
 a. Ichthyosis vera.
 " cornea.
 851. Xeroderma. *Syn.*, Sclero-
 derma, Scleriosis.
 852. Leucoderma. (This term in-
 cludes Vitiligo.)
 853. Albinismus.
 854. Canities.
 855. Melasma.
 (286.) *Melasma Addisoni*. English
 name, *Addison's disease*.
 Syn., *Bronzed skin*.
 856. Lentigo and Ephelis. *Syn.*,
 Freckles.
 857. Chilblain.
 858. Frostbite.
 859. Ulcer.
 860. Fissures.
 (353.) *Cancrum oris*.
 861. Boil.
 862. Carbuncle. *Syn.*, Anthrax.
 (26.) *Malignant pustule*.
 863. Onychia.
 864. Onychia maligna.
 865. Whitlow.
 a. Thecal abscess.
 866. Gangrene.
 866*a.* Senile gangrene.
 866*b.* Boil-sore.
 867. Hypertrophy.
 868. Corn.
 (816.) *Bunion*.
 869. Elephantiasis Arabum. *Syn.*,
 Barbadoes' leg, Elephas.
 (48.) *True leprosy*. *Syn.*, *Elephan-*
 tiasis Græcorum.
 870. Atrophy.
 a. Linear atrophy.
 b. Alopecia.
 c. Atrophy of nails.
 (44.) *Cancer*.
 871. Fibro-cellular tumor.
 872. Fatty tumor.
 (267.) *Nævus vascularis*.
 873. Nævus. *Syn.*, Port-wine
 stain.
 874. Nævus pilaris. *Syn.*, Mole.
 875. Sebaceous tumor.
 a. Steatoma.
 876. Cornua.
 877. Warts.
 878. Condyloma.
 879. Molluscum.
 880. Cheloid.
 881. Framboesia. *Syn.*, Yaws.
 882. Delhi boil.
 883. Aleppo evil.
 (46.) *Lupus*.
 (49.) *Scrofulous disease*.
 884. Ingrown nail.
 (912*a.*) *Silver stain*.
 (992.) *Burns and scalds*.
 884. Cicatrices (state the cause).
 (102.) *Hyperæsthesia*.
 885. Pruritus.
 (103.) *Anæsthesia*.
 886. Ephidrosis.
 887. Audrosis.

PARASITIC DISEASES OF THE SKIN.

888. Tinea tonsurans. *Syn.*, Ring-
 worm. *Parasite*, Achorion
 Lebertii. *Syn.*, Trichophy-
 ton tonsurans.
 889. Tinea decalvans. *Syn.*, Alo-
 pecia areata. Porrigio de-
 calvans. *Par.*, Microspori-
 on Audouini.
 890. Tinea favosa. *Syn.*, Favus.
 Porrigio favosa. *Par.*, Achorion
 Schœnleinii; Puccinia
 Favi.
 891. Tinea versicolor. *Syn.*, Pity-
 riasis versicolor. *Par.*, Micro-
 sporon furfur.
 892. Tinea Polonica. *Syn.*, Plica
 Polonica. *Par.*, Trichophy-
 ton sporuloides.
 893. Mycetoma. *Syn.*, Madura
 foot. *Par.*, Chionyphe
 Carteri.
 894. Scabies. *Syn.*, Itch. *Par.*,
 Sarcoptes scabiei.
 895. Phthiriasis.
 896. Irritation caused by
 a. Pediculus capitis.
 b. " palpebrarum.
 c. " vestimentum.
 d. " tabescentium.
 e. Phthirus inguinalis
 897. Irritation caused by Pulex
 penetrans. *English Syn.*,
 Chigoe.
 Pulex irritans.
 898. Cimex.
 899. " Leptothrix autumnalis.
 English Syn., Harvest-
 bug.
 900. " Wasps, bees, and other
 stinging insects.
 (985*a.*)
 901. " Nettles and other sting-
 ing plants.

CONDITIONS NOT NECESSARILY ASSOCIATED WITH GENERAL OR LOCAL DISEASES.

902. Still-born.

| 903. Premature birth.

| 904. Old age.

| 905. Debility.

¹ Register the diseases printed here in *Italics*, not under this heading, but at the place referred to in each instance by number within brackets.

POISONS.¹

METALS AND THEIR SALTS.

906. Arsenic.
 907. Mercury.
 a. Mercurial tremor.
 (393.) *b. Mercurial inflammation*
 of the dental periosteum.
 908. Lead.
 a. Lead colic. Syn. Painter's colic.
 b. Lead palsy.
 c. Blue gum.
 (124b.) *d. Stain of the conjunctiva*
 from lead.
 909. Copper.
 910. Antimony.
 911. Zinc.
 912. Silver.
 a. Silver stain.
 (124a.) *b. Stain of the conjunctiva*
 from nitrate of silver.
 913. Iron.
 914. Bismuth.
 915. Chromium.
 a. Bichromate of potash.

CAUSTIC ALKALIES.

916. Potash.
 917. Soda.
 918. Ammonia.
 919. Alkaline salts.

METALLOIDS.

920. Phosphorus.
 (394.) *a. Phosphoric inflammation*
 and necrosis of the alveoli.
 921. Iodine.

ACIDS.

922. Sulphuric acid.
 923. Nitric acid.
 924. Hydrochloric acid.
 925. Phosphoric acid.
 926. Oxalic acid.
 927. Tartaric acid.

VEGETABLE POISONS.

928. Savin. (*Juniperus sabina.*—*Linnaeus.*)
 929. Croton Oil. (*Croton tiglium.*—*Linnaeus.*)
 930. Elaterium. (*Ecbalium officinarum.*—*Rich.*)
 931. Colchicum. (*Colchicum autumnale.*—*Linnaeus.*)
 932. Black hellebore. (*Helleborus niger.*—*Linnaeus.*)
 933. White hellebore. (*Veratrum album.*—*Linnaeus.*)
 a. Veratria.
 934. Squill. (*Scilla maritima.*—*Linnaeus.*)
 935. Ergot of rye. (*Sphæria purpurea.*—*Fries*)

- a. Ergotism.*
 936. Opium. (*Papaver somniferum.*—*Linnaeus.*)
 937. Indian hemp. *Cannabis Indica.* (*Cannabis sativa.*—*Linnaeus.*)
 938. Alcohol.
 a. Delirium tremens.
 939. Ether vapour.
 940. Chloroform vapour.
 941. Henbane. *Hyoscyamus.* (*Hyoscyamus niger.*—*Linnaeus.*)
 942. Deadly nightshade. *Belladonna.* (*Atropa belladonna.*—*Linnaeus.*)
 a. Atropa.
 943. Thorn apple. *Stramonium.* (*Datura Stramonium.*—*Linnaeus.*)
 944. Prussic acid.
 a. Oil of bitter almonds.
 b. Laurel water.
 945. Cyanide of potassium.
 946. Nitro-benzole.
 947. Wourai. *Curara.* *Woorara.* (*Strychnos toxifera.*—*Schomburgk.*)
 948. Hemlock. *Conium.* (*Conium maculatum.*—*Linnaeus.*)
 949. Monkshood. *Aconite.* (*Aconitum napellus.*—*Linnaeus.*)
 a. Aconitia.
 950. Foxglove. *Digitalis.* (*Digitalis purpurea.*—*Linnaeus.*)
 a. Digitaline.
 951. Tobacco. (*Nicotiana tabacum.*—*Linnaeus.*)
 a. Nicotia.
 952. Hemlock Dropwort. (*Enanthe crocata.*—*Linnaeus.*)
 953. Nux vomica. (*Strychnos nuxvomica.*—*Linnaeus.*)
 a. Strychnia.
 b. Brucia.
 954. Upas tieute. (*Strychnos tieute.*—*Leschenault.*)
 955. Upas antiar. (*Antiaris toxicaria.*—*Leschenault.*)
 956. Calabar bean. (*Physostigma venenosum.*—*Balfour.*)
 957. Fool's parsley. (*Ethusa cynapium.*—*Linnaeus.*)
 958. Water hemlock. (*Cicuta virosa.*—*Linnaeus.*)
 959. Camphor. (*Cinnamomum camphora.*—*F. Nees and Obermaier.*)
 960. Cocculus Indicus. (*Anamirta cocculus.*—*Wight and Arnott.*)
 961. Darnel. (*Lolium tenulentum.*—*Linnaeus.*)
 962. Indian tobacco. *Lobelia.* (*Lobelia inflata.*—*Linnaeus.*)
 963. Laburnum. (*Laburnum vulgare.*—*Griesback.*)
 964. Yew. (*Taxus baccata.*—*Linnaeus.*)
 965. Poisonous fungi.
 a. Mouldy bread.
 966. Poisonous grain.
 a. Lathyrus. (*Lathyrus sativus.*)
 Paralysis from Lathyrus.
 ANIMAL POISONS.
 967. Spanish fly. *Cantharides.*
 968. Decayed and diseased meat.
 969. Poisonous meat.
 a. Sausages.
 970. Poisonous cheese.
 971. Poisonous milk.
 972. Poisonous fish.
 a. Mussels.
 GASEOUS POISONS.
 973. Ammonia.
 974. Nitrous acid vapour.
 975. Chlorine.
 976. Carbonic acid.
 977. Carbonic oxide.
 978. Coal gas.
 979. Cyanogen.
 980. Sulphuretted hydrogen.
 (393.) *Ether vapour.*
 (940.) *Chloroform vapour.*
 981. Putrid and morbid exhalations.
 982. Other noxious effluvia.
 MECHANICAL IRRITANTS.
 983. Pounded glass.
 984. Steel filings.
 POISONED WOUNDS.
Varieties:
 985. *a. By venomous animals.*
 1. Snakes.
 2. Scorpions.
 985. 3. Stinging insects.
 (900.) *Causes of death from stinging insects should be entered here, and those of irritation only from that cause at No. 900.*
 b. By animals having infectious disease.
 (23.) *Glanders.*
 (24.) *Farcy.*
 (26.) *Equinia mitis.*
 (26.) *Malignant pustule.*
 (31.) *Hydrophobia, Rabies.*
 (92.) *Cowpox.*
 986. *c. By dead animal matter.*
 987. *d. By morbid secretions.*
 988. *e. By vegetable substances.*
 989. 1. Poisoned arrows.
 (947.) *Wourai.*
 990. 2. Subcutaneous injection.
 991. *f. By mineral substances.*

INJURIES.¹

GENERAL INJURIES.

992. Burns and scalds.
 993. Lightning stroke.
 994. Multiple injury. (The cause and extent to be stated)
 995. Asphyxia. *Syn.* Apnoea.
 a. From Drowning.

- b. From Hanging.*
c. " Strangling.
d. " Plugging of air
 passages: e. g.,
 With bread; with
 blood.
e. " Overlying.

- f. From Crushing.*
g. " Gaseous poisons. (See
 list above.)
 996. Privation. *Syn.* Starvation.
 997. Exposure to cold.
 998. Infant exposure.
 999. Neglect.

¹ Register the diseases printed here in *Italics*, not under this heading, but at the place referred to in each instance by number within brackets.

LOCAL INJURIES.¹

INJURIES OF THE HEAD AND FACE.

A.—OF THE HEAD.

1000. Contusion.
a. Cephalæmatoma.
 1001. Scalp-wound: bone not exposed.
 1002. Scalp-wound: bone exposed.
 1003. Concussion of the brain.
 1004. Fracture of the vault of the skull.
a. Simple, without depression.
b. " with depression.
c. Compound, without depression.
d. " with depression.
 1005. Hernia cerebri.
 1006. Fracture of the base of the skull.
 1007. Wound of the skull.
 1008. Laceration of the brain, without fracture.
 1009. Injuries of vessels. (Specify which.)
 1010. Injuries of the cerebral nerves.

B.—OF THE FACE.

1011. Contusion.
 1012. Wound.
 1013. Injuries of vessels. (Specify which.)
 1014. Foreign bodies in the ear.
 1015. " " nose.
 1016. " " antrum.
 1017. " " soft parts.
 1018. Fracture of the facial bones
 1019. " " lower jaw.
 (410, 411.)
 1020. Dislocation of the jaw.

INJURIES OF THE EYE.

1021. Contusion.
 1022. Contusion, with rupture of the sclerotic. *Syn.* Ruptured globe.
 1023. Contusion, with dislocation of the lens.
 1024. Contusion, with hæmorrhage into the globe.
 1025. Foreign bodies in the cornea or conjunctiva.
 1026. Foreign bodies in the cavity of the eye.
 1027. Wound of the eyelid.
 1028. " conjunctiva.
 1029. " sclerotic.
 1030. " cornea.
 1031. " lens.
 1032. " iris.
 1033. Dislocation of the globe.
 (158.) *Total disorganisation of the eye from injury.*
 1034. Wounds and injuries of the parts within the orbit.
 1035. Chemical injuries of the eyelids and eye.
 1036. Burns and scalds.

INJURIES OF THE NECK.

1037. Contusion of the soft parts.
 1038. Fracture of the hyoid bone.
 1039. " cartilages of the larynx.
 1040. Rupture of the trachea.
 1041. Dislocation of the hyoid bone.
 1042. Wound.
a. Superficial.

- b. Cut throat.*
c. Gun-shot.
d. From the mouth.

1043. Injuries of vessels. (Specify which.)
 (992.) *Burn and scald of the larynx.*
 1044. Foreign bodies in the air-passages.
 1045. " pharynx
 1046. " œsophagus.
 1047. Injury of the pharynx and œsophagus by corrosive substances.

INJURIES OF THE CHEST.

1048. Contusion.
 1049. Fracture of the ribs (including costal cartilages) without injury to lung.
 1050. Fracture of the ribs (including costal cartilages) with injury to lung.
 1051. Fracture of the sternum.
 1052. Wound of the parietes.
 1053. Perforating wound of the chest.
 1054. Penetrating wound of the pleura or lung.
 1055. Wound of the anterior mediastinum.
 1056. Wound of the pericardium and heart.
 1057. Injuries of vessels. (Specify which.)
 1058. Rupture of the heart or lung without wound or fracture.

INJURIES OF THE BACK. (Including the whole spinal region.)

1059. Contusion.
 1060. Sprain.
 1061. Wound.
 1062. Fracture and dislocation of the spine.
 1063. Injury of the cord, without known fracture.

INJURIES OF THE ABDOMEN.

1064. Contusion.
 1065. Contusion with rupture of muscles.
 1066. Contusion with rupture of viscera.
 1067. Wound of the parietes.
 1068. Wound of the parietes, with protrusion of uninjured viscera.
 1069. Wound of the parietes, with protrusion of wounded viscera.
 1070. Wound of the parietes, with wound of unprotruded viscera.
 1071. Wound of viscera without wound of parietes.

1072. Injuries of vessels. (Specify which.)
 1073. Foreign bodies in the peritoneal cavity.
 1074. Foreign bodies in the stomach.
 1075. Foreign bodies and concretions in the intestine.
 1076. Fistula from injury, and artificial anus.

INJURIES OF THE PELVIS.

1077. Contusion.
 1078. Wound of the male perineum, scrotum, and penis.

1079. Wound of the female perineum and vulva.
 1080. Wound of the vagina and internal female organs.
 1081. Wound of the rectum.
 1082. " anus.
 1083. " bladder.
 1084. Rupture of the bladder without wound.
 1085. Rupture of the bladder from fracture.
 1086. Injuries of the pregnant uterus.
 1087. Injuries of vessels. (Specify which.)
 1088. Foreign bodies in the vagina.
 1089. Foreign bodies in the rectum.
 (570, 601.) *Foreign bodies in the bladder and urethra.*
 1090. Fracture and dislocation of the pelvis.
 1091. Fracture and dislocation of the pelvis, with rupture of the bladder or urethra.

INJURIES OF THE UPPER EXTREMITIES.

1092. Contusion.
 1093. Sprain. (Specify which joint.)
 1094. Wound.
 1095. " of joint.
 1096. Injuries of vessels. (Specify which.)
 1097. Foreign bodies imbedded.
 1098. Separation of epiphyses.
 1099. Greenstick fracture, or bending of bone. (Specify which bone.)
 1100. Fracture. (State whether simple or compound.)
 1101. Fracture of clavicle.
 1102. " scapula.
 1103. " humerus.
 1104. " forearm.
 1105. " carpus, metacarpus, and phalanges.
 1106. Ununited fracture, or false joint. (Specify which bone.)
 1107. Dislocation. (When compound, to be so stated.)
 1108. Dislocation of the sternoclavicular joint.
 1109. Dislocation of the acromioclavicular joint.
 1110. Dislocation of the shoulder.
 1111. " elbow.
 1112. " wrist and carpus.
 1113. " thumb.
 1114. " phalangeal joints.

INJURIES OF THE LOWER EXTREMITIES.

1115. Contusion.
 1116. Sprain. (Specify which joint.)
 1117. Wound.
 1118. " of joint.
 1119. Injuries of vessels. (Specify which.)
 1120. Foreign bodies imbedded.
 1121. Separation of epiphyses.
 1121. Fracture. (When compound, to be so stated.)
 1122. Fracture of the femur.
 1123. " cervix femoris.

¹ Register the diseases printed in italics, not under this heading, but at the place referred to in each instance by number within brackets.

1124. Fracture of intrascapular.	1135. Dislocation of knee.	INJURIES OF THE ABSORBENT SYSTEM.
1125. " trochanter major.	1136. " head of fibula.	
1126. " patella.	1137. " foot at the ankle.	
1127. " leg, both bones.	1138. Dislocation of the foot, at calcaneo-astragaloid, and scapho-astragaloid joints.	1142*. Foreign bodies and concretions.
1128. " tibia alone.	1139. Dislocation of the foot, at astragalus.	1143. Wound of lymphatics.
1129. " fibula alone.	1140. Dislocation of the foot, at os calcis.	INJURIES NOT CLASSIFIED.
1130. " bones of the foot.	1141. Dislocation of the foot, at other tarsal bones.	
1131. Ununited fracture, or false joint, (Specify which bone.)	1142. Dislocation of the foot, at metatarsus and phalanges.	
1132. Dislocation. (When compound, to be so stated.)		1144. Rupture of muscle.
1133. Dislocation of the hip.		1145. Rupture of tendon.
1134. " patella.		1146. Foreign substances in the cellular tissue.

The topics of the Appendix to the Nomenclature of the College of Physicians—namely, HUMAN PARASITES and CONGENITAL MALFORMATIONS—have been already described at pp. 148-150, and 252-254, of this Text-Book, where the lists are printed under the subjects which they embrace as "Topics relative to Pathology."



THE SCIENCE AND PRACTICE OF MEDICINE.

PART III.

THE NATURE OF DISEASES—SPECIAL PATHOLOGY AND THERAPEUTICS.

It is my object in this part of the Text-book to treat of the diseases comprehended in the two groups or classes into which the London College of Physicians has arranged them—namely, GENERAL and LOCAL. The first—namely, GENERAL DISEASES—comprehend the two sections A and B, as defined at p. 335, *ante*.

Section A commences with what are generally known as *specific* or *miasmatic* fevers—terms which may be sufficiently understood, but which are far from being unobjectionable. Next come the *malarious fevers*; and then follow the *epidemic* disorders, which differ more or less distinctly from the previous members of the group. Lastly come those *febrile affections* which are either solely or occasionally excited by the introduction of some animal matter in a state of change.

Section B embraces the *constitutional diseases*, which are to be described, together with those forms of disordered nutrition in which the affection, whether localised or not, has a tendency to invade more organs than one in the same individual, either simultaneously or in succession.

In the following chapters on topics relative to the pathology of the GENERAL DISEASES, it is intended to describe—*First*, The common properties or characters peculiar to *specific* or *miasmatic* diseases. *Second*, The nature of *malarious* diseases. *Third*, The nature and management of epidemics of disease. *Fourth*, The nature of constitutional diseases; and, *Fifth*, To describe in detail the several diseases individually, their *general nature and causes*; *pathology and morbid anatomy*; *symptoms, course, and complications*; and their *diagnosis, prognosis, and treatment*.

The employment of the words *miasmatic* and *zymotic*, as they are terms in common daily use, and expressive of preventible diseases whose causes are removable, ought not to be understood as signifying more than this, nor expressing scientifically any rigidly defined group of diseases. The terms are used to bring together those diseases which possess the peculiar character, in common, of suddenly attacking great numbers of people, at intervals, in unfavourable sanitary conditions. The diseases so named, in the language of Dr. Farr, “distinguish one country from another—one

year from another; they have formed epochs in chronology; and, as Niebuhr has shown, have influenced not only the fall of cities, such as Athens and Florence, but of empires; they decimate armies, disable fleets; they take the lives of criminals that justice has not condemned; they redouble the dangers of crowded hospitals; they infest the habitations of the poor, and strike the artisan in his strength down from comfort into helpless poverty; they carry away the infant from the mother's breast, and the old man at the end of life; but their direst eruptions are excessively fatal to men in the prime and vigour of age. They are emphatically the *morbi populares*" (Appendix, p. 76, *Sixteenth Annual Report of the Registrar-General*, 1856). The name *Zymotic* is not to be understood now as implying the hypothesis that these diseases are fermentations, which the derivation of the term would lead one to believe. It has become extensively used of late as applied to the diseases whose characters as a group are already indicated, and for which some convenient term was required. The group, then, to which the term ZYMOTIC has been applied, was intended to comprehend all the principal diseases which have prevailed as *epidemics*—all those which are due to *paludal* or *animal malaria* (*endemic*), and those which are due to *specific disease poisons*, capable of propagation from one human being to another, and *communicable either by direct contact*, or indirectly through various channels of human intercourse, contaminating drinking-water or infecting the air, *or by animals in a state of disease*, as well as *dietic and parasitic diseases*.

The following chapters are therefore intended to set forth the salient points in the Pathology of Class A of the General Diseases, so far as they can be conveniently grouped together.

CHAPTER I.

ON THE NATURE OF THE ACUTE SPECIFIC OR GENERAL DISEASES, COMMONLY CALLED MIASMATIC.

"IN a rude manner, the ACUTE SPECIFIC or GENERAL DISEASES may be classed as the first section of LOCAL DISEASES—the blood itself being regarded as the organ affected." In the greater number of the diseases to which the name *specific* or *miasmatic* may be given, the *blood* is more or less changed, and by some is presumed to be the first localisation of diseases which result from *specific poisons* of organic origin, either derived from without or generated within the body. These *specific poisons* tend to produce in the blood an excess of those decomposing organic compounds which physiology teaches us are always present to some extent in the circulating current. The GENERAL DISEASES constitute a group sometimes termed "*acute specific*" (WALSHE), because they primarily and essentially implicate the entire system. Throughout their course, and from the first, they each variously modify the composition of the blood, the calorification and the enervation of the body. Each of them,

during their progress, gives rise to some lesions in the textures, of a special anatomical character, when the disease is not too rapidly fatal to allow of these pathological features to become developed, as is sometimes the case in *yellow fever*, *typhoid fever*, *plague*, *cholera*. These maladies run an acute and rapid course; they are attended with more or less fever; and in the majority of instances, the fever which accompanies them has a fixed duration and range of temperature. The greater number of them are contagious, or capable of being propagated from person to person, by inoculation or under certain conditions not yet well understood; and, lastly, all of them are produced by an extrinsic poison, either of a *miasmatic* or *specifically contaminating nature*, or by the implanting of a *specific virus*.

The *specificity*, so to speak, of these diseases, consists in certain characters which distinguish each of them from any other disease, and in the constancy by which, from time immemorial, such characteristics have continued to distinguish them. Although medical opinions regarding their pathology may change, yet the essential characters of these "*specific diseases*" are not known to change. Each of them observes a constancy and regularity of plan in the construction and development of its morbid processes (PAGET). Each of them has some essential character or characteristics by which they are severally distinguishable. The course of the febrile phenomena in them is found to be distinctive; the duration of the febrile state not less so; and also the anatomical signs which distinguish the local lesions, the development of which are concurrent with the general or constitutional phenomena. But of all truths relating to the phenomena of disease, the most important are those which relate to the *order of succession of the phenomena of specific diseases*. In this respect *specificity* cannot be denied to those diseases in which, during their natural course, every phenomenon is related (in a uniform manner, so far as *exact* investigation has extended) to certain phenomena that co-exist with it, and to others that have preceded and will follow it. When it is found that a series of phenomena occur in (thousands, millions) a number of instances in the same order, within similarly uniform periods of time, and altogether with so much regularity that those who are instructed, on visiting a patient for the first time, can not only affirm what has gone before, but may predict what is to come after (the highest achievement of science), it is impossible to avoid concluding that such an invariable sequence has as constant a cause. This conclusion flows from the very constitution of our nature, and is inevitable. On our knowledge of the facts relating to such order of succession is founded every reasonable anticipation of future events, and whatever power we possess of influencing those phenomena in the management of the disease, to the advantage of our patients and the community at large. When it is found, moreover, that there are many series of these phenomena, which may be called A, B, C, D, &c., occurring in different persons, and at different times, all perfectly distinguishable, and never by any chance capable of being confounded by a properly trained person, it is impossible to avoid concluding that the causes of A, B, C, D, &c. are not identical, and must be in fact dissimilar. Moreover, mere *uniformity* in the sequence of such phenomena as obtains in the natural course of the respective

diseases is of itself enough and sufficient (to most minds) to warrant the belief that the diseases they represent are specific. The term "specific," from this point of view, necessarily means that such unlike effects must have unlike causes; and the term "specific," as we use it, is derived from the fact that (following the analogy of natural history) the different diseases just named, A, B, C, D, &c., have been considered as so many different species, preserving their individuality through all time, as the rose, the apple, the dog, the whale, or any other animal or plant preserves theirs. Like animals and plants, also, such specific diseases may disappear from off the face of the earth, when they can no longer "struggle for existence" against the well-directed measures of sanitary science. These measures may eventually render the existence of many specific diseases an impossibility—as much so as the existence of a megatherium, or even a wolf, would now be an impossibility in this country.

The *origin* of all specific diseases, or "how their respective first *contagia* arose," is alike unknown. "This in Pathology is just such a question as in Physiology is 'the origin of species.' Indeed, it is hardly to be assumed as certain that these apparently two questions may not be only two phases of one. Hourly observation tells us that the *contagium* of *small-pox* will breed *small-pox*, that the *contagium* of *typhus* will breed *typhus*, that the *contagium* of *syphilis* will breed *syphilis*, and so forth; that the process is as regular as that by which dog breeds dog, and cat cat,—as exclusive as that by which dog never breeds cat, nor cat dog; and prospectively we are able to predict the results of certain exposures to contagion as definitively as the results of any chemical experiment. But retrospectively we have not the same sort of certainty; for we cannot always trace the parentage of a given case of small-pox or measles" (SIMON, *Sixth Report on Public Health*, p. 54). The same may be said of animals; given any individual calf, cat, dog, or child, we cannot always trace its parentage.

But this view of the *specificity* of each of these diseases is also held by some independently of the causation of them being also specific. This is essentially the view adopted by Dr. Murchison in his observations on the origin of fevers—a multiple origin, in fact. Hence another important question has been much debated of late regarding the etiology of the acute specific diseases, namely—"whether they can arise spontaneously?" *Typhoid fever*, for example, is held by some to be undoubtedly contagious; and being so, that it can never originate *de novo*. That is to say, as it can be proved in some cases to have its origin in the entrance of the emanations of the sick into the previously supposed healthy, it can never arise in any other way. On the other hand, while admitting that the disease may be spread by the emanations from those suffering from the disease, it is said that the admixture of sewage decomposing animal excreta, with fluids used for drinking purposes, *although no typhoid excreta are present*, will yet produce typhoid fever *de novo*, in those that drink the polluted fluid; and it is further said, that the inhalation of sewer-gas is sufficient itself to produce in persons previously healthy typhoid fever *de novo*. Writing thus in 1875, Sir William Jenner was of opinion, from the then known facts, that the question should be held to be still *sub judice*; and that it is only to be solved by the combined efforts of those who can investigate the origin of

typhoid fever when it occurs as an epidemic, or in the form of severe local outbreaks; and especially by those practitioners who, being engaged in private practice in country districts, can examine into and report on the solitary cases occurring under circumstances favourable for the exclusion of fallacies. It is rare that solitary cases can be satisfactorily investigated in towns, where typhoid fever is practically endemic; and where the network of drains prohibits successful investigation. As the question at present stands as regards typhoid fever, "it well illustrates our ignorance, the difficulty of ascertaining facts, the danger of reasoning upon insufficient data, on an insufficient number of facts, on insufficiently observed facts, and the unwisdom of drawing general conclusions from facts which bear only on one side of a question." By those who advocate the sole origin of *enteric fever* by contagion, one general assumption is made, namely,—“that no admittedly contagious disease ever originates in any other way than by contact with the emanations from the sick, and, therefore, that *enteric fever* spreading by contagion, can be spread in no other way.” Sir William Jenner considers that this assumption is still wanting in such proof as should convey conviction to any unprejudiced mind accustomed to investigate questions of this nature. Although personally prejudiced in favour of this opinion—of the specific origin of *enteric fever* in common with all contagious diseases—and having long advocated the specific differences (of these diseases), and held that each had its specific cause—*i.e.*, a cause which is unable to produce any other acute specific disease—Sir William Jenner is nevertheless constrained to admit that at this moment the weight of evidence and of argument is rather on the other side. If each case could, in every instance, be referred to emanations from those previously sick of the same disease, it would greatly strengthen those general views which Sir William Jenner has long held and advocated; but when an admittedly contagious disease is epidemic, it is evident that facts to elucidate the question of its spontaneous origin cannot be collected. There is so much of the special poison in the locality, that no one can be sure that he has not, in some way, been exposed to its influence. But when the disease is no longer epidemic—when isolated cases occur, then it is that, if a disease never originated *de novo*, we ought, in the majority of cases, to be able to trace back (if we inquire carefully) the apparently spontaneous case to its source.

Of *small-pox*, the most contagious of all diseases, Sir William Jenner does not hesitate to say, that the large majority of *solitary* cases can be traced (with due care) to the probable source; and that when a case has been imported into a locality previously free from the disease, new cases almost invariably spring up in its vicinity. Thus the contagious origin is proved—(1.) By tracing the case to its source; and (2.) By the new cases which spring up from the first isolated case. *Small-pox* is eminently contagious, and not only so, but from the vast proportion of instances in which we can trace isolated cases to their source, from the rarity of the cases in which it is impossible to trace the probable origin to contagion, we may fairly conclude that *small-pox* never originates *de novo*.

On the other hand, again, it would appear that there are diseases of which—all being contagious—some are more and some are less contagious than others. In this respect *small-pox* is at one end of such a scale,

and *diphtheria* and *erysipelas* at the other end. These two diseases are undoubtedly both contagious, but they are not so contagious as *small-pox*; for it is more common for the healthy exposed to the emanations of the sick to escape from these diseases, than it is for those exposed to the effluvia of *small-pox* to escape *small-pox*. Again, in regard to *small-pox*, while it is the exception not to be able to trace the source of its contagion in any given case, in *diphtheria* it is comparatively rarely that the first case, even of several, can be traced back to contagion. In regard also to *erysipelas of the face and head*, undoubtedly contagious, it is the exception to be able to trace individual cases to contagion. Reasoning, therefore, *à priori*,—"the more contagious a disease is—*i.e.*, the less the quantity of poison needed, the shorter the exposure to the poison necessary, and the less preparation required in the system of the individual receiving the poison—the less frequently should we expect that the disease would be traced back to its primary source; while with diseases of the class which are the *least contagious*—*i.e.*, those which require for their spread a larger exposure, a more decided dose of the poison, or the administration of that poison in a particular way, by a special channel—we should expect that in every new case (if every one were due to emanations from the sick of the same disease) we should the more easily be able to trace them to their original source, the more easily should the parent case be discoverable. But it is not so; the facts are otherwise; for, those diseases which are the most contagious are the most commonly traceable to their origin; and those which are the least contagious can the least frequently be proved to have originated in contagion. Hence Sir William Jenner is of opinion that we are thus driven to the conclusion that *erysipelas* frequently, and *diphtheria* not uncommonly, originate *de novo*;—that *enteric fever*, holding, like *diphtheria* and *erysipelas*, a low position in regard to its contagious quality, is also in the same category as a disease possibly arising *de novo*. It rarely spreads, excepting when the *excreta* from the sick are administered by a particular channel; and Sir William has never seen a case removed from its place of origin to give the disease to the inmates of a house into which it was removed, unless there were a communication between the source of the drinking fluids and the bowel excreta of the patient. But that *typhoid fever* can spread from one person to another without the swallowing of the excreta, if the person be exposed to the concentrated emanations from the sick frequently and for a considerable time, there can be no doubt. Twice has Sir W. Jenner known students contract the disease who were diligently taking the temperature of enteric fever cases (before the registering thermometer was in use), being thus obliged many times daily to put their head into the bed of the patient. Having thus such limited power of propagating itself, that the poison must be in large doses, or long breathed, or taken into the stomach to produce the disease, it ought, in the majority of cases in country places and isolated houses, to be able to be traced to its source, *if the source be in every case the excreta or emanations of those suffering from the disease*. But the facts are said to be otherwise. Its origin has not hitherto been able to be traced to pre-existing cases in a very large proportion of the solitary cases observed. And not only do numerous cases occur in which *enteric fever* is not traceable to exposure to emanations or excreta from the sick of

enteric fever—i.e., to contagion; but there are a sufficient number of cases to render it *probable* that the admixture of sewage (not typhoid) with drinking water, may produce the disease *de novo*; and most of us have met with cases in which the breathing of greatly diluted sewer gas continuously for some time, especially at night, has seemed to produce *typhoid fever de novo*. Nevertheless, new cases, sceptically scrutinised and carefully recorded, are still required to settle the question. It is only in exceptional instances that cases free from all possible, at least from all probable, sources of error can come under notice; and such cases can only be seen in *private practice*, especially *country practice*. Hence the continued necessity of carefully observing and reporting such cases. And, although Sir William Jenner does not say, and does not think, "that the arguments and facts able to be adduced in favour of the origin, *de novo*, of the contagious diseases are conclusive, he still maintains that they are strong enough to make us pause before we accept the theory advocated by Dr. William Budd, and to which Professor Tyndall has lent the weight of his great name—a weight which would, however, be greater on this point in question if he had leisurely studied the subject in which he has addressed the public in a strain calculated to check unprejudiced individual inquiry" (President's Address to Clinical Society of London, Feb. 12, 1875). It must at the same time be conceded, that if small-pox is contagious, as it is universally admitted to be, and if it is impossible also to believe that it can originate *de novo*; it appears to me just as impossible to believe that any disease, if it is similarly proven to be specific and contagious, can originate *de novo*, as to realise that any plant or animal can come into existence spontaneously. If such a disease as enteric fever can be proven to arise *de novo*, then I conceive that it cannot be classed with those diseases which are *specific* in the sense in which that word is used in this chapter.

But there appears to me to be such good evidence of the specific and contagious nature of *enteric fever* from just such men as Sir William Jenner desires to have records, that to my mind the facts already existing *do not point to a de novo origin, but that the weight of evidence is in favour of a specific contagium vivum*. Dr. Robert S. Hudson, of Redruth, has furnished a record of such cases. Educated to believe in the non-contagious nature of enteric fever, and that it was begotten of filth (pythogenic), that typhoid stools, however concentrated, do not poison the air, but must be mixed with decomposing sewage to acquire their virulence, and that decomposing sewage—animal matter—in a state of putridity is sufficient to develop the poison *de novo*, and produce the fever, he found that this theory failed to explain the facts coming within his own experience. Hence he became converted to believe in the *contagium vivum* theory of its causation from the cases he had seen in his practice. In Redruth *enteric fever* is rare, and is never epidemic. Few districts are more favourably situated for the study of infectious diseases than the peninsula corresponding to West Cornwall. The main lines of traffic, whether by road or rail, may be said to converge at Truro. There is little cross-country commerce, except in the neighbourhood of mines; and the sea, which surrounds the district to the greater part of its extent, forms an effectual barrier to the admission of any *contagium*, unless at Falmouth, Penzance, St. Ives, Hayle, and a few

of the minor ports. The cause of any infectious disease arising from without might, therefore, readily be traced by proper sanitary organisation, and at little cost the whole district might be kept as free of epidemic disease as were the Scilly Islands during the ten years 1851 to 1860, when not a single death was reported from *small-pox*, *measles*, or *diphtheria*. The majority of those attacked at Redruth had recently visited or came from neighbouring towns. It has no common system of sewers to which water-closets are attached. It has but one source of a pure, though insufficient water supply. The cases of enteric fever in his experience he has invariably traced to importation. In a population of nearly 11,000 (2,000 being suburban), there were during six years forty-one convalescents from enteric fever admitted to the Miners' Convalescent Hospital, only three of which came from Redruth, and these from the suburban districts, none from the town. It also appears that of these forty-one cases, thirty-eight were chargeable to six hamlets miles away from one another, and without any apparent bond of connection, showing how typhoid, once introduced, lurks in certain spots, to break out into virulence generally every autumn. One of these hamlets, four miles from Redruth—namely, Portreath—had an outbreak of enteric fever, traceable to an imported case from Truro. It spread to thirteen people, and the conditions of filth which existed previously to, and during the outbreak, were no worse than they had been for years. Hence, just like small-pox, his cases were found to be contagious, (1.) by tracing the first case to its source; (2.) by the new cases springing up from the first isolated case. If filth in a state of decomposition could of itself generate the specific poison which produces such a definite train of symptoms, is it not strange that Portreath should have been free ever since that outbreak in 1874, and that its appearance in the village should have then been coincident with the arrival of a girl from a fever-stricken house twelve miles distant? And, again, if filth in a decomposing state originated typhoid, or scarlet fever, or measles, why do these diseases appear at certain times and follow certain tracks of communication, travelling as surely in certain directions as an engine on a railway? The conditions of filth are only powerful in the spread of the disease, but do not in any sense originate it; as precisely similar conditions exist in other parts of the country to an equal extent, yet no typhoid. Fæcal fermentation in the cess-pits of Redruth (as of other similar places) is of everyday occurrence, yet no typhoid (*Brit. Med. Journ.*, 16th June, 1877, p. 740).

Again, Dr. George Buchanan, as President of the Association of Medical Officers of Health, "On Some Directions for Scientific Work," makes the following observations respecting the origin and means of diffusion of the so-called zymotic diseases:—

"Over and over again, one or other of these diseases is witnessed, arising under conditions that are not at once explicable by current notions of their production and dissemination. For a first example, let us consider some of the difficulties that we are liable to encounter in interpreting the occurrence of a disease so comparatively well understood as enteric fever. In an out-of-the-way house, looking as if isolated from all chances of infection, where there has been no previous sickness for months or years, but where a number of common nasty conditions exist,

a case of this fever occurs, and the impulse of the doctor who investigates it is to say that the disease has had a *de novo* origin from the ordinary excrement-filth of the place. Whether or not this has been so is a question of the highest moment to science, but by no means to be settled out of hand by the apparent absence of specific cause in a few instances. Repeated observations and very complete record of all circumstances are wanted before any deduction can be properly drawn from such an occurrence as I am supposing. Often room has been left in the records of such cases for doubt as to the nature of the disease in question; or an oversight on a point so demonstrably important as the period of incubation has led observers to hasty and untenable conclusions; or the possibility of dangerous material passing a long distance through a soil or along a stream may not have been held in view; or some vehicle for the conveyance of specific contagion may have been overlooked. If we consider how, a dozen years ago, before Dr. Ballard's great piece of inductive work, which our Society is proud to have among its archives, those many wonderful outbreaks of enteric fever that we now trace to the agency of milk were unintelligible, and how all of them, instead of being merely regarded as unexplained, were liable to be regarded as demonstrating the *de novo* origin of the disease—we shall the better understand the caution with which it is necessary still to proceed before we can affirm the adequacy of any common filth condition to originate enteric fever. Even since we have known of milk, it would not until lately have occurred to many of us to suspect the baker and other food-purveyors as possibly distributing fever-contagium along with their daily supplies of articles of diet. Obviously we have to be incessantly on the lookout, not only for these, but for other and even obscurer vehicles for the specific poison; and there are most serious difficulties in arriving at a definite conclusion that no antecedent case has been concerned in the production of a given fever occurrence. But supposing, by exhaustive inquiry into a sufficient number of such cases, we satisfy ourselves that the fever must have had in a sense a *de novo* origin, we are only at the entrance of one of the most important problems concerning the disease. These particular houses, which have got enteric fever in them without any chance of the disease having been imported, wherein do they differ from the multitude of apparently similarly circumstanced houses that have in them no fever at all? and what are the circumstances of to-day that have produced the fever to-day and that did not produce it six months ago? Quite certainly there must be differences, or the effect would not be different. The nature of these differences is, however, in such a case as I am supposing, beyond our present knowledge; and it is by the study of differentiating circumstances, in cases where we can exclude antecedent occurrence of the disease, that we may best hope to discover the nature and affinities of the special material of the fever. This much is clear, that, whether our next step be to find out means of infection-conveyance, at present ill-understood or unsuspected, or whether it be to learn fresh lessons respecting the infection itself, the step may be expected to be gained by following directions of research that are peculiarly open to officers of health to follow" (*Medical Times and Gazette*, Oct. 30, 1875).

But in addition to such facts and arguments as have been supplied by clinical and epidemiological observations regarding these important questions concerning the acute specific diseases, there are the more recent experiments of a pathological kind which require careful consideration.

These especially relate, (1.) to the *occurrence of organic forms* and their *identification* as infective agents in connection with contagious and infective diseases; (2.) to the explanation of the successive changes which these effect on the living body, from the beginning to the end; (3.) to the intimate etiology of the acute infective diseases—the results of which all tend to establish the general doctrine of the *existence and vitality of contagia*. The first of such studies were instituted, and the first steps of discovery were made in connection with investigations regarding “a contagious fever of horned cattle,” made at the instance of H. M. Government in 1865, in aid of the then Cattle Plague Commission; when “Dr. Beale, working at the microscopy of the disease, drew attention to the swarms of extremely minute particles which he found universally present in the textures and juices of the animals; and which he believed to be the *contagium* of the disease.” It is only very recently, therefore, that we have had the beginning of any true insight into the respective *contagia* which excite these diseases through experimental inquiries.

In the class of ACUTE SPECIFIC or GENERAL DISEASES there are, from this point of view, two sets of morbid processes to be distinguished, namely—(1.) Those by which the seeds of disease are introduced from outside; and, (2.) Those by which disease, having already taken root in the organ or tissue, *affects* or rather *infects* the whole organism. In the former set of cases we attribute the results observed to the action of a *morbid poison* or *contagium*; in the latter, all that we are able to observe is either that the primary process spreads from the part first affected to other parts which are connected with it by continuity of structure, or by channels of absorption, or that it repeats itself in distant organs. To these processes of extension the term “*infective*” is applicable—the affected parts acting towards neighbouring, or even distant, parts as sources of infection (BURDON SANDERSON). Such theory being accepted, we must concur in the opinion expressed by Dr. Burdon Sanderson, “that no contagious disease can originate *de novo*, any more than that an organism can originate *de novo*.” There are some, however, who think otherwise on both questions. About some infections (*contagiums*) we are *practically* certain that the true plant never comes up unless the seed has been sown—*e.g.*, *syphilis*, *small-pox*, or *hydrophobia*. About others more latitude may be allowed. But during the past eight or ten years the notion of *infectiveness* as an important element in disease processes, has become more familiar than formerly. The progress of pathological knowledge has induced us to recognise *infective action* in a much larger number of instances than formerly. No one now hesitates to include among infectious diseases some in respect of which we have no knowledge of the infective agent. There are few now who doubt that a tuberculous process, once begun, spreads infectively. Similarly, it has been long familiar that an inflamed part is a focus from which irritating material is distributed to healthy parts by radiating lines of absorption; but it is only of late years that it has been distinctly seen and recognised clinically, that every exudation-liquid of an inflamed part carries with it more or less the properties of an inflammation-producing virus (phlogogenic). Hence, Dr. Burdon Sanderson distinguishes inflammation into *two* classes—infective and noninfective. In the noninfective (or normal) inflammations,

the pathological effects are co-extensive with the injury, so that no tissue is destroyed, except so much as has been spoilt by the *noxa* itself; in the infective the surrounding tissues are damaged, and to a greater or less degree destroyed by the extension of the pathological process—a destruction which constitutes the source of the common infectivity of wounds.

With regard, therefore, to these ACUTE SPECIFIC or GENERAL DISEASES there are two pathological theories to be noticed, each of which has been the subject of much contention—namely, the theory of a "*contagium vivum*," and the "*germ*" theory—two theories so closely associated in the mind of many persons, that they sometimes seem to be regarded as identical. In reality they have no necessary connection with each other. The expression *contagium vivum* is meant to designate the doctrine that, when a contagious disease is communicated by the atmosphere, by personal intercourse, or in any other way, that conveyance takes place by *specifically endowed organisms*, which stand in a similar relation to the disease to that in which the seed does to the plant. For just as the seed is at once the origin and offspring of the plant, so, according to this view, the *morbific contagium* produces the disease and is in its turn produced by it. This being understood, it is evident that the term *contagium vivum* relates exclusively to SPECIFIC DISEASES, and therefore belongs mostly to Medicine. On the other hand, the *germ theory* teaches that certain organised and living particles of *extreme*, if not *ultra-microscopical minuteness*, which are always suspended in the atmosphere, are in such sense the causes of the suppuration and other destructive changes which interfere with the healing of a wound; and that (1.) if these organisms are excluded, the wound must enter at once on the processes of reparation; and, (2.) that if these organisms are present, it cannot so heal; and, consequently, that the whole secret of the successful treatment of wounds consists in the expulsion of that finest form of atmospheric dust, which, whatever be the size of its particles, has been identified, as well by physicists, such as Professor Tyndall, and by mycologists, such as Nägeli, with the contaminating agents in question. Hence, the "*germ theory*," as it relates to the inflammatory and other consequences of injuries, involves questions which almost exclusively belong to Surgery, the object of surgical treatment being "*to defend the patient against the septic germ*" (BURDON SANDERSON in *Brit. Med. Journ.*, 1878, p. 179).

The one common property of the acute specific diseases is that they are all contagious; that is, "from some part of the sick *something* is given off which can, when properly applied, excite the same disease in another, and which can excite no other disease. Little is known of what this *something* is; and yet it is most important for the prevention and even for the treatment of these diseases that we should know. Little is known of those conditions of the system which render this *something* sufficient to the production of the disease in one person, while in another it is inert. For example, at a certain time one of these diseases is what is called *epidemic*; scarcely a person who has not had it, and is exposed to the emanations of the sick, escapes taking it. On another occasion the disease is not epidemic, and then many with equal degrees of exposure escape" (Sir WILLIAM JENNER). The *contagium*, therefore, of these acute specific diseases is *something* that is alive—*something* which

exhibits the phenomena of growth and self-propagation; and the doctrine of a *contagium vivum* has been proven to be undoubtedly true as regards some contagious fevers. "In an argument of this kind, it is of capital importance to get hold of an authentic instance; because it is more than probable, looking to the general analogy between them, that all infectious diseases conform, in some fashion, to one fundamental type. If *septic bacteria* are the cause of *septicæmia*—if the *spirilla* are the cause of *relapsing fever*—if the *Bacillus anthracis* is the cause of *splenic fever*—the inference is almost irresistible that other analogous organisms are the cause of other infectious inflammations, and of other specific fevers" (Professor WILLIAM ROBERTS, M.D., F.R.S., *Brit. Med. Journ.*, August, 1877, p. 168). It is therefore necessary to state the scientific position of the issues involved in language as clear and precise as possible; and that first on one side and then on the other.

In regard to *vaccinia*, *small-pox*, *sheep-pox*, *diphtheria*, *erysipelas*, and *glanders*, the virus of these diseases, it is believed, has been proved to consist of minute particles having the character of micrococci; and that in regard to *typhus*, *scarlet fever*, *measles*, and the rest of the contagious fevers, their connection with pathogenetic organisms is as yet a matter of pure inference (Prof. W. ROBERTS). The able reports of Dr. Braidwood and Mr. Vacher on the life-history of *contagium*, made on behalf (and published in the journal) of the British Medical Association, from 1875 to 1877, furnishes the following details:—It is only within a few years previous to 1855, that scientific research has been directed to the discovery of the vital and physical properties of *contagium*, or the laws which regulate its behaviour in the propagation of disease. The points thus inquired into are,—(1.) What is *contagium*? (2.) In what manner is it generated or communicated? (3.) What are the conditions on which its life or activity depends? Modern research has not yet fully answered these questions; but as to the first—namely, "What is *contagium*?"—"it is characteristic," says Dr. Burdon Sanderson, "of many of the communicable diseases, that at some period in the course of their development liquids exist in the diseased body, whether human or animal, which have the power of reproducing the original disorder when brought into contact with the living substance of a healthy individual. Such liquids are said to be virulent or infecting. Their occurrence in human pathology is familiar to us in *small-pox* and *syphilis*; and lately we have learnt experimentally to recognise their virulence and infectiveness in *tuberculosis*, and inferentially in other allied diseases. Among the lower animals the maladies of which the liquid *contagium* is known are much more numerous. In some, as in *cattle-plague*, and in *anthrax* or *charbon*, all the tissues and juices become virulent; in others the contagious property is found only in liquids derived from the special seats of diseased processes similar to ordinary inflammation; as, *e.g.*, in *glanders*, in *pleuro-pneumonia*, and in *sheep-pox*." This so-called "*liquid contagium*" is obviously one of two things, either it is itself *contagium*, or a fluid holding *contagium* in solution or suspension; and as there is no evidence that all contagious diseases are communicable by infecting liquids, it is not contended that all varieties of *contagia* are liquid, or dissolved or suspended in liquid, but only that the several *contagia* of diseases known to be inoculable

must be contained in the liquids with which inoculation can be performed. Etiologists and pathologists have thus agreed to recognise an active principle which has essentially to do with the cause of propagation of disease, termed (provisionally) *contagium* or *virus*, which is believed to be contained in the secretions of the affected person. Beale's description of its material substance is the following :—

"The minute contagious bioplast is less than the $\frac{1}{1000000}$ of an inch in diameter, and often so very clear and structureless as to be scarcely distinguishable from the fluid in which it is suspended. Such a minute particle may readily be transferred from the affected organism to an apparently sound organism. It may be carried a considerable distance from its source without losing its marvellous power of causing, in the organism invaded, a series of changes resembling, and often in every minute particular, the phenomena which have occurred in the organism from which it was derived. And it is established that there exist different kinds of contagious living bioplasm, each capable of occasioning specific phenomena which distinguish it. The poison of small-pox will produce small-pox, not typhus fever, or measles, &c. ; nor will any of these produce small-pox. Without, therefore, pretending to identify the actual particles of the living bioplasm of any contagious disease, or to be able to distinguish it positively from other forms of bioplasm, healthy and morbid, present in the fluids on the different free surfaces and in the tissues in such vast numbers, I think the facts and arguments I have advanced prove—(1.) That the contagious virus is living and growing matter ; (2.) That the particles are not directly descended from any form of germinal matter or bioplasm of the organism of the infected animal, but they have resulted from the multiplication of particles introduced from without ; (3.) That it is capable of growing and multiplying in the blood ; (4.) That the particles are so minute that they readily pass through the walls of the capillaries, and multiply freely in the interstices between the tissue elements or epithelial cells ; and, *Lastly*, That these particles are capable of living under many different conditions—that they live and grow at the expense of various tissue elements, and retain their vitality, although the germinal matter of the normal textures, after growing and multiplying to a great extent, has ceased to exist" (BEALE on *Disease Germs*, p. 244).

Dr. Chauveau of Lyons was the first to prove and to contend that *contagium*, so far as vaccine was concerned, consisted of minute particles suspended in the contagium-liquid. His experiments enabled him to conclude that "the vaccinal serosity is not virulent ; and that the activity of the vaccine vesicles in the solid granules, is either in all or only in one part of these little elementary organisms." We thus possess the strongest indirect proof (we can have no direct proof till we have learned how to wash off all trace of plasma from the bodies it suspends) that the *contagium* of the virus with which we are most familiar, consists of transparent particles, not exceeding, according to Sanderson, the 50,000th to 20,000th of an inch in diameter, and neither soluble in water nor in watery liquid, requiring lenses of 1,600 diameter to define them. They are even then easily overlooked, because their refractive powers are similar to that of serum ; and they are best examined with an immersion lens. It is not capable, without losing its properties, of assuming the form of vapour. Fresh

variola matter and the virus of sheep-pox were subjected to similar experiments with a like result, showing that the *contagium* of these diseases is really "particulate." So far the doctrine of a "*contagium vivum*" would seem to have been established on a solid foundation. But there are experiments which point to an opposite conclusion which are still to be considered. Nevertheless "the principle it (the doctrine) involves, if firmly grasped in capable hands, will prove a powerful instrument of future discoveries" (Professor WM. ROBERTS).

As regards the second question—namely, In what manner is *contagium* generated and communicated?—although much labour has been bestowed upon the inquiry, the results are disappointing; and observers are yet at variance whether these minute particles of *contagium* are always produced from like bodies previously existing, or whether they do not, under certain favourable conditions, spring into being *de novo*; whether they are vegetable or animal substances in a communicable state of chemical change, or of the nature of ferments. Of the fungus-theory, the late Dr. Parkes says, "It is clearly a view which would explain many phenomena of the contagious diseases, and has been supported by the experimental evidence of Hallier and many others who have believed either that they have invariably identified special fungi in some of these diseases, or that they have succeeded in cultivating fungi from particles of *contagia*. At the present time, however, the evidence of true and recognisable and special fungi being thus discovered and grown, and forming the efficient causes of disease, is very much doubted by the best observers." That there is any fungus peculiar to cholera stools has been positively denied by Dr. Lewis, in his "Report on the Microscopic Objects found in the Cholera-Evacuations." Many, again, there are who, holding the theory that *contagium-particles* are of the nature of *bacteria*, *vibrios*, or *monads*, wholly reject the fungus-theory. It is not impossible the *contagium-particle* may be a *bacterium* (NÄGELI). Some *contagia* cannot be distinguished from bacteria under the most powerful objectives; while, that organisms (belonging to the same class as these minute bodies) are concerned in many diseases, is clear. Klebs, Recklinghausen, and Burdon Sanderson, have shown what an important part they play in septicæmia; their presence in the blood and secretions of the subjects of many contagious diseases has been demonstrated; and the late Dr. Parkes has written that they have been proved to cause disease of the intestinal mucous membrane, the uterus, the kidneys, and the heart. "They are so widely spread throughout nature, their food is so plentiful, their tenacity of life is so great, and they multiply so rapidly, it is at least possible (as some are inclined to contend) that they may constitute the elementary particles of some of the specific contagious diseases. Experiments regarding their resistance to heat appear to have given contradictory results, until the researches of Professor Cohn of Breslau seem to have solved the mystery. All the confusion has arisen, it is said, from our having failed to distinguish between the growing organism and its seed or spore, between which there is an immense difference in vital endurance. Some spores are shown to have an extraordinary power of resisting heat. Mr. Dallinger and Dr. Drysdale have demonstrated that while the living *septic monads* are killed by a heat of 140° Fahr., the spores of one variety (which are so minute that they cannot be

seen except in mass) are capable of germinating after being subjected to a heat of 300° Fahr. for ten minutes; there is therefore no reason why bacteria should not be able to survive the feebler heat of boiling water. "Two years ago," says Sir Joseph Hooker, "Cohn and Kock observed (1876) the development of spores within the rods of *Bacillus subtilis* and *B. anthracis*. These observations have been confirmed, with important additions, in these two species by Mr. Ewart; and have been extended to the *Bacillus* of the infectious *pneumo-enteritis* of the pig, by Dr. Klein; and to *Spirillum* by Messrs. Geddes and Ewart; and thus a very important step has been made towards the completion of our knowledge of the life-history of these minute but important organisms. Dr. Klein has shown that the infectious *pneumo-enteritis*, or *typhoid fever*, of the pig, is, like *splenic fever*, due to a *Bacillus*. Having succeeded in cultivating this *Bacillus* in such a manner as to raise crops free from all other organisms, Dr. Klein inoculated healthy pigs with the fluid containing the *Bacilli*, and found that the disease in due time arose and followed its ordinary course. It is now therefore distinctly proved that two diseases of the higher animals—namely, "*splenic fever*" and "*infectious pneumo-enteritis*"—are generated by a *contagium vivum*. The practical utility of investigations relating to *Bacillus* organisms (as affording to the pathologist a valuable means of associating, by community of origin, various diseases of apparently different character), is exemplified in the "*Loodiana fever*," which has been so fatal to horses in the East. The dried blood of horses that had died of this disease in India has been recently sent to the Brown Institution, and from seeds therein contained a crop of *Bacillus anthracis* has been grown, which justified its distant pathological origin by reproducing the disease in other animals. Other equally interesting experiments have been made at the same institution, showing that the grains which are so largely used as food for cattle afford a soil which is peculiarly favourable for the development and growth of the spore filaments of *Bacillus*, and that by such "grains" the anthrax fever can be produced at will, under conditions so simple that they must often arise accidentally. The bearing of this fact on a recent instance in which anthrax suddenly broke out in a previously uninfected district, destroying a large number of animals, all of which had been fed with "grains" obtained from a particular brewery, need scarcely be indicated" (President's Address to Royal Society, Nov. 30, 1878). The investigations of the pathological relation of *Bacteria* have thus become of great importance.

The complete distinctness of disease-producing (pathogenous) bacteria from the commoner bacteria of putrescence, has been recognised by Cohn and others; and it is only by the recognition of this complete distinctness that the question of contagion by such germs has made any advance. In addition to the species mentioned under Group I. (p. 151, *ante*) as pathogenous or disease-producing micrococci—namely, *vacciniae*, *diphthericus*, *septicus*, and *bombyces*, several observers (RECKLINGHAUSEN, COZE, FELTZ) have now described other disease-producing micrococci. For example, the cause of the little abscesses (multiple abscesses) in the infective diseases, such as *pycemia*, *puerperal fever*, *typhus*, *acute joint rheumatism*, *urine infiltration*, and *lung gangrene*, are due to *miliary collections of bacteria micrococci*, most common in the kidneys, and especially in the venous

blood-vessels. They have also been seen inside Bowman's capsules, and in the tubules of the kidney (RECKLINGHAUSEN). In cases of variola, Wiegert has seen in the skin, after death, vessel-like passages (blocked up lymphatics) with contents exactly resembling *bacteria*. These bacteria were—(1.) In the vicinity of the pustules; (2.) In the tissues under or in the periphery of the pustules; (3.) In hæmorrhagic patches with or without perfectly developed pustules. They lay in the deeper strata near the subcutaneous tissue, where arteries also were blocked up by them. In the blood of *septicæmia*, *typhoid fever*, *puerperal fever*, there is present a linked or chained bacterium (*Bacterium catenula* of Dujardin), the growth of which has been credited as the efficient cause of these diseases. Professors Coze and Feltz, of Strasbourg (1866), also still further state that "every infectious substance is of bactiferous nature;" that the form of the bacterium is different in *small-pox*, *scarlet fever*, and *measles*, from what it is in *typhoid fever*; that the rapidity of multiplication of *bacteria* is the source of their activity, danger, and specific nature, and not because they are the carriers of any soluble principle. Their forms and size depending upon the soil in which they are produced, the activity and deadly effects of them seem to be due to mechanical causes rather than anything specific. Hence the local lesions in these several diseases are localisations arising from the rapid growth of bacteria—from the detritus formed of them when dead—from leucocythes simultaneously developed in excess—and from fibrillary deposit of fibrine. It has been fully determined by experiment that the blood of animals infected with a poisonous liquid is itself infectious. The red globules of the blood are altered. Multitudes of *bacteria* exist; and in successive inoculations death ensues more and more rapidly, so that the virulence of the infection becomes greater and greater as it passes through several individuals. The virulence or power of the poison multiplies. Mr. Davaine (1872) showed that by injecting blood in *certain* stages of putrefaction into the areolar tissue of several mammiferæ, he produced a disease capable of being transmitted to others in an unlimited series by inoculation of the unputrefied blood of the animals so affected. Majendie had previously recognised the transmissibility of such disease, as well as of an increase in the virulence of the poison when so transmitted; but the experiments of Davaine brought into prominence the *very infinitesimal doses in which the poison retains its activity and certainty of action*. Taking *one drop* of blood putrefied for ten days as unity, and diluting it with distilled water, he inoculated varying doses of blood so putrefied and diluted, which produced a fatal disease. None died with a dose smaller than $\frac{1}{2000}$ of a drop. But transmitted blood (*i.e.*, the septicæmic blood of the animal inoculated with putrid blood) was much the most virulent; and he inferred from his experiment that such transmitted blood found its limit of effect in the *trillionth* part of a drop; whereas for any putrid blood it was $\frac{1}{2000}$ of a drop, thus far confirming the observations of Coze and Feltz. He further showed that such transmitted blood had its virulence destroyed by putrefaction; and that putrefied blood loses its virulence after the putrefaction has passed a certain stage. These *bacteria* die in the body, and especially in the lungs. In *septicæmia* the blood is profoundly altered—tending to putrescence, induced (it is believed) by the *bacteria*; and unless *septicæmia* goes along

with *pyæmia*, bacteria are not present in pyæmia. This is an important distinction between the two diseases. In typhoid blood the presence of bacteria seems to be proven, at least in some cases, but not in all that have been examined after death; but the examinations during life of typhoid blood are still too few. Experiments show that blood taken from human typhoid patients, and injected into the blood-vessels of rabbits, brings about an infectious disease in them (with implications of Peyer's patches); and the blood of rabbits so infected was in its turn able to communicate a similar disease to other rabbits. Further, the dried and powdered blood of an infected rabbit preserved its power to produce infection for more than a year. Articulated bacteria are said to have been found in great numbers in the blood of human small-pox cases by Coze and Feltz; and on injecting it into rabbits, it produced feverishness; and bacteria were found in their blood, similar to those in the human blood; and similarly to typhoid fever, the virulence was intensified in passing through several successive individual rabbits.

But although we have thus a fairly definite idea of the physical characters of such *contagia* as belong to *vaccinia* and *variola*, we know positively nothing as to the organic development, the mode of generation and origin, it being not yet proven that such individual *contagia* are capable of organic development. We have, however, some positive knowledge "with regard to the respective *contagium* and respective morbid processes of *typhoid fever*, *diphtheria*, *erysipelas*, *relapsing fever*, and the *splenic fever* of veterinary practice" (Mr. SIMON), although of the mode of origin and development of the organic forms inseparably connected with these diseases we are yet in ignorance.

The whole subject of the origin and development of *contagia* being still involved in obscurity, researches having for their object the disproving or confirming of any existing theories are of use. Among investigations of this class are those of Drs. Burdon Sanderson, Ferrier, Onimus, T. R. Lewis, and Douglas Cunningham. (a.) They incontestably prove that there is no developmental connection between *microzymes* or *bacteria* and the *fungi* with which their appearance is associated, thus showing that Hallier's theory that *contagia* are *bacteria*, or allied organisms, originating in the nuclei of spores, is untenable. (b.) As regards the theory that *bacteria*—creatures belonging to the lowest stratum of the animal world—are the efficient agents of the contagious diseases, the facts against it are thus summed up by Dr. Murchison:—(1.) *Bacteria* may be injected in large numbers into the blood of the lower animals, and they suffer nothing. (2.) *Bacteria* exist in large numbers in certain tissues of the living body in a state of health. (3.) They are said to multiply very greatly after death in persons who have not died of any infectious disease. (4.) We have been told that *bacteria* are developed in large numbers in a vesication on the skin produced by a *chemical irritant*. (5.) It is also said that in certain contagious fluids the more the *bacteria* multiply the less virulent the fluid becomes. Lastly, there is the statement, made on good authority, that neither *bacteria* nor *bacterial-germs* can be found in certain fluids eminently contagious (*Path. Soc.*, May 4, 1875). Dr. T. R. Lewis has recently (Calcutta, 1879) submitted a most interesting report on the minute organisms which have, from time to time, been found in the

blood of man and of animals, with the following results:—It is admitted that the circulation may become the habitat both of plants and of animals, and of that group of organisms (*Protista* of HÄCKEL) so closely related to both as to be regarded as a third organic kingdom; but, for convenience, it is assumed that the organisms found in the blood are either plants or animals. The blood of man and of all animals contains also an infinite number of mobile molecules, granular masses composed of aggregations of corpuscular elements, which are particularly plentiful in the blood of foetal and newly born animals. It is necessary also to be able to recognise the forms of the cleft-fungi—*Schizomycetes*—or fission fungi. They are minute spherical or oval bodies, multiplied by fission only (NÄGELI), which sometimes remain isolated, at others form unbranched rows (rods, threads), and only occasionally assume a cubiform aspect—e.g., *bacterium*, *vibrio*, *vibrio-bacillus*, and *spirillum*. These fission-fungi do not germinate, and have no relation to the moulds, nor to the sprouting fungi. It is only the various forms of the *fission-fungi* (the group which does not germinate) that have hitherto been unequivocally found in the blood. Dr. Lewis gives the following brief description of the various forms of the fission-fungi (*Schizomycetes*) which have been found in the blood:—

- (1.) *Spherical bacteria*—minute vitalised bodies, barely visible with the highest powers (*Bacterium punctum*, Fig. 76, A);
- (2.) *Elongated bacteria*—almost equally minute cylindrical rods (*Bacterium termo*, Fig. 76, B);
- (3.) *Vibriones*,—short undulating filaments, manifesting somewhat screw-

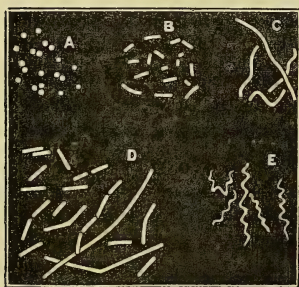


Fig. 76.* × 600 diam.

like movements (Fig. 76, C); (4.) *Bacilli*, or *vibrio-bacilli*—fine short filaments, indistinctly jointed, which, when they attain considerable length, are sometimes described as *leptothrix* filaments (Fig. 76, D); (5.) *Spirilla*—fine, more or less flexible, spiral filaments, which manifest well-marked screw-like movements (Fig. 76, E).

Examples of each of these forms may commonly be detected in the muco-salivary fluid from the mouth of healthy persons; and the question naturally suggests itself,—

Under what conditions are organisms of this character found in the blood? By some, the blood in health is held to be absolutely free from anything of the kind (PASTEUR); and by others “that the blood of man is not free from them; and that there is probably not a tissue in which these germs are not” (BEALE). This simple question of fact has not been yet settled; but “it is very certain that their existence in the plasma of healthy blood is of comparatively short duration” (T. R. LEWIS); and that healthy blood is not conducive to the growth of bacteria, has been definitely set at rest by the experiments of Drs. Lewis and Douglas Cunningham, as well as by the observations of many pathologists. *Bacteria*, *vibrios*, *bacilli*, very speedily disappear from the *liquor sanguinis* when introduced into it during life in considerable numbers. It is, however, obvious that though it is possible that the blood may be constantly replenished with a greater or less

* Various forms of fission-fungi—*Schizomycetes*—found in the blood.

number of those organisms, yet they do not accumulate to any great extent therein; and it may be safely affirmed that their presence in appreciable numbers is, judging from experience, incompatible with a state of perfect health.

It must also be admitted that there are certain diseases in which *fission-fungi* are very generally, though not invariably, present, nor is their number coincident with the gravity of the malady. Thus one or other of the *fission-fungi* have been found in cases of *charbon* or *splenic fever*; in *relapsing* or *recurrent fever*; in *septicæmia*, and in the so-called *typhoid fever* of the pig. The subject has received great attention; and probably no subject of a scientific nature is being now more closely investigated; and rightly so, because the pathology and practice of medicine must be materially affected by the result.

The discovery of the yeast-plant by Schwann (1836) led to the subsequent recognition of low organisations in association with different kinds of fermentation; and twenty years later (1856) Pasteur announced as the result of his research, that low organisms acted as the invariable causes of fermentation and putrefaction—those changes being capable of initiation only by the agency of living particles. This seemed to give support to an ancient medical theory, that disease was propagated by means of a ferment or leaven—*Zymosis*; and which gave the name, as we have seen, to a class of diseases—*Zymotic*. But this *physico-chemical* theory, which held that the only requisite for inducing the fermentive process was contact with matter which was itself undergoing change, does not take sufficient account of vital action, which cannot be ignored in the pathology of disease.* Hence, Pasteur's *vital* theory recognises fermentation as essentially a correlative phenomenon of a *vital* act, beginning and ending with it—the result of a biological reaction; and that where there is fermentation there must also be organisation, development, and multiplication of the globules of the ferment itself. Thus we have many fermentations, all of them having one feature in common—namely, the reproduction of the ferment (C. T. KINGZETT, *Journ. Soc. of Arts*, March, 1878). The fact that certain diseases are communicable by inoculation, suggested the theory that in reality the phenomena of such diseases were the result of a ferment of some kind—a theory which was also assumed to be applicable to diseases not proven to be inoculable, but which appeared to be similar as regards their phenomena. Then the observations and experiments of Davaine, Birch-Hirschfeld, and Pollender, showing that in animals which had died of the diseases known as *malignant pustule*, *charbon*, *rot*, *splenic fever*, *splenic apoplexy*, or *anthracoid disease*, minute organisms were, to a greater or less degree, constantly present in their bodies—in the blood

* Certain organic compounds, when exposed to the action of air, water, and a certain temperature, undergo decomposition, consisting either in a slow combustion or oxidation by the surrounding air, or in a new arrangement of the elements of the compound in different proportions (often with assimilation of the elements of water), and the consequent formation of new products. The former process, that of slow combustion, is called *Eremacausis* or *Decay*; the latter is called *Putrefaction* or *Fermentation*,—*putrefaction* when it is accompanied by an offensive odour, *fermentation* when no such odour is evolved, and especially if the process results in the formation of useful products: thus, the decomposition of a dead body, or of a quantity of blood or urine, is putrefaction; that of grape-juice or malt-wort, which yields alcohol, is fermentation.—*Watt's Dictionary of Chemistry*, Vol. II., p. 624, 1872.

during life, where they were developed, and not in the spleen; that the disease was communicable with the food, mixed with the tissues of the diseased animal,—gave rise eventually to the doctrine of “disease germs,” a doctrine which ought to be held as distinct from that of a “*contagium vivum*.” An accomplished mycologist, Professor Cohn, having examined



Fig. 77.* $\times 600$ diameters.

these organisms, found them to be *bacilli*, and suggested the name of *Bacillus anthracis* (Fig. 77). Dr. Koch afterwards demonstrated (as he believed) the existence of “*resting spores*,”—spores which had the power of retaining their vitality for a long time, and of giving rise anew to *bacteridia*. In the morphology of vegetable growth, these so-called spores may be interpreted as a mode of multiplication by fission, the filaments dissolving away, and leaving divided portions in the form of the strongly refractive molecules, to which the name of “*resting spores*” has been given. Dr. J. Cossart Ewart confirmed Dr. Koch’s experiments in many points. “The spores,” he says, “divide by a process of division, and afterwards lengthen into rods;” and further, the experiments of Ewart prove that the so-called *Bacillus anthracis* is *not* absolutely motionless (a fact confirmed by A. Frisch also), but at certain stages manifests active movements; that such *bacilli* and their spores were killed after being boiled for only two minutes, the fluid after this treatment becoming absolutely inert. A pressure of twelve atmospheres of oxygen produced a like result—a fact confirmed by M. Felz (*Comptes Rendus*, 15th July, 1878). On the other hand, it has been shown by Mons. P. Bert, that compressed oxygen rapidly kills all living things; but that material in solution, such as scorpion venom, vaccine matter, whether liquid or dried, and re-dissolved, resist the action of compressed oxygen; and from other experiments he was led to infer that the active principle in vaccine and in glanders is not a living cell. He exposed splenic blood to the action of compressed oxygen: the blood retained its virulent properties intact, as proved by inoculation, but in no instance did the blood contain *bacilli*. MM. Jaillard and Laplat came to similar conclusions, namely:—(1.) That charbon is not a parasitic disease; (2.) that the presence of *bacteridia* is to be considered as an epiphenomenon, and not as a cause; and, (3.) that the fewer *bacteridia* the blood of *charbon* contains, the more virulent it is. Hence cases of *charbon* are described—some *with* and some *without* *bacteridia*; just as cases of relapsing fever are now described—some *with* and some *without* *spirilla*. While experiments prove that splenic fever is an inoculable disease, those of Brauell, Bouley, and Bollinger prove that the organisms found in the blood cannot be the carriers of the virus, seeing that the blood *not* containing *bacilli* had been found to propagate the disease; that the disease may exist without the presence of *bacilli* in the blood; that such blood will induce the disease in other animals; and that even under such circumstances, organisms may develop in the blood of the inoculated animal, and be detected during life as

* *Bacillus anthracis*, obtained after death in the blood of an ox which had died of splenic disease. (After COHN.)

well as after death (LEWIS, p. 55). Bert subsequently concluded that the blood in splenic fever contains a toxic and virulent principle, which resists the action of compressed oxygen, and can be isolated in the same manner as *diastase* or *pancreatine* (*Comptes Rendus*, lxxxiv., p. 1130, May, 1877).

Generally, it seems to me that the result of all those observations and experiments, from Davaine's downwards till now, is that the *bacilli* have nothing to do, even as a germ, with the propagation of those diseases; but that the isolation of such a toxic principle as that suggested by Bert, points rather to the generation, development, and multiplication of a *contagium* as the media of propagation, and that this *contagium* is the result of a *vital* morbid process—in short, a *contagium vivum*. The experiments of M. Onimus with dialysed blood also confirm this view, and that the poisonous material is an albuminoid substance not dialysable.* But as regards the propagation of *septicæmia* by *bacteria* as the active agent, it seems to stand in the same category as *charbon*, and with the *typhoid of the pig*, and with *recurrent fever*—i.e., in all of them the results of experiments are against the doctrine of a "germ" being the cause of

* "Blood from the ox or swine, or human blood from a patient suffering from typhoid fever, is placed in dialysis paper, and this paper is then placed in a vessel containing distilled water, and the whole submitted to a temperature of about 35°C. After fourteen hours the distilled water has become troubled to lactescence; and, examined by the microscope, is found to contain a prodigious quantity of bacteria and vibrios, identical in form with those contained in the blood inclosed in the dialysis paper. This being established, Onimus injected *one drop* of the putrefied blood contained in the dialysis paper into several rabbits; whilst several drops of the external fluid (in which were myriads of bacteria) were injected into other rabbits. All the animals which received a drop of putrefied blood died in a short time; all those which received the water with the bacteria survived. Similar experiments made with the (transmitted septicæmic) blood of those rabbits that died from the injection of the putrid blood he found to give similar results; hence he maintained, in opposition to Davaine, that the presence of *bacteria* is not essential to the result. Dr. Thin and Gesnaldo Clementi, under the advice and assistance of Professor Stricker, have repeated these experiments in the famous Institute for Experimental Pathology in Vienna. They frequently examined microscopically the blood of the affected animals at different stages of their illness, with the general result that *vibrios* and *bacteria* were not once seen in blood taken from the living animal, even when it was *in articulo mortis*, whilst they were found abundant in every specimen of putrefied blood. Their experiments showed a mortality in the animals inoculated with transmitted blood very much greater than those inoculated with putrefied blood (i.e., with non-bacteriferous blood as against bacteriferous); and that an approximative dose of one decimilligramme of transmitted (non-bacteriferous) blood was sufficient to kill a rabbit. Dr. Thin also makes the important observation that in these experiments transmitted blood to the twelfth generation was used, hence it became evident that the noxious *something* must have multiplied in the animals after inoculation. For, he says, even if we suppose that the blood alone, and not the other fluids of the animal's body, acted as a diluent of the dose injected, we would have by the time we came to the twelfth generation (of transmitted blood poisonings) fractions of a gramme in a dose that would have to be written with fifty to sixty cyphers. He considers that the noxious substance or substances present in putrefied blood must be designated *miasmata*, and those conveyed from one animal to another, and multiplied in the organism of the animal, as *contagia*. Hence Dr. Thin draws the conclusion from his experiments that a *miasm* has become transformed into a *contagium*, but as to the nature of either he expresses no opinion. Dr. Thin and Clementi also constructed a dialysis apparatus to test the experiments of Onimus; from which, although they could not prove that the poison itself is dialysable, for it was possible that putrefiable substances passed through the parchment and afterwards putrefied in the external fluid, they concluded that the inference of Onimus, that the poison is not dialysable, has no foundation; also that it is not destroyed by boiling" (*Edin. Med. Journal*, July, 1873).

the disease, and that the existence of the bacteria are as epiphenomena. All the representatives of the group can be introduced into the system with the greatest impunity,—(a.) by food; (b.) directly into the blood by injection into the veins; (c.) indirectly through the lymphatics in the subcutaneous tissue. And organisms in no respects different from Davaine's bacteridia have been shown by Lewis and Douglas Cunningham to exist in the tissues of *healthy* animals a few hours after death; while the conclusive experiments of T. R. Lewis clearly demonstrate that no sufficient grounds have been adduced for accepting the doctrine that *bacilli* have been found in the diseases mentioned which differ not only in any *material* respects, but in any respects whatsoever, from *bacilli* which may be found under certain easily induced conditions. With regard to the *spirillum* of recurrent fever, Heydenrich is compelled to own that sufficient reason has not been shown to warrant its being described as specifically different from the spirillum of water and the ordinary spirillum of the mouth; and Motschutkowsky states that he succeeded in inoculating persons with the blood of patients suffering from recurrent fever whether it contained *spirilla* or not (HEYDENRICH, quoted by LEWIS). The disappearance of bacteria from the blood immediately before or shortly after death, shows their occurrence to be epiphenomenal, and suggests that the initiatory changes occur in the blood itself favourable or the reverse for their existence; while the mere finding of such organisms in the blood of the diseases under consideration in no way proves any causal relationship between these diseases and bacteria. They are never to be detected in the early stages of the disease, but rather at brief periods before and after a fatal termination, concomitants of impending dissolution. It has also been shown that the virulence of septicinuous substances is not dependent on such organisms, by the experiments of Panum, Lewis, and Douglas Cunningham, but that septicinuous fluids, after filtration through porous porcelain, were still virulent; and therefore it has been justly concluded that a fluid which can retain its specific property after being filtered, boiled, evaporated to dryness, and the residue digested in cold and in boiling alcohol, then redissolved and again filtered, cannot owe this property to living *organisms* of any kind. Dr. W. B. Richardson has also shown that sero-sanguineous fluid from the peritoneal cavity of a person suffering from pyæmia, would communicate fatal disease from one animal to another in a direct series; and that the poison—to which he gives the name of "*septime*"—which effected this, could be made to combine with acids so as to form salts, which retained the poisonous qualities of the original substance (*Lancet*, 3rd April, 1875; and *Brit. Med. Journ.*, 27th May, 1876). Bergmann also succeeded in obtaining a similar poison, and named it "*sepsin*;" from all which results Dr. Lewis concludes that the living tissue elements of the body itself play a much more important part in the elaboration of septicinuous and allied poisons than what has been of late ordinarily ascribed to them.

Our knowledge regarding the third question—"How contagion is communicated"—is more satisfactory, as it is also of much practical importance. *Contagia* are known to be communicated from body to body directly and indirectly;—directly when they, or the natural fluids containing them, are, without the intervention of media, transferred from

the diseased to the healthy; as when vaccine lymph is taken from beneath the surface of one body, and inserted beneath the surface of another—a process known as *inoculation*;—indirectly, when the contagium-particles are transferred from the diseased to the sound through the intervention of media. In this process—

“The special and distinctive phenomena of each disease are usually attended with special implication of some part of the body, and it is especially these parts which contain the *contagia*. In these parts there is frequently rapid growth; and if the parts are on the surface, frequent detachment; in fact, those parts of the body which are the breeding-places of the contagious particles, give off the poison in greatest amount. The portions of the body thus thrown off, and containing the *contagia*, may then pass into air, or find their way into water or food, and in this way be introduced by breathing, drinking, or eating, or through broken surfaces of the body (PARKES). The evidence, showing that contagium-particles are carried by air and water, and received into the system by the stomach, air-passages, and wounds, is of the following kind:—That minute particles of matter are suspended in ordinary air is proved optically when a sunbeam penetrates a dark room; that a large proportion of these particles are combustible, and therefore organic, has been demonstrated recently by Professor Tyndall; while that many of these aerial particles are living germs, was many years ago noted by Pasteur. Dr. Maddox, also, by means of his apparatus for collecting from the atmosphere all foreign particles suspended in it, obtained from air pollen, bacteroid bodies, and microscopic germs, a few of which germinated under observation. Yet stronger proof that air is a medium through which contagium-particles are conveyed, is afforded by the following experiment, recorded by Küchenmeister. For one hour a sheep was made to breathe air which had traversed a shirt worn for twelve hours by a patient suffering from small-pox. Five days afterwards variola commenced in the sheep, and by the eighth day a well-marked eruption was developed. Indeed, if we admit that air readily floats innumerable portions of matter, which, inasmuch as they are visible, are fifty or a hundred times larger than *contagia*; and also that bodies, healthy and diseased, are always throwing off and attracting little particles, the conclusion almost necessarily follows, that air is a medium by which the contagiferous subject communicates infection to the healthy. The results of Küchenmeister's and other similar experiments prove that this is so;—that air is, in the immense majority of cases, the medium by which *contagia* spread. It must be remembered that gases, on the one hand, and the particulate ferments, on the other, stand in widely different relations to air and water as their respective media of diffusion. The ferments, so far as we know them, show no power of active diffusion in dry air; diffusing in it only as they are passively wafted, and then, probably, if the air be freely open, not carrying their vitality far; but as moisture is their normal medium, currents of humid air, as from sewers and drains, can doubtless lift them in their full effectiveness (SIMON). While, therefore, air, in the condition in which it usually obtains, more or less charged with moisture, is a medium by which many forms of *contagia* are communicated, it would appear to owe its property in this respect to the aqueous vapour it contains; and of all media taking part in the wide diffusion of disease-germs, and facilitating their introduction into man's organism, water, there is reason to think, is the most general, and, perhaps, with the exception of

air, the most effective. The evidence that water is *the* medium by which some of the specific *contagia* are spread—those of the entero-zymotic diseases especially—is so conclusive, it is impossible to doubt it. As illustrating the relations of polluted public water-supplies to the dissemination of cholera and enteric fever, reference may be made to the collection of cases in Mr. Simon's *Twelfth Report*, or to Appendix No. I. to his *Supplementary Report*, a tabular digest from Inspectors' Reports for the four years 1870-73, showing the circumstances in which enteric fever and epidemic diarrhoea are commonly found prevalent. That milk is one of the media by which certain *contagia* are spread, was first suggested by Dr. Ballard, as the result of an inquiry into the origin of an outbreak of typhoid at Islington in 1870; and since then it has happened to this gentleman to trace the same method of infection at work in two considerable outbreaks of the same fever—one at Armley in 1872, and one at Moseley and Balsall Heath in 1872-73. The still more recent report of Messrs. Radcliffe and Power on the Marylebone outbreak proves that this was also referable to the milk supplies. That other articles of diet, especially animal foods, are occasional media by which *contagia* are disseminated, rests on sufficient testimony (experimental and casual observation), food affording a nidus for the reception of particles from other media. Wearing apparel, bedding, furniture, paper-hangings, everything belonging to a sick room, and even books (especially of circulating libraries), and the instruments used by the medical attendant, are all efficient agents in the communication of contagious disease.

"In support of the proposition that there are three passages by which *contagium-particles* may reach the blood, there are three noteworthy experiments—two placed on record by M. Chauveau, and the third by Mr. Crookes. To prove the penetration of virus by the respiratory and digestive tracts, M. Chauveau perforated with a trocar the trachea of a sheep, and, by means of a valvular apparatus, made it inhale a small quantity of dry sheep-pox virus reduced to powder. The animal became affected in due course. The experimenter then mixed in a drench ten centigrammes of sheep-pox virus, and gave it to a healthy sheep to drink, with the result that he established a variola having all the characters of the disease caught spontaneously, and running a like course. M. Chauveau also obliged a horse to breathe air containing pulverised dry vaccine lymph, and gave another horse a dose of vaccine in a draught of water; and in each case horse-pox was produced. Mr. Crookes's experiment proves that *contagia* may be received into the system through a skin-wound. The air from a cowshed, in which there were several animals in the last stage of cattle-plague, was passed through a glass tube containing cotton-wool, and the wool inserted beneath the skin of a healthy calf, with the effect that the animal took the cattle-plague, and died of it in a few days."

With regard therefore to the causes of the specific or general diseases, the weight of evidence adduced is decidedly in favour of the belief that each of them is produced by some specific morbid agent—some morbid poison—which is capable (as regards some of them) of being generated, living, and multiplying in the body during the progress of the particular disease. In this respect they are capable of self-augmentation (PAGET). No evident fresh cause is applied, and yet the infectious elements of disease increase (e.g., *syphilis*, *small-pox*, *vaccinia*, *glanders*, *hydrophobia*, and *malignant pustule*). The theory of each of them, expressed in

the most general terms, is, that each of them depends upon a definite specific virus, which induces a morbid condition of the blood and juices; and that during the development and course of the disease, the system endeavours to discharge or transform in some way the peculiar morbid agents which have given rise to the symptoms, or which have multiplied in the body during the course of the affection. The whole blood thus seems to be diseased, and nearly every function and sensation in the frame is impaired or disturbed from the state of health before any local lesion is developed. Sometimes, indeed, the severest constitutional disturbances of a specific kind may co-exist with the smallest local development of any specific lesion (PAGER); and Dr. Robert Williams has justly observed, and numerous examples have been noticed, in which "it may be laid down as a general law, that when a morbid poison acts with its greatest intensity, and produces its severest forms of disease, fewer traces of organic alterations of structure will be found than when the disorder has been of a milder character." Time, duration, or *chronicity*, is a peculiarly important and characteristic element in the nature of these diseases. They run a definite course; and we know of no specific remedy which will at once effect a cure and prove an antidote to the poison. The nearest approach to an antidote is that of quinine in the malarious fevers. They have all—(1.) A more or less defined period of incubation or latency, best determined in those which are inoculable; (2.) A period of development towards the *fastigium* or acmé of the disease; (3.) A period of *defervescence*, during which the febrile phenomena abate; (4.) Etiologically they are quite distinct from one another; and, lastly, they have a period of convalescence. The causes, then, of A, B, C, D, &c. diseases, being thus specifically different, it still remains to be determined what these causes really are. This is now being done by the principle of exclusion chiefly—i.e., that such and such an alleged cause cannot produce such and such effects. Thus, it is certain that cold, a chill, or sudden change of temperature, never cause a *specific* disease such as any of those mentioned in this chapter, and which are to be described; that mere moisture in the air does not; that such and such gases do not; that such and such *bacteria*, *bacilli*, or *spirilla* do not; and so on. The question seems now narrowed to this point,—that in the case of many specific diseases (probably in all) the cause is something quite special, and, in fact, as yet of unknown origin as regards the majority of them; and it is quite a matter of inference that the different *contagia* by which they severally are communicated have respective peculiarities of their own—peculiarities which are primarily governed by the nature and anatomical relations of the morbid process in which each particular *contagium* originates, lives, and multiplies. All of them are essentially unstable and transitory; but, while some of them tend, under ordinary circumstances, to undergo a rapid extinction, others of them can with comparative ease retain their power for long periods of time, and some apparently have not their full force till some time after they have left the diseased body—e.g., *cholera* and *enteric fever* evacuations. Some of them associate themselves indistinguishably with one or more of the common excretions and exhalations of the body, others are separately tangible in vesicles and pustules, or at ulcerating or suppurating

surfaces, and may or may not also exist in other products of the body, such being usually inoculable; some of them are evolved in small quantity, others in very large quantity, or with very large natural admixture; some of them are fixed, others but very scantily volatile, others as volatile as if they were vapours; some of them operate easily on a second body by mere contact (more or less prolonged) with the outer or inner surface to which they are applied, others are not found to act unless they come into contact with accidentally abraded surfaces, or be thrust into the bodily substance by inoculation. Thus, in *vaccine lymph* and in the matter of *chancres* respectively, there is a *contagium* which we know only in a fixed form, and only as communicable by intentional or accidental inoculation. Also *ophthalmia* and *gonorrhœa* and *glanders* are communicable by the fixed *contagium* which their pus contains; but this *contagium* does not need inoculation to infect the mucous membrane to which it is applied; and as regards *ophthalmia*, there are reasons for suspecting that to some extent the contagious pus may retain its activity when dry enough to float as dust in the air. In some forms of *milzbrand* (including, probably, the so-called '*malignant pustule*,' which is the best-known human form of the disease) the highly virulent fluids can, it is alleged, infect by soakage through the cuticle. In *diphtheria* the characteristic exudation is capable of infecting by contact; and though often the disease is communicated from person to person without any manifest transplantation of matter, it may be that in such cases particles of the decomposed false membrane are conveyed as a volatile *contagium*. *Cholera* and *typhoid fever* send forth their respective *contagia* for the most part, if not exclusively, as matter dissolved or suspended in the evacuations which pass from the patient's bowels; and probably these evacuations (which, at least in cholera, gradually develop their full infective force after their discharge from the body) can, under some circumstances, bring into similar contagious fermentation the excrement with which they are mingled in privies, drains, and cesspools, and can thus convert the effluvia and leakage from such sources into means of extensive secondary infection of air and water. The theory of contagion just noticed, as expounded by Dr. B. W. Richardson, might, to some extent, explain this process, inasmuch as he has shown that an organic poison from the peritoneal cavity could be made to combine with acids so as to form salts, which retained the poisonous properties of the original substance. The volatile *contagium* of *hooping-cough* is probably disengaged in large quantities by the air-passages, and, as it forms, is sent forth with the breath. In *typhus*, *small-pox*, *measles*, and *scarlatina*, the diffusion of volatile *contagia* occurs to a vast amount, probably with all exhalations from the body; and, in addition to this, *contagia*, more or less fixed, collect abundantly about the patient's person and bedding; and, in a far less degree, something of the same sort probably occurs in *erysipelas*" (SIMON, l. c.) As regards the spread or modes of propagation of these diseases, each of them "has its own laws of communicability,—laws which must be properly understood if the danger of contagion is to be guarded against. The communication of some diseases (of *scabies*, for instance, and *favus*) is not by any true product of the human body, but consists in the migration of *parasites*, or *germs of parasites*, animal or vegetable, from one person's

body to another,—a migration which, of course, the recipient may to any extent facilitate by dirty personal habits, and which, as regards some parasitic diseases, can scarcely be conceived to occur otherwise than in consequence of such habits" (SIMON).

In the Section A of the General Diseases to be presently described, *the essential constituent of the specific morbid agent enters the body from without*; while in the second Section, B, *the essential constituent of the morbid agents appears to be inbred in the body, and makes itself manifest by various constitutional indications previous to the development of local lesions, or the characteristic expression of the disease by other phenomena.*

There is sufficient circumstantial evidence with regard to all of these diseases, and absolute proof with regard to some, that there is—(1.) A morbid condition of the blood and fluids; (2.) That the nature of that condition is, in many, definite and specific, inasmuch as it may be produced at will by the introduction of a definite substance into the blood, which then manifests itself by establishing a local disease, and which, within certain limits, has constant characters; (3.) That the morbid matter or poison by which the condition of the blood is changed may accumulate or augment in quantity and virulence, and at length may be discharged in various ways from the body, and under a variety of organic forms and fluid solutions, chiefly through excretory products. But some are also discharged at the seats of local lesions which are set up—the morbid poisons being for the time accumulated in the morbid structures.

As far as some of the *specific* or *miasmatic diseases* are concerned, such as *typhus* and *typhoid fevers*, *erysipelas*, *scarlet fever*, *cholera*, and the like, there is undoubted evidence of a disturbing morbid material in the blood, although it has never been isolated nor proved to exist in the products of the local morbid processes or specific lesions. In many of the *constitutional diseases*, too, similar evidence exists of a morbid matter *inbred* in the blood. It will soon appear obvious to the student that the diseases about to be considered differ very materially in their nature from the purely *local diseases* to be noticed lastly. While the LOCAL DISEASES may be regarded as *common* or *simple* diseases, those GENERAL DISEASES, first to be described, are so very distinct from them, and are possessed of such constant features by which they may be distinguished from the local, common, or simple diseases, that they are described under the term "SPECIFIC."

Certain types of local lesions, however, are common to both; and therefore the SPECIFIC DISEASES have some constant and characteristic modification, or something in addition which distinguishes them. These additional elements appear to consist—(1.) In a certain constancy and regularity of development, metamorphosis, duration, and decline, during which certain common morbid processes become manifest and modified according to the *special* nature of the disease. "In some the most evident specific characteristics are peculiar affections of the movement of the blood, as in the *cutaneous exanthemata*; in some, affections of certain parts of the nervous centres, as in *tetanus*, *hydrophobia*, *whooping-cough*; in some, peculiar exudations from the blood, as in the *inoculable diseases*; in some, peculiar structures formed by the exuded materials, as in *variola*, *vaccinia*, and other *cutaneous pustular eruptions*; in some, destruction of tissues, as in the *ulcers of syphilis*, the *gangrene of ergotism*, and the *sloughs of boils and carbuncles*; in some,

peculiar growths, as in *cancers*; in some, or indeed in nearly all, peculiar methods of febrile general disturbance" (PAGET). In the specific diseases the phenomena of these local and general morbid processes are concurrent. (2.) The most striking feature (assuming specific diseases to be due to the presence of specific poisons in the blood, which, by a morbid process, is again separated from it, and eliminated from the system during the progress of cure) is, that "the whole blood for a time seems diseased, and nearly every function and sensation is more or less disturbed from its health. The patient feels 'ill all over' before the local disease appears" (PAGET). In the common or local diseases, on the other hand, the local phenomena precede the general or constitutional disturbance. (3.) There exists in the SPECIFIC DISEASES an apparent want of proportion between the cause and the effect. Thus, in *small-pox*, *measles*, *hydrophobia*, *bites of poisonous serpents*, or *syphilis*, the severity of the disease does not bear any proportion to the amount of poison applied; and numerous diseases have been described in which the morbid poison appeared to act with so much intensity, and produced such severe forms of disease, that the patient died before local lesions had time to become developed—e.g., in *typhus fever*, *specific yellow fever*, *paludal fevers*, *scarlatina*, and the like.

From what has been here written regarding the acute specific diseases, it is clear that as regards their etiology, much has yet to be proved; and that the work to be performed is great and difficult, "before the opinions entertained on the subject can be converted into knowledge." There is hardly any one of those diseases, however common, "respecting which it can be said that its etiology is quite determined; and no report of a case can be clinically complete, the etiology of which is not given. There is no disease respecting which it can be said that the symptoms which mark its very outset, the symptoms which mark its decline, the symptoms which enable us to tell at the outset what will be its course, the effects which follow after the disease has apparently ended, and the influence which pre-existing conditions of health and disease, as well as that which drugs and other therapeutic agents exert on its progress, termination, and sequelæ, are perfectly known. Regarding all these points in every disease, even the most common, there are undetermined questions. Many of those admit of answer; many of them ought to be answered; and that many of them are not answered is, I think, discreditable to us as a profession. Medicine has made great progress—few sciences have made more; but still in regard to every disease, in reference to every one of those points mentioned, we are wanting facts—facts which could and ought to be supplied" (Sir WILLIAM JENNER, l. c.)

CHAPTER II.

THE PHYSIOLOGICAL MODES IN WHICH POISONS OR DRUGS ACT ILLUSTRATE BY ANALOGY THE ACTION OF THE MORBID POISONS OF SPECIFIC DISEASE.

If the reader will now consider the following statements as to the modes in which poisons act physiologically, he will be prepared to appreciate the effects of those conditions which, like poisons, induce diseases of the kind termed *specific* or *miasmatic*. The actions of poisons are subject to certain general laws,—the most important of which are, *first*, that they have all certain definite and specific actions; *second*, that they all lie latent in the system a certain but varying period of time before those actions are set up; and, *third*, that the phenomena resulting from their action vary, in some degree, according to the dose and to the receptivity of the patient. These laws are common to all poisons, but some are peculiar to individual poisons or classes of poisons; and it may be of advantage to notice a few of them. The *first* law, or that of the definite and specific actions of the poisons cannot be doubted; for, if it be supposed that agents acting on the human body do not produce their effects according to certain definite laws, we can neither determine the seat nor the course of any disease, nor direct nor judge of the operation of remedies. No one, for instance, has seen *castor-oil* produce tetanus, or *colchicum* intoxicate the brain, or *opium* inflame the spleen. The physician perfectly well knows that the first of these substances acts on the intestines, the second on the ligaments, and the third on the nervous system generally. The action of poisons, therefore, is not accidental, but determined by certain definite laws. But the action of poisons or drugs, though definite, is variously limited. Some, for instance, act on the cells composing one structure, or one organ, or one system of organs; while other poisons extend their action over two or more membranes, or organs, or systems of organs, or even over the whole animal frame; and, conversely, the cells composing one structure take up and are acted on by some drugs or poisons, and not at all by others; while other structures are much affected by the very substances which had so little action on the first (Dr. T. L. BRUNTON, in *Brit. Med. Journ.*, April, 1877, p. 41). We have examples in *aloes* and *jalap* of substances that act mainly upon the mucous membrane of the alimentary canal. In *digitalis* we have an instance of a medicine that principally acts on the heart, greatly reducing or even stopping its action; while *strychnine* acts on the parts supplied by the spinal cord, producing powerful and sometimes fatal tetanic action of every voluntary muscle in the body. It is seldom, however, that the action of poisons is limited to one membrane, organ, or system of organs. The greater number of these noxious agents more usually act on two or more membranes, organs, or systems of organs. *Elatarium*, for instance, acts on the mucous membrane of the intestinal canal, and on the kidneys. *Tobacco* nauseates

the stomach, intoxicates the brain, and affects the action of the heart. *Antimony* has an equally extensive range: it induces cutaneous perspiration, acts cathartically and emetically, and in large doses appears to cause gangrene of the lungs. *Alcohol* and *opium* are examples of substances acting still more generally, affecting not only the action or secretion of every organ or tissue of the body, but even in some instances altering their structure. Thus alcohol, in its most limited action, has been shown to cause structural disease of the liver, of the stomach, and of the coats of the arteries. From the circumstance of these and other substances acting so extensively, they resemble in their specific effects those of many so-called *morbid poisons*, as that of typhus fever, of scarlet fever, of small-pox, or of syphilis. Another important law is, that poisons lie *latent in the system* for a period of time, which varies in different individuals, before they set up their specific actions. *Rhubarb*, for instance, produces no immediate result, but lies dormant in the system six or eight hours before its action is apparent on the bowels; *opium*, in the usual dose, is generally thirty minutes before it subdues the brain. The convulsions from *strychnine* do not follow till twenty minutes after its administration; and perhaps every substance has a more or less definite period of latency. When a medicine acts on more parts than one, a considerable space of time may elapse after it has affected one organ before it affects another; thus, *digitalis* frequently occasions emesis before it acts on the heart, and the action of *mercury* on the bowels is frequently sensible for many weeks before the gums and salivary glands are affected. The doctrine of the latency of poisons is indeed so generally admitted, that the actual period has been a point on which the condemnation or acquittal of a prisoner tried for murder has turned in our courts of justice, when certain poisons have been supposed to have been given. A *third* great law is, that the effects of poisons are modified by the dose, the temperament, and the existing state of the constitution, mentally and bodily, of the recipient. The effect of the dose in modifying the pathological phenomena of disease may be exemplified in the actions of *oxalic acid* and of *arsenic*. The specific action of *oxalic acid* is to inflame the mucous membrane of the stomach; but to insure this effect the dose must be limited so that this poison may lie in the system many hours. On the contrary, if the dose be excessive, and rapidly absorbed, the poison so disorders all the functions of the three great nervous centres, that life is destroyed in a few minutes. *Arsenic*, likewise, is a poison which inflames and ulcerates the mucous membrane of the alimentary canal, but it requires some hours to set up its specific actions; for, when the dose is large, it, in like manner, destroys by general irritation, and before traces of morbid change of structure can be appreciated after death. It follows from this law, that the larger the dose, or the greater the intensity of the poison, the more rapid its action, and the less the probability of finding any trace of specific lesion induced by it.

In studying the effects of dose on the constitution, we find some poisons are absorbed and are *cumulative*; while others are not absorbed into the system, or they are so rapidly removed that no cumulative effect is produced. Thus, in persons predisposed to the effects of *digitalis*, a dose so small as to produce no sensible effect whatever, will, if frequently repeated, at last destroy the heart's action. This cumulative property of

poisons, however, is by no means universal. There is no instance of *jalap* or of *castor-oil* proving cumulative. That the habitual ingestion of decomposing matter in the water used as drink is cumulative and capable of inducing conditions favourable to the development of specific diseases, admits of no doubt. Cogent instances of this are to be found recorded in the bitter experience of epidemics of cholera.

Temperament also is a circumstance which greatly influences the action of poisons. There are a few persons—rare exceptions—altogether insensible to the action of *mercury*, so that no quantity will affect their gums, or increase the secretion of the salivary glands. There are others, in like manner, the action of whose heart no quantity of *digitalis* will control. On the contrary, there are some constitutions—and these not so rare—so morbidly susceptible of these remedies, that it is scarcely possible to administer even a fractional dose of these drugs without giving rise to their specific effects. Besides natural temperament, *habit*, which may be termed an artificial temperament, has a powerful influence in reconciling us to particular classes of poisons, and of making them even sources of enjoyment. Thus *tobacco*, *alcohol*, *opium*, are all substances which are productive, in the first instance, to many persons of great discomfort; but by frequent repetition they cease to have any unpleasant effects, and their stimulus at length becomes a necessary indulgence. Still there are many poisons to which no repetition can habituate us. On the contrary, each repetition only the more debilitates the constitution, and renders it more susceptible of the action of the poison. A peculiar existing state of the constitution has also a powerful influence on the action of poisons; and it would seem proved, with some exceptions, that these agents act with an intensity proportioned to the debilitated state of the patient. There is indeed no duty more imperative on the physician than that of adjusting the dose to the strength of the patient; and nothing is more common than to forbear administering a medicine because the patient's strength will not admit of it. As a general principle, therefore, medicines or poisons may be said to act with a power proportionate to the debility of the patient. Still there are states of disease which render the constitution of the patient, though greatly debilitated, insusceptible to the action of even powerful remedies. Thus, in *typhus fever* the patient will often bear a considerable quantity of alcoholic stimulant without being affected by it. The experiments of Majendie may be referred to as affording many curious proofs of the state of the constitution in accelerating or retarding the action of poisons. He has shown that if a poison be introduced into the system, of such potency as usually will destroy life in two minutes, on bleeding the animal the same result will follow in half a minute, or in one-fourth of the time; and this experiment has often been repeated. Majendie also brought to light the curious fact, that if, after having poisoned the animal, and even after the poison has begun to act, we inject an aqueous fluid into its veins in such quantity as to cause an artificial plethora, as long as this artificial plethora can be maintained the action of the poison is superseded. No sooner, however, does the plethora cease, from the general effusion of fluid into every cavity of the body, than the poison acts in the usual time, and with

even perhaps more than its accustomed severity. Mr. Hunter thought that no two poisons could *co-exist* in the same system together, or that, co-existing they could not set up their specific actions at the same time. This hypothesis, however, is unquestionably erroneous; for we constantly see *opium* and *digitalis*, *jalap* and *mercury*, as well as many other combinations of medicines, producing their respective effects in the same system, and at the same time. There is no truth better established in medicine than that a combination of Epsom salts and *senna* produces a much more efficient action than the administration of either remedy separately; and *opium* is an agent possessing a modifying or controlling power over every organ or tissue, without which it would be impossible, on many occasions, to reconcile the system to the introduction of many necessary and essential remedies. Poisons, therefore, are capable of co-existing together, and of so influencing the system that they reciprocally accelerate or retard each other's actions. The co-existence of two or more specific diseases has been already noticed.

The general laws observable in the actions of medicinal substances are, for the most part, precisely similar to those which govern morbid poisons, or only differ in a few minor points; for these latter poisons have their specific actions and their periods of latency, while their phenomena are not less variable, although the conditions of their varied actions are not yet clearly determined.

Morbid, like other poisons, have their periods of latency, or incubation—*i.e.*, “the time which elapses between the poison of an infectious disease entering the system and the first manifestation of symptoms” (MURCHISON); and, generally speaking, a much longer time elapses before their specific actions come into operation than with medicinal substances. A knowledge of this incubation-period is of great practical importance; but is still vague and unsatisfactory as regards most of the infectious diseases. The results of Dr. Murchison's observations on the incubation-period of some of these diseases, especially of *scarlet fever*, are to the following effect:—(1.) In none of the infectious diseases does the period during which the poison incubates appear to be fixed. Even in small-pox the latent period is not “limited with precision.” It is usually assumed to be twelve days when the poison enters the system in the ordinary way, and only seven days when the poison is introduced by inoculation. Of three cases carefully observed, in two it was thirteen days, and in one eleven days, thus confirming the ordinarily accepted view that the incubation-period of *small-pox* is about twelve days. But that the incubation-period is far from being fixed has been shown especially by the late Dr. Otto Obermeier. Of eighteen cases in which the actual moment of infection was determined, the periods varied from five to thirteen days. (2.) In one case of *varicella* the incubation-period was eleven days. Thomas makes it from thirteen to seventeen days; and Trousseau from fifteen to twenty-seven days. Of ten cases noted by Dr. W. Squire the period was from ten to twelve days. When produced by inoculating the contents of the varicella vesicle, as Professor Steener of Prague did in ten cases (eight of which were successful) the period of incubation was eight days. (3.) The incubation-period of *measles* is usually about ten

or twelve days, and when introduced by inoculation only seven days. In a group of cases noted by Dr. Bristowe the period was from twelve to fourteen days. (4.) Of *hooping-cough* little is known as to the incubation-period; according to Squire it is usually about a week; to Bristowe probably about a fortnight, the disease being contagious at its very commencement. (5.) In *typhus fever* there is no fixed period of incubation. Dr. Murchison's conclusions from thirty-one cases are—1. That the period varies in different cases. 2. That in a large proportion of cases it is about twelve days. 3. That in exceptional cases it is longer than twelve days; but it rarely, if ever, exceeds three weeks. 4. That in many cases (one-third or more) it is less than twelve days; and occasionally there is scarcely any latent period, the symptoms commencing almost the instant of exposure to the poison. (6.) *Enteric fever*.—The difficulties of obtaining evidence regarding the *incubation-period* of this fever are stated to be,—1. In deciding when an attack of enteric fever really commences; 2. That in fever hospitals nurses and patients rarely take enteric fever; 3. That in private practice, when the disease has been imported into a healthy locality it very rarely spreads; 4. That there is often a difficulty in determining when a person is seized with enteric fever soon after changing his residence, whether he has brought the disease with him, or whether he has merely been predisposed to the disease by recent arrival in an infected locality. But, from facts collected by Dr. Murchison, he has come to the following conclusions respecting the incubation-period of *enteric fever*, namely:—1. It is most commonly about two weeks. 2. Instances of a longer duration appear to be more common than in typhus or in relapsing fever; but it is very doubtful if the incubation-period ever much exceeds three weeks. 3. It is often less than two weeks, and may not exceed one or two days. (7.) With regard to *relapsing fever*—1. The period is not fixed, and is even more variable than that of *typhus*; 2. It is on the whole shorter than that of *typhus*. In nine of twelve cases it did not exceed nine days; in none of thirty-two cases was there reason to believe that it exceeded sixteen days; in only four did it certainly exceed twelve days, and in only four others was it possible for this period to have been exceeded. In thirteen of thirty-two cases it did not exceed five days. 3. Occasionally, as in *typhus*, there is no latent period, the symptoms commencing almost immediately after the first exposure to the poison. 4. In cases of successful inoculation in healthy persons, the period of incubation never was less than five nor more than eight days. (8.) In *Rötheln*, or *German measles*, the period of incubation is variously stated at ten to fourteen days (SQUIRE); two to three weeks (THOMAS). (9.) In *mumps* the incubation-period has been fixed at from eight to twenty-two days (SQUIRE).

In none of these diseases is there anything like a fixed period of incubation; but for practical purposes the specific diseases may be divided into two groups, in one of which the incubation-period is long—from one to three weeks, or more commonly from ten to fourteen days, as in the above examples; and in the other it is short—from one to three or four days—rarely longer than a week, as in,—(10.) *Erysipelas*, the incubation of which never exceeds a week, and as a rule is from one to three or four days. (11.) *Diphtheria*, which has been stated positively to occupy from

two to five days (OERTELS); and that in twelve to twenty-four hours after artificial inoculation upon the surface of wounds, a greyish-white discoloration, a dirty greyish layer, and other signs of infection appear. (12.) *Dengue* has a latent period variously estimated at from one to seven days, but there have been many cases in which it has lasted only a few hours. The rule is about five days. (Dr. CHARLES, in Calcutta.) (13.) *Scarlet fever* has been variously stated to have a period of incubation of from a few hours to a month, no attempt having been made to collect a large number of observations. Dr. Murchison has now collected all the cases of scarlet fever that have any bearing on the period of incubation during the last twenty years (seventy-five in number). In some the incubation-period was less than twenty-four hours, in none did it exceed six days; and, when a large number of persons became infected from a common source (as at a dinner-party, and on board ship), the incubation-period was in every instance short. In three of the cases the moment of infection could be fixed with precision—namely, thirty-six hours, three days, and four and a-half days respectively. In seventy-three of the cases the period of incubation could not have exceeded five days; in fifty-four it could not have exceeded four days; in twenty it could not have exceeded three days; in sixteen it could not have exceeded two days; and in three it could not have exceeded twenty-four hours. The longest period made out was four and a half days, and in only two of the cases was it as long as four days. The period is not longer in adults than it is in children. It is essentially a short period of incubation, and a person who has been exposed to *scarlet fever*, and does not sicken after a week's quarantine, may be pronounced safe. The poison of *malarious fevers* may lie dormant in the system for many weeks; and that of *hydrophobia* may not manifest itself for years. These are examples of periods of latency far beyond anything that has been observed in the action of medicinal substances; and *syphilis*, in its remote effects upon the organs and the constitution generally, is still more remarkable (MURCHISON).

It occasionally happens that morbid poisons which usually act on a plurality of membranes, exhaust their influence on one or more without affecting others. In *scarlatina simplex* the poison sometimes exhausts itself entirely on the skin, without affecting either the mucous or serous membranes of the body. The *rubeola sine catarrho* is a similar example of the poison exhausting itself on the skin. In *intermittent fever*, when the dose of the poison is limited, and the disease properly treated, it is seldom that a lesion occurs in any organ or tissue; yet, left to run a slow course, with constant exposure to the morbid influence, scarcely any organ or tissue would escape being affected, or having its function impaired. Sometimes when the morbid poison acts on many membranes, the usual order of attack is inverted. In *scarlet fever* the affection of the skin may precede that of the throat, or the reverse may take place.

It has been seen that the period of latency of medicinal substances having passed over, the effects vary in a considerable degree according to the dose, temperament, or existing state of the constitution of the patient. With respect to the dose of a morbid poison, we rarely possess any direct measure of its strength. The *paludal poison* of tropical climates to which

malarious fevers are due, unquestionably greatly exceeds in intensity that of more temperate climates, and its effects are proportionally marked. Thus, in the West Indies the severe remittent fevers occur with hardly a trace of organic lesion after death, so rapid is their course; in Holland, a paludal fever of less severity exists, but it is followed by enlarged livers or spleens, or by dropsy; while, in this country, the same fever is comparatively mild, and, if properly treated, for the most part terminates without any visceral affection. With respect to the influence of temperament in modifying disease, *small-pox* offers very striking instances. Different persons inoculated or poisoned from the same source have suffered in every degree from this formidable malady—from the *horn*, the *distinct*, the *confluent*, and the *bloody small-pox*; while, in the worst cases, children have died in the primary fever, and even before the specific action on the skin had time to be developed. It may therefore be laid down as a general law, that the more intense the dose of the morbid poison, the more severe the form and rapid the course of disease; and that fewer traces of organic alteration will be found after death when the poison is severe and abundant, than when the poison, or the disorder it produces, has been of a milder character, and the course of the disease more prolonged. Thus, enlarged livers, disorganised spleens, and dropsy, marked every case that died of the so-called Walcheren fever; while in the West Indian and African fevers of the same period (1799 to 1809), though resulting from the same poison, scarcely a trace of disease was to be found.

The existing state of the constitution also influences the event. Thus, persons of a good constitution, but ignorant of their danger, are often seen to pass through a mild form of typhus fever, while the nurses and others contaminated by the same poison, but more alive to their critical state, have sunk in a short time. A presentiment of death is a very unfavourable circumstance in the progress of *remittent* fever, especially in tropical climates. A soldier will sometimes say to the medical officer, "You have been very kind to me, sir; but this time I shall not get over it." There may be no appearance of absolute or immediate danger at the time—yet the man generally dies (SIR RANALD MARTIN). As a principle, therefore, it may be stated that morbid poisons act with an intensity proportioned to the enfeebled or depressed state of the constitution; but this law is not universal. Want of a sufficient amount of food is most powerful among the conditions which predispose or help to bring about specific diseases, and most constant in operation. It is a popular belief that the lowering of all the vital forces by deficiency of food constitutes the particular condition which renders a starved population so peculiarly open to the invasion of these diseases; but it is also a curious phenomenon of starvation that a state of general putrescence supervenes during life, as if the want of material for the generation of new tissue were an obstacle to the deportation of that which has become effete (CARPENTER). The hardy mountaineer is a surer victim, whether *he visits* the low countries of the tropics or the marshes of a more temperate climate, than *the feeble natives* of those countries. The immunity the latter enjoys is probably owing to his habit of living in the noxious atmosphere; for let him remove to a more healthy climate, and then return to those regions of pestilence, and he will be found as

susceptible of the poison as the hardier stranger. As to the influence of pre-existing individual constitutions in regard of susceptibility to contagious poisons, reference may be made to the well-known fact of the intimate relation between the state of the system left after *measles*, and the susceptibility to the poison of *hooping-cough*. The individual who has first suffered from one of these diseases is prone, in a remarkable degree, to take the other. That is to say, after the occurrence in an individual of one or other of these ailments, a very small dose of the poison of the other disease is sufficient to produce the development of that disease (Sir WILLIAM JENNER). The influence also of a previous residence in a malarious region, in deteriorating health and increasing susceptibility to disease, was clearly shown by the influence of the previous residence in Bulgaria on the health of our troops in the Crimea during the Russian War of 1853-1855, (AITKEN, *Medico-Chirurgical Transactions*, Vol. XL.)

The principal points in which the effects of poisons which induce specific diseases agree with those of poisons generally having been stated, it will now be necessary to state those circumstances in which they principally differ. Many medicinal poisons have the property of accumulating in the system, and acting with an intensity proportioned, not to the last dose, but to the aggregate of the whole quantity that has been administered. Thus, the last few minims of *digitalis* may stop the action of the heart, or the last few grains of *mercury* salivate the patient, or the last minute dose of *strychnine* become fatal. There is, however, no well-authenticated fact which can be arranged under this law in the whole circle of morbid poisons, except, perhaps, the cumulative and persistent pernicious action of *paludal malaria*. The actual quantity required to establish disease, according to the experiments of Dr. Fordyce, is probably extremely small. That physician, in the hopes of mitigating the small-pox, inoculated with virus greatly diluted; and although the disease was not always produced, yet when produced, it assumed every form, character, and degree of severity that small-pox has ever been known to assume. In accordance with Burdon Sanderson's experiments, such dilution tends to separate and diffuse more widely apart the living *contagia*, which are the efficient carriers of the poison; hence the greater the dilution or diffusion, the more likely one would be to introduce a portion of the fluid destitute of the specific *contagia*, and so fail to inoculate the poison.

The puerperal female is not only highly susceptible of specific disease poisons, but she is proved to favour their further development; and forms of puerperal fever seem capable of generation by *materies morbi* of a kind other than that which might be considered peculiar to it. It is a well-known fact, unhappily not of rare occurrence, that a medical practitioner or a nurse from a case of puerperal fever going to attend on other cases of labour, the chances are that these will be attacked with the disease. I was recently called in consultation to see a lady within a few hours of her death from puerperal fever. I found the nurse in charge of the patient had been in attendance, four months before, on a case of puerperal fever—the wife of a medical man—which terminated fatally. The nurse had not attended any case between the two. Both cases died under similar morbid conditions. Further, the practitioner or nurse may go to cases of labour from attendance on cases of *scarlatina*, *typhus*, *erysipelas*, or

small-pox, and the parturient patients may then become the victims of puerperal fever. In the Vienna Lying-in Hospital, it is on record that a mortality of 400 to 500 in an average of 3,000 deliveries per annum appeared traceable to the introduction of cadaveric matters, through the uncleanness of the attending students, these matters being especially potent when derived from the bodies of those who died from the adynamic forms of specific disease. Students of practical midwifery should bear in mind this fact. They ought not to attend cases of labour while they are engaged with practical anatomy in the dissecting-room; and no nurse ought to be allowed to go from a puerperal fever case to attend other cases of labour without destroying all clothes she has had in use in attendance on the fever case, and otherwise purifying herself.

Another peculiar law of morbid poisons, and one wholly unknown in medicinal substances, is the faculty which the human body, during the progress of the specific disease, possesses of generating to an immense extent a poison of the same nature as that by which the disease was originally produced. A quantity of *small-pox* matter, not so big as a pin's head, will produce many thousand pustules, each containing infinitely as much of the specific pestilential matter as was originally inserted; and, moreover, the blood and all the secretions of the body are equally infected with the specific poison of the pustules. The miasmata from one child labouring under *hooping-cough* are sufficient to infect a whole city.

There is still perhaps a more remarkable law of morbid poisons, which is, that many of them possess the extraordinary property of exhausting the constitution of all susceptibility to a second action of the same poison. This is the case with *syphilis*, *scarlatina*, *measles*, *typhus fever*, *small-pox*, *hooping-cough*, and, indeed, with a considerable number of others. But with regard to *enteric fever*, *diphtheria*, and *erysipelas*—three of the specific diseases which are the least contagious—they are considered more liable than the most contagious, such as *small-pox*, *measles*, and *scarlet fever*, to recur in the same individual (Sir WILLIAM JENNER). Still, it would seem that a temporary protective influence is imparted by most morbid poisons, for it is certain that few persons suffer a second attack of the same specific epidemic disease; and, consequently, it follows that the previous action of the poison must for a time impair the susceptibility of the constitution to its attacks. This beneficent law is of great importance in social life; it enables those who have recovered to attend on those who are sick; and allows a mother fearlessly to nurse her child in a dangerous and contagious distemper she has herself passed through, if such an inducement is ever necessary to strengthen the moral courage of a mother. The great problem involved in the pathology of those diseases which commonly occur but once in a life, is expressed in the question, "What is that change in the living body which gives exemption from a certain disease for the remainder of life, when the individual has once gone through it?" Or, stating the question still more specifically, "In what physical conditions does A, having had *small-pox* or been vaccinated, differ from himself before such protection was given, or from B, who has never obtained it?" The question thus put by Sir Henry Holland, is yet unsolved, and includes within itself many collateral problems the object of future research, subjects of deep interest, and as obscure as interesting.

All the diseases thus characterised are *contagious* in kind. The processes of *life* is a material element in the question; and the body, or its susceptible parts, having entered into a new state during the period of life under the morbid condition—a state not the same as its previous one—no longer presents the same reaction to the original *contagium* or poison. The exact nature of this resulting new condition is unknown. (Sir HENRY HOLLAND'S *Medical Notes and Reflections*.)

By experiments Fontana, Segalas, and Majendie completely demonstrated the absorption of poisons by the veins, and consequently of their circulating with the blood; and that no doubt may remain on the subject, modern chemistry has demonstrated the actual presence of many medicinal substances, either in the blood itself, or in the secretions formed from it. Thus after the free use of *soda*, large quantities of uncombined *alkali* have been found in the serum. *Alcohol* has been obtained by distillation from the blood; while *iodine*, *rhubarb*, the *nitrate of potash*, *iodide of potassium*, and a large number of other substances taken into the stomach, have been found in the urine. Hence poisons are absorbed and mingled with the blood, and are conveyed directly to the parts on which they act, passing with impunity over others for which they have no affinity. The fact of morbid poisons in like manner being absorbed and mingling with the blood, has been shown by many observers experimentally. For example, Professor Coleman produced *glanders* by first removing the healthy blood from an ass, until the animal was nearly exhausted, and then transfusing from a glandered horse blood from the carotid artery into the jugular vein. The glanders in the ass was rapid in its progress, violent in degree, and from this animal he afterwards produced both glanders and *farcy*. *Scarlatina*, *measles*, *syphilis*, and others have now been produced by inoculation from the blood of patients labouring under these diseases.

The circumstance of the presence of a poison in the blood produces, besides its toxic states, certain alterations in its physical condition. Thus, an acute *specific* disease has a tendency to destroy or reduce the quantity of fibrine in the blood, in some instances to one part in a thousand. Hence, whatever may be the nature of the specific pyrexia, the blood always exhibits the following characters, whether it be taken from a vein or collected from the heart and arteries after death,—namely, that the serum and clot are incompletely separated the one from the other, so that the clot is consequently large, and often appears to fill almost entirely the bleeding-basin. Its edges are never raised, and its consistence is inconsiderable, so that it is easily torn, broken down, and reduced to a state of diffuence; in this state it becomes grumous, and discolours the serum. It is also remarkable for the absence of all buff, which is rarely met with in *typhus*, in *measles*, in *scarlatina*, or in *small-pox*, unless there has been some inflammatory complication; and even when it does exist, as in *confluent small-pox*, with large collections of pus, the buff is soft and gelatinous, and, by expression of the serum, is easily reduced to a thin pellicle. This defect of fibrine may be the cause of the great tendency to hæmorrhage, and to that stasis or congestion so remarkable in *typhus fever*, *scarlatina*, and other diseases dependent on morbid poisons (ANDRAL).

The facts and arguments which have been adduced prove that morbid poisons act in all instances not capriciously, but according to certain

definite and specific laws, modified by the influence of climate, temperament, or the magnitude of the dose; also, that they mingle with the blood, with which they continue in latent combination a certain but varying period of time; and likewise that many of them are capable of co-existing together in the same system. A knowledge of these facts is necessary to the proper understanding of this class of diseases, and it is hoped that by their application many of the difficulties which have hitherto obscured the doctrines of fever, of syphilis, of hydrophobia, and of many other diseases incident to this class of morbid poisons, may be removed, and that this portion of medical science may be placed on a surer foundation than heretofore, if not on a permanent basis.

Deaths from Specific or Miasmatic Diseases.—The average annual rate per cent. of mortality in Great Britain for the past seventeen years is represented by 2·245—i.e., nearly 22 per 1000, or 1 in 45 of the population. This statement is given as a fact by which the student may compare the numerical statements which are made in estimating the fatal nature of individual diseases, or of diseases considered in classes. With regard to diseases of a specific or miasmatic kind, it may be stated generally, that from 21 to 26 per cent. of the total number of deaths which take place in Great Britain during a year are due to them. Generally speaking, also, they may be arranged in the order of their greatest fatality, as follow, namely:—(1.) *Cholera*, *typhus*, and other forms of *continued fever*; (2.) *Scarlatina*, *hooping-cough*, *measles*, *croup*, *small-pox*, *erysipelas*; (3.) The other diseases of this kind are less fatal; and it has been observed that of late years *small-pox*, *influenza*, and *typhus fever* are less fatal than they used to be.

It is not yet clearly established how far the bodies of animals may not be a soil for the propagation of diseases communicable to man. (See the Sections on “Small-pox” and “Cow-pox.”) The diseases of the lower animals are not sufficiently studied by us. The diseases of plants are almost entirely neglected. Yet it is clear that until all these have been studied, and some steps taken to generalise them, every conclusion in pathology regarding the nature of the propagation and dissemination of specific, miasmatic, and even of parasitic, dietic, and enthetic diseases, must be the result of a limited experience from a limited field of observation. How do we know that the blights of plants, or the causes of them, are not communicable to animals and to man? We know how intimately related the diseases of man and animals are with famines and unwholesome food; and of famines with the diseases of vegetable and animal life, as much as with the destruction and loss of food. Dr. William Budd, of Bristol, has also recently directed attention to the occurrence of *malignant pustule in England*, in a paper read at the great meeting of the British Medical Association in London, in August, 1862. He has shown that the disease has not been so uncommon in England as had been supposed—that it is common and very fatal to oxen and sheep in this country—that in man and in sheep the disease is identical—that it is communicable to man by direct inoculation, and also by eating the flesh of the animals affected—that it may be conveyed and disseminated by the bites of insects, such as gnats—and that the disease may be recommunicated from man to animals (*Brit. Med. Journal*, January 24, 1863).

Grouping the *acute specific or general diseases* together according to more or less natural alliances, they may assume the following arrangement:—

I. EXANTHEMATA—namely, *small-pox, cow-pox, chicken-pox, measles, scarlet fever*—five in number.

II. CONTINUED FEVERS, defined as such by the College of Physicians, —namely, *dengue, typhus fever, enteric fever, relapsing fever, simple continued fever (specific), puerperal fever*—seven in number.

III. MALARIOUS or PAROXYSMAL—namely, *yellow fever (paroxysmal), ague* in its various forms, *remittent fever, puerperal ephemera* or weed.

IV. EPIDEMIC and MALIGNANT, but paroxysmal rather than continued —namely, *cerebro-spinal fever, and specific yellow fever.*

V. SPECIFIC, EPIDEMIC, and CONTAGIOUS, but not otherwise characterised —namely, *plague, cholera, diphtheria, whooping-cough, mumps, influenza.*

VI. DISEASE-POISONS connected with DISEASES of ANIMALS, with unhealthy states of WOUNDS or SORES, or with epidemic EXANTHEMATA—namely, *glanders, farcy, equinia mitis, malignant pustule, phagedena, sloughing phagedena, hospital gangrene, erysipelas, pyæmia.*

It has been usual to limit the application of the term *contagion* to matter palpable and appreciable, like the fluid or germinal matter of the small-pox or cow-pox vesicle; but when the substances could not be seen in any form, but were believed to exist as emanations not apparent to the senses, such disease-producing matter has been known as “miasms.” Such impalpable emanations or “miasms” were understood to act by *infection*. Considerable confusion has thus always pertained to the use of these terms, *contagion* and *infection*; but when observations like those of Drs. Chauveau and Burdon Sanderson can demonstrate that the essential poison-carrying substance is infinitesimal, capable of living, and of indefinite multiplication, when it comes in contact with suitable soil, the contagiousness of infecting miasms becomes quite intelligible, and the one word becomes synonymous with the other. It would be a great step gained were we able to individualise the *something* which is the poison-carrier or disease-producer; although we may be unable to show what is the nature of the peculiar vital property it possesses, which is capable of producing effects so specific as the disease its inoculation brings about. We are just as far on in our knowledge here, as in regard to spermatic or ova germs, each of which are merely particles individualised. We know only that IT (the *something*) may be implanted, that IT (the *something*) lives, multiplies, and is reproduced a thousandfold. We know also that time for incubation and time for development and maturation are important elements in the course, duration, and final termination of all of these diseases. We have no antidote for any of them. We cannot cure any of them; we can but guide the patient with the best of our knowledge and experience through such dangerous periods of life as the occurrence of many of these diseases imply.

A distinction ought to be made, however, between the poisons that are reproduced during the processes of disease, as of the *exanthemata* and *continued fevers*, contrasted with those poisons which do not appear to be reproduced in the system, as in the case of *malarious* and *paroxysmal* fevers, and which are not believed to be propagated by *contagion* or intercourse with the sick.

CHAPTER III.

ON THE NATURE OF ENDEMIC, EPIDEMIC, AND PANDEMIC* INFLUENCES.

THERE are some characteristic features pertaining to specific diseases of a miasmatic kind which require special notice—First, *They suddenly spring up in a locality—under unfavourable sanitary conditions.* Second, *They may rapidly spread at irregular intervals, so as to incapacitate or destroy great numbers of people.* These two marked and striking features are technically described as being due respectively to *Endemic and Epidemic influences.*

Endemic Influences result from those conditions or agencies peculiar to a locality which favour the development of various miasmatic diseases, and may thus account for their sudden origin. These endemic influences, for the most part, are exerted through the geological properties of a district, and are traceable to the constitution and state of the soil, water, and air; to elevation above the level of the sea, vicinity of the sea, rivers, or stagnant water, woods, and vegetation; variations of temperature, prevalent winds; in connection with avocations, modes of life, quality of food and quantity, as modified by moral agencies, such as indolence or activity; privation and comforts, filth or cleanliness of people; together with their habits of life and employments, ignorance or mental culture; and, lastly, their social, moral, religious, and political conditions. *Endemic influences* become mainly active or *epidemic* through the following conditions, namely:—1st, That the specific poisons by which the communicable diseases, such as *small-pox* or *typhoid* fever, propagate their kind are never totally in abeyance, but always in existence. 2nd, That the specific communicable diseases are constantly extant somewhere, and only under conditions favourable to their dissemination do they spread or become *epidemic*. Although their *living contagia, germs, specific gases, active principles, or media of propagation and development,* may lie dormant or latent for a time, it is not to be inferred that they have ceased to exist. 3rd, That the history of all the specific communicable diseases demonstrates the same alternations of slumber and activity; of widespread prevalence in one place, while neighbouring places may remain free; and, finally, the same successive invasion of neighbouring places, such that the prevailing disease only begins to prevail in the new locality after it has already died out in the old. 4th, That one element always remains constant in the history of endemic influence, and that is the *specific morbid poison* which is the origin of each case. It is susceptible of transmission from place to place, gathering strength as it proceeds, again to die out or become dormant, so

* Surgeon-General Dr. Lawson recently called the attention of the profession to what he considers to be *oscillations* of influences over the whole world, determining febrile diseases. To such influences he gives the name of "Pandemic" (*Transactions of Epidemiological Society* for 1862; also, *Statistical, Sanitary, and Medical Reports of the Army Medical Department* for 1861).

that its track is with difficulty followed or traced out. 5th, That in large cities such specific poisons are always more or less active, and their diseases always present; but in the country districts they only now and then occur. The occurrence of long intervals of rural exemption is not traceable to any feebleness of the poison to act; for when the disease does become developed in these places, the ratio of persons or of animals attacked is incomparably greater than is ever seen in cities under like circumstances. 6th, That in large towns the sewers are constantly charged with the *materies morbi* of specific diseases always abounding in towns. In small villages, and other places where no sewers exist, the air only may be infected, or the water contaminated, by the direct or indirect importation of cases of specific disease or their equivalents—the poison itself—so that the organic impurities, the dung-heaps, the open soil which surrounds the dwellings of the patients, the cesspools, and the privies common to several houses, gradually but eventually become impregnated with the specific poison of the disease. Thus the atmosphere of the village may become incomparably more virulent than the atmosphere of the sick-chamber itself. Hence the rapid epidemic spread of some miasmatic diseases in the limited space of rural villages; which gives rise to the popular error, that such diseases are invariably contagious in country places, and only rarely so, or by exception, in cities or large towns. 7th, That all these specific diseases multiply their kind after similar modes of propagation. 8th, That each of them establishes a constant series of morbid changes and lesions, and always issues in the reproduction of its own *contagium vivum*, *specific germ*, *morbid poison*, or *active principle*, by which it propagates its kind. Thus *small-pox* propagates *small-pox*, *measles* multiplies *measles*; *scarlatina* reproduces *scarlatina*; *typhoid fever* breeds *typhoid fever*; *typhus*, *typhus*; and so on. In “races the most diverse, under climates the most various, age after age, through endless generations of man, these diseases pass down through the human body (sometimes through animals—e.g., *ovine small-pox*?), perpetuating their own kind, and each maintaining its separate identity by marks as specific as those which distinguish the ash from the alder, or the hemlock from the poppy.” Such being the case, it is difficult to conceive “*that diseases of whose propagation this is the history can ever be generated in any other way*” (WM. BUDD, Sir THOS. WATSON). Most of these miasmatic diseases also are peculiar to man; while animals, on their part, are infested by a whole brood of communicable diseases, no less specific in their kind, each distinct from the other, and most of them, although some may be communicable to man, are incapable of multiplying in the human body. Cattle appear to be subject to a variety of malignant and communicable fevers from which man is altogether exempt as yet. 9th, That certain receptive conditions, or a predisposition (the nature of which is unknown), exists in individuals, which appear essential to the development of the specific poisons and the establishment of the disease; and immunity against a repetition of the disease is generally conferred by one attack of the same disease—an immunity which has been proved by experiment on an enormous scale with regard to small-pox; and with regard to the other diseases of this kind, the belief in such immunity is deduced from extensive observation. But the immunity acquired by one attack of any of these diseases is of no

avail against the rest. Measles, for example, renders the body proof against measles, but leaves it as open to small-pox as before, and still more so to hooping-cough; and so with hooping-cough to measles, and so on of the rest. 10th, That with regard to fermentation, putrescency, or decomposition, there is some reason to believe that it may quicken the activity or facilitate the development of specific morbid poison, in the way of a predisposing cause. There is no small amount of circumstantial evidence tending to show that endemic conditions may be thus far favourable to the propagation of specific diseases, even to the extent of epidemics, in consequence of the predisposing agency of putrescent emanations; and, on the other hand, both *endemic* and *epidemic influences* are often held in abeyance by the tendency to decay, decomposition, and destruction of the *specific germs, living contagia, or disease poisons* themselves. They are stamped with the tendency to change and to perish. Like all organic substances which propagate from minute or invisible beginnings, myriads perish for one that is fruitful. This is especially demonstrable in respect of the parasitic diseases, whose germs would overrun the world if they all came to maturity; but the extinction or the dispersion of the specific poisons is abundantly provided for, through the operation of many natural causes; and by imitating some of these operations of nature, we may be able eventually to exterminate, or, at all events, greatly to modify the severity, and reduce the mortality from many of these diseases.

The belief in the spontaneous *endemic* origin of the specific miasmatic diseases rests on evidence entirely *negative*—namely, the fact that cases do spring up in which it is impossible to trace the disease back to a personal source of specific propagation and dissemination—an event which is inherent in the very nature of these diseases. For the active principle of the poison is invisible, although the matter that is known to contain it may be capable of isolation and inoculation, as in small-pox; yet the existence of the specific disease-poison is known to us by inference only. Again, we know that ample provision is made, and ways are open, for the dissemination of the active agent of propagation in a thousand unseen modes, so that it is obvious that the precise source of infection and its track must often baffle the wisdom of man to discover or trace it out. Cases thus constantly arise which appear to give countenance to the belief that the disease has had a spontaneous origin—*sporadic*, as it is termed. Numerous cases of small-pox occur which can never be traced to their source, or to communication with persons similarly diseased; yet the history of small-pox is decisive against the notion of its spontaneous origin; and if of small-pox, so for all other specific diseases of the same nature. Small-pox never occurs except from contagion. *It was quite unknown in Europe till the beginning of the eighth century.* No mention of any such malady is to be found in the Greek or Roman authors of antiquity. Now, whatever may have been the deficiencies of the ancient physicians, they were excellent observers and capital describers of disease; and it is impossible that a disease so diffusive, and marked by characters so definite and conspicuous, should have escaped their notice, or have been obscurely portrayed (if known) in their writings. On the other hand, Mr. Moore, in his learned and interesting *History of Small-Pox*, has shown that it prevailed in China and Hindostan from a very early period—even more

than a thousand years before the time of our Saviour. That it did not sooner extend westward into Persia, and thence into Greece, may be attributed partly to the horror which the disease everywhere inspired, and the attempts that were subsequently made to check its progress, by prohibiting all communication with the sick, partly to the limited intercourse which then took place among the Eastern nations; but principally to the peculiar situation of the regions through which the infection was diffused, separated as they are from the rest of the world by immense deserts and by the ocean" (WATSON, *Lectures on the Practice of Physic*, 3rd edition, Vol. II., p. 709). "If anything were wanting," writes Dr. Budd, "to show what is the true inference to be drawn from these events, it would be found in the fact that, *once imported into the West*, it spread with the most fearful rapidity and havoc; and that, while almost all men are prone to take the disorder, large portions of the world have remained for centuries exempt from it, until at length it was imported, and that then it infallibly diffused and established itself in those parts. In this country the (endemic) conditions for the spread of the disease existed in the most intense degree, as was shown by the event when the disease was once introduced. The long lapse of ages during which we remained entirely free from small-pox showed, with equal clearness, that, until this introduction occurred, all the conditions favourable to the development of small-pox were powerless to cause a single case. The spectacle witnessed in Europe was repeated over again in the Western World in a still more striking way. Our knowledge of the events here is precise and sure. There was no small-pox in the New World before its discovery by Columbus, in 1492. In 1517, the disease was imported into St. Domingo. Three years later, in one of the Spanish expeditions from Cuba to Mexico, a negro, covered with the pustules of small-pox, was landed on the Mexican coast. *From him* the disease spread with such desolation that within a very short time (according to Robertson) three millions and a half of people were destroyed by it in that kingdom alone." "Again, small-pox was introduced into Iceland in 1707, when sixteen thousand persons were carried off by its ravages—more than a fourth part of the whole population of the island. It reached Greenland still later, appearing there for the first time in 1733, and spreading so fatally as almost to depopulate the island," (BUDD, l. c., p. 35, *et seq.*) No common conditions of human life gave rise to such phenomena. Propagation from the actual poison of a pre-existing case was the one necessary and all-sufficient condition for these endemic outbreaks and their epidemic prevalence.

The precise mode in which the miasmatic diseases, with their specific poisons, first came into existence, is beyond our *ken*—hidden from us as yet by a veil, and remaining an inscrutable, at least as yet an unpenetrated mystery. But everything tends to show that once created, they all propagate only in one way—namely, by continuous succession.

Epidemic Influence.—The second characteristic feature peculiar to some of the miasmatic diseases is, that they sometimes spread rapidly, so as to incapacitate and destroy great numbers of the people. The disease is then said to be *epidemic* (επι, upon; and δῆμος, the people). "All we can say, with certainty, regarding epidemics, is, that there must be some distempered condition of the circumstances around us—some secret power

that is operating injuriously upon our system—and to this we give the name of *epidemic influence* or *constitution*," which is believed to predispose towards the receptivity of specific disease-poisons.

Laws of Epidemic Influence, and Conditions under which they act.—

(1.) This influence frequently predisposes to diseases, apparently independently of any other known cause, as in the case of influenza and cholera. It makes itself manifest by appearing to give increased energy to causes which produce particular diseases; so that *small-pox*, *scarlatina*, *measles*, *typhus*, and the like, sometimes rage with great violence as epidemics. It also appears to predispose to new and anomalous forms of disease, as witnessed in the *furunculoid epidemic* which recently prevailed both in Europe and America, from 1849 till 1852. (2.) Sometimes the *epidemic influence* manifests itself by a certain type or direction which existing diseases appear to take. Thus, at one period diseases take a low, or what is called a *typhoid* type, so that depletion is not tolerated; at another time an *inflammatory* tendency predominates, and antiphlogistic treatment is required. At one period there is a tendency in disease to complicate its course by a disposition to affect particular organs. At one time head affections predominate; at another time affections of the chest, or of the alimentary canal, complicate the course of a prevailing disease. Consequently the same disease may demand very different, and even opposite modes of *management*. (3.) During epidemics other diseases are apt to assume more or less of the prevailing epidemic features. Thus, when cholera prevails, looseness of the bowels often complicates the course of other affections. When influenza prevails, catarrhal complications increase the danger of other diseases. Ill health of any kind, therefore, favours the action of the epidemic influence. (4.) Some change in the character of prevailing diseases of a constant and recurrent kind often indicates the approach of an epidemic and the prevalence of the epidemic influence. (5.) The first effects of the *epidemic influence* are usually the most violent and marked, and the cases of the epidemic disease become mild as the *epidemic influence* passes away. (6.) The *epidemic influence* sometimes disappears entirely after a short prevalence; sometimes continues with irregular intermissions for two, three, four, or even six years, or longer. Influenza and cholera are examples. (7.) An epidemic tendency, after continuing for several years, may give place to one of a different kind, which, in its turn, may again give place to the first. *Malarious fevers*, *yellow fever*, and *typhus* illustrate this in America. The eruptive affections seem to run in somewhat similar cycles. After the introduction of vaccination *small-pox* seemed for many years to be almost entirely subdued; but more recently again the disease has seldom been entirely absent from among us, alternating as an epidemic now and then with *measles*, *scarlet fever*, and *typhus*. We look forward to the time when vaccination, enforced by law, will completely eradicate the disease. (8.) The lower animals are also subject to epidemic influences; and seasons of unusual fatality among them have coincided with those in which the human race have suffered. This fact has been well shown in an elaborate and erudite analysis of the census of Ireland, by Sir William R. Wilde, of Dublin, the diseases of the population having been recorded at the time.

Pandemic Influences.—The expressions of the hitherto prevailing doc-

trines regarding *endemic* and *epidemic* influences appear so unsatisfactory to many minds, and leave so many circumstances regarding the spread of diseases unexplained, that attention is being directed to more comprehensive views and investigations of the questions involved in the preceding paragraphs. An ingenious theory has been propounded by Inspector-General Dr. Lawson, who has attempted to establish the occurrence, between 1817 and 1836, of a series of *oscillations of febrile diseases*, following each other over the world with amazing regularity. The mode of occurrence of such febrile diseases he attributes to a cause or influence which, from its extent and *progressive* character, he names a "*pandemic wave*," to distinguish the influence from that usually understood as *epidemic*, referring to a single form of disease affecting a limited space. Under the influence of this *pandemic wave* Dr. Lawson believes that there is a constantly progressive tendency to the development of all *endemic* febrile diseases in the Atlantic and western parts of the Indian Ocean, from south or south-east to north or north-east. But the facts and data on which this theory is made to rest are not of sufficient number, many of them are not sufficiently trustworthy, to rest a judgment upon; and in not a few instances a totally different interpretation may be given to that which Dr. Lawson has assigned to them. Although, therefore, it may be premature to propound such a theory, especially as it is still open to the verdict of "not proven," yet the expression of it is calculated to do good, by drawing attention to the subject, and to the comprehensive world-wide range which must be given to such investigations; and to whom can science look with more hope for results than to the medical officers of Her Majesty's British and Indian Army and Navy?

A successful study of these peculiar and characteristic features of miasmatic diseases—namely, the *endemic*, *epidemic*, and *pandemic influences*—is of the utmost importance to the student. He will learn to appreciate how much mortality may be diminished by well directed hygienic measures, such as cultivation and improvement of the soil, extension of commerce, improvements in diet and the social circumstances of the lower classes—especially in regard to cleanliness; ventilation; domestic management of improved dwellings; efficient sewerage; care in the separation and treatment of the sick when in numbers; and the use of strict measures of a prophylactic kind suited to the circumstances of the case. Next to large towns, the health of the army is of the greatest importance, especially when we consider the tendency that exists to a high rate of mortality in that service. In the military age (which is the age between eighteen and forty) the mortality of the general population in England is less than 1 per cent. per annum. The mortality of the British army is much above this. On home service it has had a mortality double that of the civil population at the corresponding ages; and seven-ninths of the entire mortality among the infantry of the line has arisen from diseases of the specific preventible kind. Disease and mortality are much greater during campaigns, when more than 22 per cent. are constantly on the sick list. The causes of high rates of mortality require constant investigation, by carefully observing, recording, and comparing the facts over a sufficiently large area, thus arriving at certainty as to the causes, and whether they can be mitigated or removed.

An observation of great interest in connection with *animal malaria poison*, as well as with *epidemic influences*, may be appropriately referred to here. It seems clearly proven, especially by the valuable and decisive observations of Dr. William Budd, of Bristol, that the communicable poisons of typhoid fever and of cholera are capable of being imported or carried from place to place by persons who have the disease. Dr. Budd's history of the North Tawton fever and its offshoots (*Lancet*, July 9, 1860) is most conclusive on this point. His arguments are also cogent to the general effect that *especially* the bowel discharges are *means* by which a patient, whether migrating or stationary, can be instrumental in disseminating typhoid fever and cholera. Mr. Simon makes the important remark, however, that these bowel discharges may not be the *sole means* of multiplying and disseminating these diseases; although, provisionally, the conclusions of Dr. Budd must be acted upon in their present unqualified form; while it is of the greatest practical importance to learn, as exactly as possible, whether it is in all states of the disease, and under all circumstances, that the bowel discharges of typhoid fever and cholera can communicate and multiply the means of dissemination. In illustration of such possible contingent results, Mr. Simon refers to some interesting and important experiments made in 1854, by Professor Thiersch, of Erlangen. These experiments seemed to show that cholera evacuations, *in the course of their decomposition*, either acquire the power of communicating or multiplying their specific poison, or that the specific poison inherent in them becomes intensified by decomposition (zymosis?). That the decomposition or change may begin even in the bowels, after the secretion and accumulation of the material in them, as well as in cesspools, seems to be possible; and perhaps, as Mr. Simon justly remarks, may furnish an explanation of the many cases in which human intercourse has apparently disseminated the disease. According to the observations of Professor Pettenkofer at Munich, and Professor Acland at Oxford, it would seem that during cholera periods the immigration of persons suffering apparently only from diarrhœa has been followed by outbreaks of cholera in places previously uninfected; and Professor Pettenkofer ascribes this fact to an influence (zymotic?) exerted by the decomposing fæces of such diseased persons in the cesspools and adjoining soil of ill-conditioned places to which they go. Specific poison-properties of this kind would thus arise, and probably extend to the pollution of well-waters of such soils, and might render them, if swallowed, capable of exciting cholera, or typhoid fever or dysentery, by direct *contagion*; and so any of these diseases would thus have all the appearance of having arisen *de novo*. It is encouraging to sanitary reformers, as Mr. Simon justly remarks, that cases of the apparent introduction of cholera contagion by human intercourse are essentially different from cases of the dissemination of such specific diseases as small-pox or measles. The multiplication of the specific poison in the latter diseases takes place exclusively within the human body. The multiplication and dissemination of them have no immediate dependence on differences of medium; but wherever human beings can cross one another's path, to the susceptible or unprotected person these specific diseases may be communicated. On the other hand, it seems really to be the fact that the cholera poison (and probably, also, typhoid fever poison and

dysentery), if it can at all be multiplied within the body, almost certainly has its great centres of multiplication *elsewhere*—namely, in those avoidable foci of corruption where excrement accumulates and decays. Military authorities ought to remember this fact. They have had abundant evidence of it in the old camping grounds of the Indian Army, as well as when following the route and encamping on the ground previously occupied by retreating armies in other countries, as in the Russian War of 1854-5. For disseminating the disease and multiplying the poison, foulness of medium seems indispensable; and it is no ordinary foulness which will impart to air, food, or water, the zymotic action of decomposing excrement. The taint is something specific. Therefore, as regards cholera, it seems highly probable that the immigration of infected persons might occur to a very great extent without exciting epidemic outbreaks, if such immigration were only made into places of irreproachable sanitary conditions, especially as regards water supply and the continuous removal of house refuse or camp filth. (Consult SIMON, in his *Public Health Reports*—especially *second* and *third*, and later series, relative to the people of England; also PETTENKOFER, ACLAND, and THIERSCH.)

CHAPTER IV.

MANAGEMENT OF EPIDEMICS; AND ON PROCEEDINGS WHICH ARE ADVISABLE TO BE TAKEN IN PLACES ATTACKED OR THREATENED BY EPIDEMIC DISEASES.

THE practical questions immediately involved in the exposition which has been given of the nature of the specific or miasmatic diseases in particular is contained in the following statement, namely:—That it is possible to extinguish or “*Stamp out*” the greater number of epidemic diseases, however intense or abundant may be the atmospheric or other agencies which constitute their potential causes, by acting according to the dictates of the following elementary facts:—

(1.) That the living body of the diseased persons is the soil on which the communicable disease breeds the poison by which the specific disease is multiplied and propagated.

(2.) That excretions from an infected person, especially such excretions as are immediately related to or flow from parts affected with specific lesions, probably contain the most active elements of the specific poison by which the disease may be disseminated.

(3.) That such active elements, *germs, poisons, active principles*, or *noxious agents*, may contaminate the drinking-waters of a district, or may infect the atmosphere, or lie dormant for variable and unknown periods of time, just as seeds or ova dry up and preserve their vital properties. The principles of action which those elementary facts dictate are as follow:—

(a.) To follow out zealously the hygienic measures which flow from these

statements, and so prevent the propagation of specific diseases. (b.) To preserve as much as possible the blood of every individual in that state which shall prevent these poisons from finding the conditions of their development within the body. (c.) That these ends are to be attained, on the one hand, by preventing the production of fermentable matter in or out of the body; and, on the other hand, by promoting its removal and chemical destruction or decomposition, when it is inevitably generated, and by a free supply of pure air, and by the reduction of that air to the lowest temperature at which the condition of the individuals will allow it to be safely inhaled. Preventive measures based upon these principles are of the utmost importance.

Detail of Proceedings advisable to be taken in Places attacked or threatened by Epidemic Diseases.

1. Wherever there is prevalence or threatening of any epidemic disease, it is of more than common importance that the powers conferred by the Nuisances Removal Acts, and by various other laws for the protection of the Public Health, be vigorously, but at the same time judiciously exercised by those in whom they are vested; and with regard to armies, that the instructions relative to the guidance of the Medical Officer in sanitary matters, contained in the *Army Regulations*, be duly carried out, on the principle that *the executive should act under authority*, in order to carry out the required measures efficiently.

2. If the danger be considerable, it will be expedient that the local authorities in civil life, and the commanding officers of armies, brigades, divisions, and regiments in military life, avail themselves, as soon as possible, of the medical advice within their reach, in taking measures of prevention and protection against the spread of disease.

3. Measures of precaution for prevention and protection are equally proper for all classes of society, civil and military. But it is chiefly with regard to the poorer civil population, therefore chiefly in the courts and alleys of towns, and at the labourers' cottages of country districts, that local authorities are called upon to exercise the utmost vigilance, and to proffer information and advice. Common lodging-houses, and houses which are sublet in several small holdings, always require particular attention.

4. Wherever there is accumulation, stink, or soakage of house refuse, or of other decaying animal or vegetable matter, the nuisance should as promptly as possible be abated, and precautions should be taken not to let it recur. Especially all complaints which refer to *sewers and drains*, or to *low ditches and ponding of drainage*, or to *neglect of scavenging*, should receive immediate attention. The *trapping* and ventilation of *house drains* and *sinks*, and the state of *cesspools* and *middens*, should be carefully seen to. In *slaughter-houses*, and other places where beasts are kept, strict cleanliness should be enforced.

5. In order to guard against the harm which sometimes arises from disturbing heaps of offensive matter, it is often necessary to combine the use of chemical disinfectants with such means as are taken for the

removal of filth; and in cases where removal is for the time impossible or inexpedient, the filth should always be disinfected. Disinfection is likewise desirable for unpaved earth close to dwellings, if it be sodden with slops and filth. Generally, where cholera or typhoid fever is in a house or barrack, hospital or hut, the privies especially require to be disinfected.

6. Sources of water supply should be carefully and efficiently examined. Those of them which are in any way tainted by animal or vegetable refuse—above all, those into which there is any leakage or filtration from sewers, drains, *cesspools* or *foul ditches*—ought no longer to be drunk from. Where the disease is cholera, diarrhoea, or typhoid fever, it is especially essential that no foul water be drunk.

7. The washing and lime-whiting of uncleanly premises (houses, huts, hospitals, barrack guard-rooms, and the like), especially of such as are densely or multifariously occupied, should be pressed with all practicable despatch.

8. Overcrowding should be prevented. The sick-room should, as far as possible, be free from persons who are not of use or comfort to the patient.

9. Ample ventilation should be enforced. Window-frames should be seen to,—(1.) That they may be made to open, if not so made; and, (2.) That they be kept sufficiently open. Especially where any kind of specific disease, communicable by infection of the air, has begun, it is essential, both for patients and for persons who are about them, that the sick-room and the sick-house or hospital be constantly and efficiently traversed by streams of fresh air. This is especially necessary at night, and steps should be taken to ensure efficient ventilation, even at some real or imaginary expense of comfort.

10. The cleanest domestic habits should be enjoined. Refuse matters should never be suffered to remain or to linger within the dwelling, hospital, barrack-room, or hut. Such refuse must *at once* be removed, and at once disposed of, or cast into the receptacle provided for it. All things or utensils which have to be disinfected or cleansed should always be so *without delay*.

11. With regard to material substances discharged or separated from the bodies of the sick, special precautions of cleanliness and disinfection are necessary. Among discharges or substances separated from the body which it is proper to treat as capable of communicating disease, are those which come, in cases of small-pox, from the affected skin; in cases of cholera and typhoid fever, from the intestinal canal; in cases of diphtheria and scarlatina maligna, from the nose and throat, and the exhalations from the skin and the lungs saturating clothes; likewise, in cases of eruptive fevers, measles, scarlatina, *rötheln*, typhus, and the like, the general exhalations of the sick, and especially so of the convalescing, probably in connection with the desquamation of the skin. The caution which is necessary with regard to such matters must of course extend to whatever may be imbued with them; so that bedding, clothing, towels, and other articles which have been in use by the sick, do not become sources of mischief, either in the house to which they belong, or in houses to which they are conveyed. Moreover, in typhoid

fever and cholera, the evacuations should be regarded as capable of communicating a similarly specific and infectious property to any night-soil with which they may be mingled in privies, drains, or cesspools. This danger of multiplying the sources of communicating disease must be guarded against by the chemical destruction, decomposition, or disinfection of all the intestinal evacuations as soon as they are passed from the bowels, and certainly before they are thrown away, and so "let loose upon the world." Above all, they must never be cast where they can run or soak into sources of drinking-water.

12. All reasonable care should be taken not to disseminate disease by the unnecessary association of persons suffering from the specific communicable diseases, either with healthy persons, or in wards of hospitals where patients suffering with other diseases are being treated. This care is requisite, not only with regard to the sick-house, ward, hospital, or ship, but likewise with regard to day-schools, churches, places of public resort, courts of justice, and other places where members of many different households are accustomed to meet.

13. Where dangerous conditions of residence cannot be promptly remedied, it will be best that the inmates, while unattacked by disease, remove to some safer lodging. If disease begins in houses where the sick person cannot be rightly circumstanced and tended, medical advice ought to decide on the propriety or fitness for removing him to an infirmary or hospital. In extreme cases, special infirmaries may become necessary for the sick, or special houses of refuge for the endangered.

14. The question of quarantine ought to be decided by the circumstances of the special case, the preceding principles being kept in view.

15. Privation, as predisposing to disease, may require special measures of relief.

16. In certain cases special medical arrangements are necessary. For instance, as cholera in this country almost always begins somewhat gradually, in the comparatively tractable form of what is called "premonitory diarrhoea," it is essential that, where cholera is epidemic, arrangements should be made for affording medical relief without delay to persons attacked even slightly with looseness of the bowels. So again, where small-pox is the prevailing disease, it is essential that all unvaccinated persons (unless they previously have had small-pox) should very promptly be vaccinated; and revaccination should also be offered both to persons above puberty, who have not been vaccinated since childhood, and to younger persons whose marks of vaccination are unsatisfactory.

17. It is always to be desired that the people should, as far as possible, know what real precautions they can take against the disease which threatens them; what vigilance is needful with regard to its early symptoms; and what, if any, special arrangements have been made for giving medical assistance within the district. Especially in the case of small-pox or of cholera, such information ought to be spread abroad by means of printed bills or placards. In any case where danger is great, house to house visitation, or personal inspection of all by discreet and competent persons, may be of the utmost service, both in quieting unreasonable

alarm, and in leading or assisting the less educated and the destitute parts of the population to do what is needful for safety.

As addenda to these excellent memoranda, promulgated by Mr Simon, the following rules, the observance of which is enjoined by the government of the London Fever Hospital, might well be adopted under similar circumstances in military and in civil hospitals :—

1. It is of the utmost importance to the sick and their attendants, that there be a constant admission of fresh air into the room, and especially about the patient's bed, care being taken to prevent the wind from blowing directly on the patient.

2. Attention to cleanliness is indispensable. The linen of the patient should be often changed, and the dirty clothes, &c., immediately put into fresh cold water, and afterwards well washed. The floor of the room must be cleansed every day with a mop, and all discharges from the patient immediately removed, and the utensils washed.

3. Nurses and attendants ought to endeavour to avoid the patient's breath and the vapour from the discharges.

4. Visitors must not go near to the sick, nor remain with them longer than is absolutely necessary ; they should not swallow their spittle, but clean the mouth and nostrils when they leave the room.

5. No dependence must be placed on vinegar, camphor, or other supposed preventives, which, without attention to cleanliness and admission of fresh air, are not only useless, but by their strong smell render it impossible to perceive when the room is filled with bad air or noxious vapours.

Processes of Disinfection.—These processes have been recommended by the late Professor Miller, of King's College, London. They cannot supply the place of cleanliness, ventilation, and drainage. They are artificial, and are used for exceptional purposes, the great natural disinfectant being *fresh air*, abundantly and uninterruptedly supplied.

1. For purposes of artificial disinfection, the agents which most commonly prove useful are, *chloride of lime*, *quicklime*, *Condy's manganic compounds*, and *carbolic acid*. *Metallic salts*, especially *perchloride of iron*, *sulphate of iron*, and *chloride of zinc*, are under some circumstances applicable. In certain cases *chlorine gas* or *sulphurous acid gas* may advantageously be used ; and in certain other cases powdered charcoal or fresh earth.

2. If *perchloride of iron* or *chloride of zinc* be used, the common concentrated solution may be diluted with eight or ten times its bulk of water. *Sulphate of iron* or *chloride of lime* may be used in the preparation of a pound to a gallon of water, taking care that the water completely dissolves the *sulphate of iron*, or has the *chloride of lime* thoroughly mixed with it. *Condy's stronger fluid* (red) may be diluted with fifty times its bulk of water ; his weaker fluid (green) with thirty times its bulk of water. Where the matters requiring to be disinfected are matters having an offensive smell, the disinfectant should be used till the smell has entirely ceased.

3. In the ordinary emptying of privies or cesspools, use may be made of *perchloride of iron*, of *chloride of zinc*, or of *sulphate of iron*. But where disease is present, it is best to use *chloride of lime* or *Condy's fluid*. Where

it is desirable to disinfect before throwing away the evacuations from the bowels of persons suffering from certain diseases, the disinfectant should be put into the night-stool or bed-pan when about to be used by the patient.

4. *Heaps of manure* or of other *filth*, if it be impossible or inexpedient to remove them, should be covered to the depth of two or three inches with a layer of freshly burnt *vegetable charcoal* in powder. Freshly burnt lime may be used in the same way, but is less effectual than charcoal. If neither charcoal nor lime be at hand, the filth should be covered with a layer, some inches thick, of clean dry earth.

5. *Earth near dwellings*, if it has become offensive or foul by the soaking of decaying animal or vegetable matter, should be treated on the same plan.

6. *Drains and ditches* are best treated with *chloride of lime*, or with *Condy's fluid*, or with *perchloride of iron*. A pound of good *chloride of lime* will generally well suffice to disinfect 1,000 gallons of running sewage; but of course the quantity of disinfectant required will depend upon the amount of filth in the fluid to be disinfected.

7. *Linen and washing apparel*, requiring to be disinfected, should, without delay, be set to soak in water containing, per gallon, about an ounce either of *chloride of lime* or of *Condy's red fluid*; the latter, as not being corrosive, is preferable. Or the articles in question may be plunged at once into boiling water, and afterwards, when at wash, be actually boiled in the washing water.

8. *Woollens, bedding, or clothing*, which cannot be washed, may be disinfected by exposure for two or more hours in chambers constructed for the purpose, and heated to a temperature of 210° to 250° Fahr.

9. For the disinfection of *interiors of houses*, the ceilings and walls should be whitewashed with quicklime. The wood-work should be well cleansed with soap and water, and subsequently washed with a solution of *chloride of lime*, about two ounces to the gallon.

10. *A room no longer occupied* may be disinfected by *sulphurous acid gas*, or *chlorine gas*,—the first by burning in the room an ounce or two of flowers of sulphur in a pipkin; the second by setting in the room a dish containing a quarter of a pound of finely powdered black oxide of manganese, over which is poured half a pint of muriatic acid, previously mixed with a quarter of a pint of water. In either case the doors, chimney, and windows of the room must be kept carefully closed during the process, which lasts for several hours.

CHAPTER V.

PATHOLOGY OF THE ENTHETIC, IMPLANTED, OR INOCULATED KIND OF SPECIFIC OR GENERAL DISEASES.

DISEASES of this kind have the common property of becoming developed in the system after the introduction by *inoculation* or *implantation* of

specific poisons. The sources of such poisons are more distinctly traceable than those which produce the *acute specific* or *miasmatic diseases* already noticed; in other words, the substance or material which contains the *poisonous principle* can be obtained in most instances, although the *principle* itself has not been isolated by any chemical process. The poisons which produce the diseases of this order may be introduced through thin or abraded cutaneous surfaces, or through mucous membranes by the process of absorption, although, in most instances, it is believed that some solution of continuity exists. Others are directly introduced by weapons which inflict a wound or abrasion, and which at the same time introduce the poison. "Poisoned wounds" thus indicate or name a disease defined as follows:—"Wounds inoculated with foreign matter, producing general symptoms, or propagating inflammation to other parts of the body" (See Nomenclature under "Poisoned Wounds"). In all instances the poison is received into the system by the processes of absorption, and the individual thus becomes inoculated. Thus, germs of a *specific* kind become directly implanted, and by a zymotic-like process become developed and increased in quantity or virulence till symptoms and effects are produced characteristic of the specific affections; and hence the name given to this order of diseases. No one has better illustrated the pathology of these diseases than Sir James Paget, from whose lectures *On Surgical Pathology* the following statement is given:—

When a morbid poison is inoculated, it produces a peculiar specific effect, according to its kind, both on the tissue at the place of insertion and on the blood, and on the nerve-centres, as soon as the poison, or any part of it, is absorbed; in other words, it produces both a constitutional and a local change. In both these effects its history may be traced in the constancy of the recurring phenomena characteristic of each poison or venom. The specific local change is best seen from the implanting of certain animal poisons direct from the poison glands, such as those of venomous serpents and insects; and results most rapidly follow the direct implantation of such poisons, or indirectly by squirting the poison through the syringe ordinarily used for hypodermic injection. The bite of a bug, for example, is followed, within less than a minute, by itching in the bitten part, and very soon a wheal, or circumscribed pale swelling, with a nearly level surface and a circumscribed border, gradually rises and extends in the skin. The swelling is produced by oedema of a small portion of the cutis at and round the bite. As the itching subsides, the pale swelling becomes less defined, and the more general vascular swelling of the surrounding and adjacent tissues gradually encroaches on the primary swelling at the bitten spot. In about twenty-four hours a papule or some form of secondary inflammation appears, with renewed itching at the site of the puncture. This, too, in the case of the bug-bite, gradually subsides. The primary swelling here described illustrates the immediate effects of the morbid poison on the tissue at and round the seat of inoculation; and within the area of such a swelling the tissues are, by the direct contact or influence of the venom, altered in their nutritive relation to the blood. Such specific alterations of the tissues at the seat of inoculation occur with the *syphilitic*, the *vaccine*, and such-like virus; but the direct influence is most rapidly shown in the effects of the bites of the

vipera, the rattlesnake, and the *cobra di capella*. In such cases sloughing of the areolar tissue is established immediately after the bite. The poison seems to operate at once on the tissue, neither in the direction of the nerves, nor of the absorbents, nor of the blood-vessels; but the slough forms at the puncture, as if the venom had completely and at once killed the tissue (BRODIE, PAGET). A secondary inflammation soon appears at the bitten or punctured part; and the occurrence of this new inflammation may be ascribed, in some measure, to an influence exercised by the virus on the blood; and it proves that the part does not return to health, although the first effects of the inoculation may subside. It proves that some material of the virus remains, or that the effects it has already produced upon the tissues at the injured part alter their relations to the blood, and render the part prone to specific disease. These specific effects upon the part may remain locally quiescent for a considerable length of time—during all that period of latency or incubation which intervenes between the inoculation and the appearance of the specific phenomena peculiarly the result of the venom. But during all this interval—during all this period of incubation—the tissues at the site of inoculation are constantly changing; and the virus itself, like all organic matter, is probably in constant process of transformation till *zymosis* is complete, and the specific disease is fully developed and expressed by various constitutional phenomena. Dr. George B. Halford, Professor of Anatomy in the University of Melbourne, gave recently (*Brit. Med. Journal*, July 20, 1867) an interesting account of the action of the poison of the *cobra* upon the blood.

“When a person is mortally bitten by the *cobra di capella*, molecules of living ‘germinal’ matter are thrown into the blood, and speedily grow into

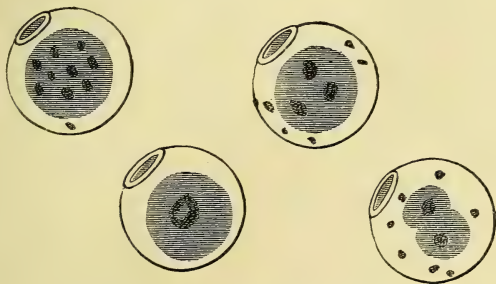


Fig. 78.—Magnifying power $\times 1050$. *



Fig. 79.—Magnifying power $\times 400$. *

* Figs. 78, 79.—Changes in the blood-corpuscles subsequent to the bite of the *cobra di capella*.

cells (Figs. 78, 79), and as rapidly multiply, so that in a few hours millions upon millions are produced at the expense . . . of the oxygen absorbed into the blood during respiration; hence the gradual decrease and ultimate extinction of combustion and chemical change in every part of the body, followed by coldness, sleepiness, insensibility, slow breathing, and death. The cells which thus render in so short a time the blood unfit to support life, are circular, with a diameter, on the average, of $\frac{1}{1700}$ of an inch. They contain a nearly round nucleus of $\frac{1}{2800}$ of an inch in breadth, which, when further magnified, is seen to contain other still more minute spherules of living 'germinal' matter. In addition to this, the application of magenta reveals a minute coloured spot at some part of the circumference of the cell. This, besides its size, distinguishes it from the white pus or lymph-corpuscle."

From this account it is inferred that such changes take place in the blood when the person bitten is still alive; but in a paper read to the Medical Society of Victoria (a copy of which he sent to Sir Joseph Fayrer of Calcutta), Dr. Halford says of these corpuscles,—“he had never seen those cells *before death*, but he believed the organic germinal matter of the serpents' poison to be the efficient agent, and the *post-mortem* changes in the blood to be in some way connected with a metamorphosis of the fibrine of that fluid, which, so far as coagulation was concerned, appeared destroyed by snake poison.” As Dr. Halford's conclusions involve too hasty generalisation, and have not been confirmed by the careful observations of several other competent observers, it may be well to give here a summary of what has been recently determined by careful experiment and observation regarding the—

Physiological Action and Pathology of Specific Venoms.—Francisco Redi, from 1669 to 1675, was the first fairly to examine the poison or venom secreted by serpents. Charas wrote soon after him (*New Experiments upon Vipers*, London, 1673), setting at rest many popular fallacies, and preparing the way for the more elaborate work of Felix Fontana, first published in Lucca in 1767. Fontana made at least 3,000 experiments on all classes of animals, with an amount of industry and scientific sagacity rarely equalled before or since. In the East Indies, Russell and John Davy have given an account of experiments with the venom of the cobra; and more recently (1868, to March, 1871) a most elaborate and exhaustive series of experiments by Sir Joseph Fayrer, “On the Venomous Serpents of the East Indies,” and especially on the action of the poison of the cobra, the daboia, the bungarus and the ophiophagus elaps, demonstrate the nature of those specific poisons. Dr. Ruzf has given an excellent account of the *vipère fer de lance* of Martinique; and in America the toxicology of the rattlesnake and copperhead have been studied with all the advantages of modern research. In Australia, Professor George Halford has illustrated the pathology of the venom of the cobra *di capella*; and Dr. John Shortt, of Madras, has described the nature of the cobra poison, in the Indian snakes of that species, and has come to the conclusion that *potash* neutralises and renders their poison inert; but the practical difficulty remains—namely, how to apply the remedy so that it may rapidly overtake and destroy the action of the poison in the living animal.

When it is considered that in the Madras Presidency of India alone the

deaths from snake-bites during the four years, from 1866 to 1869 inclusive, have been 8,361; that during the nine years, from 1860 to 1868 inclusive, the Commissioner of Burdwar reports to the Bengal Presidency a mortality of 9,232 persons killed by snake-bite, out of a population of 5,701,072, it is no wonder that the subject of snake-bite challenges attention in India. In the district of Midnapore, in 1865, there were 530 deaths, out of a population of 1,200,000 persons. In the last *Oude Administration Report* (1868-69), 1,127 persons died from snake-bite during the year; and in the *Central Provinces Administration Report*, 1,074 had died from the same cause during the three preceding years. These figures, referring as they do to isolated districts of India, may afford some approximate idea of the mortality arising from this cause throughout India and other serpent-infested countries of the East. The most deadly of the poisonous colubrine snakes are the *Ophiophagus elaps* and the *Naja tripudians* in the East Indies. Of the viperine forms, the most deadly are the *Daboia Russellii*, than which there is probably no more deadly viper; and of the *Crotalidæ*, the most deadly in India is the *Trimeresaurus*; but of this family none are so formidable in India as are their congeners in Africa and America, such as the *Crotalus horridus* (rattlesnake), the Jararacaca or *Craspedocephalus Braziliensis*, which are much more deadly than any *Crotalidæ* of India (Sir JOSEPH FAYRER).

The specific venom of these snakes secreted in the special poison-glands behind the eye and in front of the tympanic bones varies much in colour and viscosity in the different snakes; but its general appearance is that of a clear, slightly viscid fluid, soluble in water, and slightly acid in reaction. That of the *Ophiophagus elaps* is of a deep rich orange colour, viscid-looking, and of the consistence of mucus. It manifests its deadly effects most powerfully when inoculated into the blood, when the snake which emits the venom is fresh and vigorous in the warm weather, and when it has not bitten for some time. The result of the numerous observations and experiments that have now been made tends to demonstrate that the poison acts through the circulation, paralysing the nerve-centres, and thus destroying life. It has also been shown to be capable of absorption through the thin mucous membrane of the conjunctiva, though with much less dangerous effects than when it is introduced into the blood.

There are certain well-marked differences in the action of the venom, according as it is derived from different families of snakes. Thus the poison of the *Naja* (cobra) kills without destroying the coagulability of the blood; whilst that of the viper—the *Daboia*, for example—produces perfect and permanent fluidity. It would seem also that the venom of the *Crotalidæ*—for example, the American rattlesnake—not only destroys the power of the blood to clot, and causes the blood to decompose more rapidly than blood not so poisoned, but it seems also able to pass through the tissues, softening and destroying the minute blood-vessels, so that they rapidly rupture under the continuing force of the heart's action. Hence it is that by and by, after the venom of the rattlesnake has been absorbed, the altered blood begins to leak through the various tissues, escaping out of the vessels into the brain, lungs, or intestinal walls, giving in each case specific symptoms, according to the part injured and the function disturbed. The following experiment may explain these points:—"A

young rabbit was made senseless and motionless with chloroform. Its abdomen was then opened, the omentum exposed, and a portion spread under a microscope was kept moist by an assistant, so that the circulation of the blood was distinctly seen in the healthy vessels. A drop of rattlesnake venom was then put upon the tissue under observation; and for *thirty seconds* there was no change, when suddenly, a small vessel giving way, a rush of blood-discs obscured its view. A little way off another small vessel ruptured, then a third, and a fourth, until within five minutes the whole field of vision was obscured by blood." The same phenomena may be seen on the surface of an open wound on which the venom has been placed; and that which happens in the wound, and in the experiment just described, goes on at last everywhere in the poisoned body of the bitten animal, so that in dozens of places vessels break down, while the blood is powerless to coagulate, and Nature's hæmostatics fail. Such are some of the phenomena which attend the more chronic or prolonged cases of rattlesnake bite, as when a larger animal, a dog or a man, perishes after a few hours or days. Having lived over the first few hours of feebleness, a new set of symptoms begins. The blood and tissues become changed in the manner indicated by the experiments and observations on wounds just described. The vital fluid leaks from the kidneys or the bowels, and oozes from the gums. The wounds made by the poison-fangs begin to bleed, and from the prick of a needle blood will drop for hours. Thus exhausted the dog or man may die, or he may slowly recover; and in the meantime the wounds made by the fangs of the snake have undergone a series of changes. A large and growing tumor first marks the site of penetration, which, when cut into, is found to be a mass of fluid blood. By and by this soaks into or infiltrates every tissue in the neighbourhood, and even stains the bones themselves. But the rapid cases of death give no time for these local and constitutional lesions. When death takes place within minutes or seconds, that may be counted by tens, sixty and ninety seconds are the most rapid examples of the action of snake poison Sir Joseph Fayrer has seen—namely, in a half-grown fowl bitten by a *daboia*. He has since recorded death in a dog in seventy seconds; and when the venom of a fresh *cobra* is injected directly into the jugular vein, to the amount of ten drops, with the hypodermic syringe, no trace or alteration can be seen in the blood or solid tissues. A direct power to injure the great nerve-centres which preside over locomotion, respiration, and the heart's action, has circulated with the rapidity of the blood's flow, and so caused death. Thus, a pigeon has been known to die under the influence of *two drops* of the poison of the rattlesnake injected under the skin. On a sudden, within sixty seconds of the insertion of the poison, the bird is dead, and the tissues reveal no cause of death. If *half a drop* only is used, in a few minutes after its insertion the bird staggers, and at last crouches down, too feeble to walk. The feebleness increases, vomiting occurs, breathing becomes laboured, the head droops, a slight convulsion follows, and the pigeon is dead.

In countries where such poisonous snakes abound, and cases of snake-bites are numerous and not fatal, it is not uncommon to find that those who survive may become the victims of blindness, skin disorders, and various forms of palsy. The absolute danger to life is directly as the amount of venom

efficiently inserted into the vascular textures. (See SANDERSON *on Infection*.) Analysing the symptoms of rattlesnake poison, the first effects of the venom are a dangerous depression of all the functions, exactly like what follows an overdose of tartar emetic; and the obvious treatment is to stimulate the man or animal bitten. But no stimulant will destroy the venom in the sense of an antidote; it only antagonises its activity. Nearly all the most reputed antidotes and stimulants have been made the subject of experiment by Sir Joseph Fayrer in India regarding Indian snake poison; and by others in America regarding the rattlesnake, by mixing the venom with the antidote or stimulant, and injecting both, either together or following up the antidote immediately after the poison, without withdrawing the nozzle of the syringe, so that the antidote went immediately after, and in the same channels as the poison; yet the subjects of all the experiments died, just as if no antidote had been administered (*Atlantic Monthly* and *All the Year Round*, March 28, 1868, p. 372). Thus the poison of all snakes may be diluted with water, or ammonia, or alcohol, without destroying its deadly properties. It may be kept for months or years, dried between slips of glass (just as vaccine virus is kept on points or between glass, or as small-pox matter was wont to be kept), and still it retains its virulence.

The Prince of Cassino, Lucien Buonaparte, wrote a short chemical paper on the subject of snake poison in 1843, and gave an analysis of the venom of the *vipera* (Pelas) *Cerus* (the adder), and pointed out the presence of a principle representing the *ptyaline* of saliva. This he called *viperine*. He also found albumen and mucous fatty matter, substances soluble in alcohol, yellow colouring matter, and saline matters. But the elementary analysis of *viperine* has yet to be made. All that is really known regarding it is, that it is a very unstable substance, of neutral reaction, and that it kills through its influence on the nerve-centres (Sir JOSEPH FAYRER, in series of papers in *Edinburgh Monthly Medical Journal*, 1868, 1869, and 1870). The terms *echidnine*, *crotaline*, have been given to similar active principles derived from the venom of other snakes, and which are probably identical. The poison acts most rapidly on birds and mammals; less so on cold-blooded animals; but fishes, frogs, molluscs, and non-venomous snakes are destroyed by it, and often die very rapidly from its effects. Poisonous snakes seem to be insensible to their own venom or to the venom of others; but Sir Joseph Fayrer believes that the poison of the more virulent snakes takes effect on snakes of a less deadly character; for example, the poison of the *cobra* or *daboia* will kill the *bungarus*. The non-venomous snakes die rapidly; for example, the *ptyas*, a large, vigorous, and fierce snake (but non-venomous), succumbs within an hour or so to the bite of a *cobra*.

The flesh of an animal dead from snake poison does not seem to be rendered unfit to eat. Animals and man may eat it with impunity. So may they also eat or swallow the venom. Pigeons have been fed with rattlesnake poison, day after day, in doses sufficient to have killed forty, had the venom been injected under the skin. When the excreta has been collected, and the contents of the intestines removed, after killing birds so fed, and after using the chemical means which would separate any venom existing, and using the fluid thus obtained to inject other pigeons, no

injury follows,—showing that the poison by which the pigeon was fed has disappeared. It had been altered and rendered harmless by the process of digestion. On the other hand, the blood of an animal killed by snake poison is itself poisonous; and if injected into an animal, rapidly manifests its poisonous effects. Sir Joseph Fayrer has thus transmitted the poisoned blood through a series of three animals with fatal effect. [Query: “Does it multiply its virulent properties in the blood?” See page 370, *ante*.] “What,” says Sir Joseph Fayrer, “can more forcibly illustrate the extraordinary virulence and potency of the venom of the cobra than this experiment? A few drops of the blood of a dog poisoned by a cobra, diluted with water, and injected into a fowl’s thigh, killed the bird in scarcely five minutes. Thus diluted and mixed with blood, the quantity must have been excessively minute.” Sir Joseph Fayrer has had no opportunity of studying the local effects of the poison, for death has always occurred so rapidly that there has been no time for secondary or local changes. With regard to the *cobra* venom, Dr. J. Ewart notes, in the case of a dog bitten by a full-grown spectacled cobra, when death took place in thirty-two minutes after the bite, there was “evidence that it took place through the nerve-centres, the heart continuing to beat after the respiration entirely ceased,—which can only be through its own inherent irritability and its own ganglionic supply, and quite independent of the medulla, which in all other respects was *hors de combat* from three to four minutes before the heart actually ceased to pulsate.” This impression on the nerve-centres is doubtless made through the medium of the circulation of the blood, and is not due to any organic changes in the germinal matter or cell components of the blood; but the change in the blood seems to be of a much more subtle character than can be detected by the microscope—not dependent on any structural change which can be seen by the very highest magnifying powers. Such is the experience of Sir Joseph Fayrer, Drs. Ewart, Macnamara, Douglas Cunningham, and Mr. Sceva, of Indian experiences, in opposition to the statements of Professor Halford, in Australia, regarding the venom of the *cobra* effecting blood-changes, which have been described at pp. 409, 410.

When death is three or four hours after the bite of a *cobra*, the whole venous system is distended with blood; but sections of the brain through the central ganglia are pallid in the extreme, and scarcely a vascular point is to be seen. Locally, a coagulum marks the entry of the fang, and surrounds the ecchymosed parts; the tissues, also, are discoloured, from rapid local death of the parts and commencing decomposition. Blood coagulates firmly in all the veins, within an hour after being opened; but, microscopically, the blood was not unnatural. Such were the appearances seen in a pig—an animal not so susceptible as a dog to the poison, the former dying in three hours, and the latter in less than an hour after similar doses of the venom. In another instance (that of a mare), *ante* and *post-mortem* firm coagula existed in the heart and large vessels as well as in the veins,—death occurred in less than an hour and a half (eighty minutes).

The venom of the cobra is a slightly viscid, somewhat opalescent fluid, clear when pressed out of the poison-gland, but becoming slightly turbid afterwards, and with a slightly acid reaction.

With regard to the venom of the *Daboia Russellii* (the large viper of India), it seems as if anæsthesia was the first effect produced on the bitten limb and part. In a dog which had obviously got very little venom, where death was slow (eight hours after bite) and painless, a gentle lethargy seemed to steal over the animal, which gradually increased till death. There was no sign of pain—no convulsions; but just before death, defecation was of a muco-sanguinolent character. Ecchymosis surrounds the wounds, and discoloration by dark bloody fluid, with coagula corresponding to the points where the fangs have pierced. Blood, watched for fourteen hours after death, did not coagulate, but remained fluid; but under the microscope presented no change from the normal condition. In another case, that of a large powerful dog (experiment No. 9), where death took place nearly three hours after the bite, the venom caused profuse mucous discharge from the stomach, and blood and mucus from the bowels. The blood, on examination after death, showed the corpuscles shrivelled and collapsed, but not otherwise changed. In other instances there was no tendency of corpuscles to run together. The blood appeared to be dead—to be in a state of necræmia (*Edinburgh Monthly Medical Journal*, May, 1869, p. 1001). Convulsions do not occur when death is slow and the dose of venom small in amount; but they form a marked feature in rapid cases where, the *daboia* being vigorous, the venom is abundantly distilled from the fangs into the wound. In the case of a horse bitten by a *daboia*, death was slow, and life seemed to be dozed away until just at the last, when a few unconscious plunges terminated existence. The cardiac cavities were empty of blood; blood generally fluid, while the lungs and other viscera were congested. The first effect of the *daboia* venom on the nervous system is much more violent than that of the *cobra*. Paralysis follows more quickly, but actual death is longer—consciousness being annihilated early.

With regard to the venom of the *bungarus*, it is neither so deadly nor so active as the *cobra*. Blood flowing from the wound is thin and watery. The bitten part is discoloured, and when pressed, gas escapes in bubbles, evidence of decomposition setting in rapidly. In a dog that died in three hours and forty minutes after being bitten, and the body of which was opened an hour after death, the blood looked dark and grumous, and was imperfectly coagulated the following day. Microscopically, it was examined by Dr. Douglas Cunningham, and exhibited characters quite different from specimens (of *cobra* and *daboia* victims) which Dr. Cunningham had before examined. The reaction was faintly but permanently acid. The red corpuscles were in irregular masses, and had lost all distinctness of outline, and become, as it were, semi-fused. The colouring matter had dissolved out, dyeing the serum and white corpuscles brownish. The white corpuscles were in some places in large masses, visible to the naked eye; and the most remarkable thing about them was their extensive distension. Dr. Cunningham adds, that it would be interesting to know if the reaction was acid immediately after death. In some other cases of *bungarus* bites of dogs, the blood passed into a state of excessive crystallisation of a needle-like and long tubular form.

With regard to *Antidotes*, it is a well-known saying, that “diseases for which there are numerous remedies are either very mild or very

fatal." Sir Joseph Fayrer bases his opinion on sound pathological experience when he says, "I am sceptical on the subject of antidotes, and fear that the hopes of those who are most sanguine on the subject will not be realised." It appears to be impossible to overtake the venom and neutralise it when once in the circulation, however rapid may be the inoculation of the supposed antidote. Sir Joseph Fayrer's well-devised experiments clearly demonstrate this. He does not deny that a man, or an animal, when poisoned by snake venom, may be benefited by the use of stimulants; but such is very different from any such stimulants being an antidote in the sense of neutralising the poison. Nothing can demonstrate more strongly the extremely subtle and virulent nature of the cobra venom than some of Sir Joseph Fayrer's experiments; such, for example, as when a full-grown dog bitten in the forearm, a ligature was instantaneously tightened round the limb above the bitten part, and a pointed steel, heated to a red heat, inserted into the bitten wounds within one minute after the bite, and thus thoroughly cauterising them. Ammonia was also at once injected into the venous circulation, yet in forty-three minutes the dog was dead.

Of such experiments, writes Sir Joseph Fayrer, "nothing is more significant of the improbability of anything proving to be an antidote. If the poison finds entry into the blood-vessels, and be carried to the nerve-centres, I am inclined to believe that nothing can prove of any avail, excepting in those cases where the bite is imperfect, the quantity or quality of the poison diminished or deteriorated, or the snake itself is young, weak, exhausted, or is one of a less poisonous family; such, I believe, are the only cases in which recovery occurs through the inherent vigour of the animal or person bitten, perhaps aided by stimulants and excitement. The favourable result is attributed, and naturally enough, by those who do not understand the *modus lædendi* of the venom, to the treatment and so-called antidote. That we can aid in such recoveries, and that we may do much to help the sufferer through the troubles arising from general disorder and secondary blood-poisoning, I have no doubt; and I would offer every encouragement to all to persevere in this attempt; but I must state my conviction, that nothing that can properly be called an antidote to *cobra* or *viper* venom exists; and the more this is known the better; for mistaken notions on such an important matter can only do harm, and may be the cause of losing, rather than of saving life. My belief is, that if an animal, and probably a man, be fairly bitten by a fresh and vigorous *cobra* or *daboia*, it or he will inevitably succumb, unless some immediate and direct method of averting the entry of the poison into the circulation be practised. That such may be done, I will not deny; but the experiments recorded, performed with the greatest care and speed by two surgeons accustomed to such operations, show that, at the least, it is very difficult. The moment of time that intervenes between the injection of the snake poison by the powerful maxillary muscles, through the tube-like fang into the minute blood-vessels of the part, and the application of the ligature and actual cautery, is sufficient to allow of the entry of the poison into the circulation, and thus reaching the nerve-centres, even in a small quantity, may prove fatal. The ligature is evidently very unreliable when applied to large parts of the body, such as the limbs; for it is almost physically impossible to compress the part so tightly as to

stop the circulation; and unless this be done to the depth of the penetration of the snake's fangs, it is obvious that it can only be of very partial effect in preventing the entry of the poison. On a finger or a toe, the ligature might be of more service, as the smaller part might be thoroughly strangulated; but unless the ligature were applied immediately, it is obvious that it would be useless even there, for the poison would have already entered, and be on its course towards the nerve-centres. How quickly this occurs is proved by those experiments in which the poison was injected directly into the jugular vein. What took place there with the hypodermic needle inserted into the jugular vein has its exact counterpart in the case of the *cobra's* fang inserted, as it must be, when it penetrates a vascular part, into the minute veins. The same may be said of the actual cautery. Unless the hot iron enter the puncture directly after the fang has been withdrawn, the poison is already far on its way towards the nerve-centres; and the burning, though it destroys the tissues, and such of the poison as may not have entered the circulation, can have no influence on that which is already beyond its reach. But as the ligature, if tightly and quickly applied, and the actual cautery, if promptly and thoroughly inserted, must limit to a certain extent the entrance of the poison, both should be had recourse to as speedily and efficaciously as possible, in the hope that the amount of poison left to find, or that may have already found its way into the system, may be less than sufficient to cause death. To conceive of an antidote to snake poison in the true sense of the term, one must imagine a substance so subtle as to follow, overtake, and neutralise the venom in the blood, or that shall have the power of counter-acting and neutralising the deadly influence it has exerted on the vital forces. Such a substance has still to be found; and our present experience of the action of drugs does not lead to hopeful anticipations that we shall find it. But I repeat, that where the poisonous effects are produced in a minor degree, or when the secondary consequences are to be dealt with, we may do much to aid the natural forces in bringing about recovery. This is not, however, what is meant by an antidote" (FAYRER, *Ed. Med. Journ.*, March, 1870, pp. 818-820).

The changes which the absorbed virus of specific diseases undergo in the living and infected body are,—(1.) Increase; (2.) Transformation; (3.) Combination; and, (4.) Separation or excretion. Thus they appear to differ from venoms of animals in these physiological respects. The increase of the virus is shown in such inoculable diseases as *vaccinia*, *glanders*, *malignant pustule*, *syphilis*. In all of these diseases the inoculation of the minutest portion of virus is followed by the formation of one or more vesicular structures containing fluid, from which virus, similarly and equally potent, is produced in millionfold quantity (page 370, *ante*). Thus the virus of any contagious disease developed in an infected person may render his exhalations capable of similarly affecting thousands of other people. And it is probably among azotised materials chiefly that morbid poisons, whether of animal origin or of disease, find the means of their increase (CARPENTER, PAGET, SIMON). The transformation of the virus is indicated by the successive phenomena which supervene during the continuous course of a specific disease. For example, syphilis is followed by a series of secondary and tertiary phenomena, which follow, on the whole, a uniform course in a great variety of patients; so that these

regular syphilitic phenomena may be attributed to the *transformations* of the morbid poison, while the irregularities of the phenomena may be ascribed to constitutional peculiarities of the patient, either natural or acquired from treatment. Thus there are periods of incubation, of development, of maturity, and of degeneration in the material of virus; and the various phenomena which constitute the *symptoms* and *prodromata* of the disease correspond to such periods of transformation; while the increasing disturbance of the general health probably implies that the morbid poison is increasing while it is being transformed—that it grows or multiplies with its development. The *combination* of a morbid poison with some normal material of the blood is indicated by the circumstance, that when the same specific disease, produced by the inoculation of the same matter, affects many persons, the disease set up in each of them may present different peculiar features. The disease may have some peculiar and varied methods of expressing its development in different persons—“*personal peculiarities*,” as Sir James Paget calls them, and which he considers due, in some measure, to the combination of the virus with one or more of those normal materials of the blood which have in each person a peculiar or personal character. By such combinations the following characters of specific diseases may be explained, namely,—(1.) Changes in the disease, by transmission from one person to another; (2.) Some varieties of syphilitic sores, and varieties of their consequences in different persons inoculated from the same source; (3.) The change in the forms of secondary syphilis in transmission from parent to offspring. The *separation* or *excretion* of the virus may be accomplished in many different ways, and may be regarded as the final purpose of the morbid process, or the continuance of illness or disease. It is evident in the inoculable products of some vesicles and pustules.

In all of these *enthetic* disease-poisons, the immediate or the ultimate effect of the poison is to induce deterioration of the blood, and at the same time the poison seems to multiply itself, or to increase in power by some mode not quite well understood, which has been likened to the *zymotic* action which is known to take place in fermentation (page 373, *ante*). The process by which the poison is multiplied, or by which its virulence or strength is increased, varies much as to the time required for its completion. Some of the more intense and virulent poisons, such as that of the most venomous serpents, produce their deleterious and perhaps fatal effects in as short a time as it takes the blood to complete a circulation. The change in the blood at once commences, although incapable of recognition microscopically, and death rapidly follows. This sometimes happens with some forms of the *cadaveric poison*, as that which results from wounds received in the dissection of virulent cases of puerperal fever. Other poisons do not exert their pernicious influence till after a tardy process of incubation, the time of which is not constant, as in *hydrophobia*. In a third class of disease-poisons, a double process of the zymotic-like action seems to take place before the full effects which the poison is capable of producing are completed. The syphilitic poison is an example of this. The multiplication of this venereal poison, and its effects upon the system, seem to become developed during the existence of the hardening process which surrounds the *infecting* venereal sore. This is the first zymotic-like process, and is

attended with a local papule, and perhaps an ulcer. From this local sore the system becomes contaminated; and in the blood a second process (of zymosis?) appears to be completed, by which the original poison becomes intensified, its pernicious influence more complete, and its specific secondary and tertiary effects are then fully developed.

Many of the diseases implanted by specific poisons claim the attention rather of the Surgeon than the Physician, and therefore they may be considered as not properly coming within the scope of this Text-book of Medicine. But for the sake of the pathological doctrines they illustrate, also because of the importance of their effects upon the system, and in relation to other diseases, some of them will be described.

Under the head of "Poisoned Wounds" are the following *varieties*; namely,—(a.) By venomous animals, comprehending snakes, scorpions, and stinging insects; (b.) By animals having infectious or communicable disease, comprehending *glanders*, *farcy*, *equinia mitis*, *malignant pustule*, *hydrophobia*, *cow-pox*; (c.) By dead animal matter; (d.) By morbid secretions; (e.) By vegetable substances, comprehending *arrows poisoned by Wourali*, and subcutaneous injections of vegetable substances; (f.) By mineral substances.

CHAPTER VI.

ON THE NATURE OF MALARIA AND OF MALARIOUS FEVERS.

THREE varieties, forms, or types of malarious fever are understood to exist, having many essential features in common. These are, *intermittent fever* or *ague*, *remittent fever*, and that variety of *yellow fever* characterised by periodicity of febrile recurrence. There are also *masked*, *pernicious*, and *algide* forms, and a *cachexia paludinea* to be recognised. The specific or contagious form of *yellow fever* is now understood to be a different fever from remittent and intermittent fever; and it is treated of separately in this Text-book as a continued fever *sui generis*. The malarious form of *yellow fever* is, on the other hand, the same in kind as the *intermittent* and *remittent fevers*, but varies greatly in the extremes of its severity. So great, indeed, are the differences induced by the common malarious poison, that "if any one had seen only the milder forms of *remittent fever*, and had no opportunity of tracing up its several grades, he might well believe, when he saw suddenly the severest variety, that he had before him a distinct affection" (ALISON)—a belief entertained by not a few. All of the malarious fevers, however, which are now about to be considered, are similar pathologically. They all take their origin from terrestrial aëriform emanations, or from malaria-saturated drinking water—causes which are sometimes rendered more active or dangerous in connection with human beings congregated together, and in certain relations as to physical climate, and particularly as to temperature. In these forms of fever a malarial poison of an unknown kind, generated in paludal regions or littoral districts, is absorbed, and affects the blood, as

cholera, *typhus*, and other specific poisons do. The poison, in the absence of any better name, is known as "*malaria*"—literally, "bad air." No exact knowledge has yet been obtained as to its nature. No *poisonous* principle has yet been *chemically* demonstrated in the air of malarious regions. But many other acknowledged disease-poisons are in a similar predicament as to visible proofs of their substantial existence; and the general impression with regard to *malaria* is, that it is presumed to exist in the atmosphere of certain regions. "The most important organic impurity of the atmosphere is *malaria*; and when a climate is called *unhealthy*, in many cases it simply means that it is *malarious*" (PARKES). The air of typical marshes usually contains an excess of carbonic acid, amounting perhaps to '6 or '8 or more per 1000 volumes. Watery vapour is usually in large quantity; sulphuretted hydrogen, also, if the marsh contains sulphates, which in presence of organic matters are converted into sulphurets, from which S_1H_2 is derived by the action of vegetable acids. Carburetted hydrogen is also often present, and occasionally free hydrogen and ammonia, and, it is said, phosphuretted hydrogen. Indeed, malaria poison has been considered altogether gaseous; and Toropoff of St. Petersburg, after removing water, oxygen, and carbonic acid, found that marsh air still yielded 84 to 89 per cent. of gaseous matter, while hill air gave only 81. Organic matter also exists in considerable quantity, and seems to have much the same character always. It blackens sulphuric acid when the air is drawn through it; gives a reddish colour to nitrate of silver; has a flocculent appearance (sometimes a peculiar marshy smell); and, treated with soda-lime affords evidence of ammonia. (VANQUELIN DE LISLE, MOSCATO, BOUSSINGAULT, GIGOT, BECHI—quoted by Dr. PARKES). The amount in Bechi's experiments was '00027 gramme in a cubic metre of air—i.e., = '000118 grain in a cubic foot. Ozone led through a solution of this organic matter did not destroy it. Besides this organic matter, various vegetable matters and animals floating in the air are arrested when the air of marshes is drawn through water or sulphuric acid; and *débris* of plants, infusoria, insects, and even, it is said, small crustacea, are found. The ascensional force given by the evaporation of water seems, indeed, to be sufficient to lift comparatively large animals into the air. Dr. M. P. Balustra has described spores and sporangia of a little algoid plant in the air of Rome and its vicinity; and the same plant is found abundantly in the waters of the marshes near Rome. Balustra is therefore inclined to attribute marsh fever to this widely diffused "microphyte granule." Dr. Salisbury of Ohio has also attempted to trace ague to a *Palmella*; and others ascribe it to the *Oscillarinæ* generally (PARKES' *Hygiène*, pp. 111–116, Fifth Edition, edited by Dr. DE CHAUMONT). But even although no one has seen *malarious spores*, there is evidence of the existence of some subtle, invisible, imponderable element—the product of the decomposition and decay of vegetable matter under certain conditions of heat and moisture. It affects the human system through the lungs or through the stomach by drinking water. Its immediate and direct action is upon the nervous system, which it powerfully depresses and paralyzes, especially that of the great sympathetic system, followed by diminished action of the heart and arteries. The human system is the only field for the action of this specific poison, the existence of which becomes a matter of inference, by the

definite series of disorders which are produced by it. The phenomena are varied but constant, so that by induction we are enabled to attribute their occurrence to malaria. However varied may be the phenomena, one effect is constant—namely, the characteristic intermittence which marks the fever—an intermittence which is at once the stamp and original form of all malaria infection. As soon as malarious fever shows itself, it sets in as an intermittent, however varied and different may be its ulterior manifestations.

The now well-known factors of malaria poison are—vegetable decomposition, moisture to saturation, subsoil, and water almost at the surface, and a high temperature, heat above 60° Fahr. If the land is perfectly dry, or perfectly flooded, or frozen, the poison, if generated, is not disseminated. It is believed to be a *material* poison, because there is evidence that it may be wafted along with the wind, and so induce fever at a distance from the place where the poison is generated. It “can certainly be carried for many hundred yards, even sometimes for more than a mile, in an upward direction (up a ravine, for instance) or horizontally, if it does not pass over the surface of water” (PARKES). It may also be intercepted by a belt of trees. It appears, however, to be most intense near the surface of the ground. Thus the intimate connection between marshy districts and certain forms of disease is established by a great amount of direct and indirect testimony; but two questions still remain—namely, What is the nature of the noxious agent of malarious districts? and, What circumstances are necessary to its formation or extrication? It seems certain that the deleterious agent is neither heat alone, nor moisture alone, nor any known gas extricated from the marsh. It cannot be *heat* alone, for many of the hottest parts of the West Indies are free from fever. It cannot be *moisture* alone, for no persons enjoy better health than the crews of clean ships at sea, even when cruising in tropical climates, as long as they have no communication with the land. While *carbonic acid*, *azote*, *oxygen*, or *carburetted hydrogen*, the gases collected by stirring the bottom of marshes, have all been inspired without producing any disease similar to paludal fever; it seems to follow almost as a necessary consequence, that the remote cause must be a *miasm*, *poison*, or *malaria*, whose presence is solely detected by its action on the human body. The general evidence in favour of vegetable decomposition being the remote cause is, that all countries are for the most part free from paludal diseases while the crops are growing, and only become unhealthy after the harvest, when large quantities of vegetable matters are left on the ground at the time the rain begins to fall. Marshes are in general healthy till the summer's sun has diminished their waters, and bared a greater or less portion of their bed. The part thus exposed almost always contains a large portion of vegetable matter, which, running into rapid decomposition, generates other vegetable growths of a fungoid nature especially, which may be or may convey the poison which gives origin to this class of disease. It is during the periods of the year when the drying process is in greatest activity that unhealthiness prevails with greatest severity in the East Indies—namely, before the commencement and after the termination of the rainy season. The particular evidence of vegetable decomposition being the source of the poison is of

the following nature :—Lancisi gives the history of an epidemic which for several summers infested, and almost depopulated, the ancient town of Urbs Vetus, situated on an elevated and salubrious part of Etruria, and which was traced to the circumstance of the peasants steeping their flax in some stagnant water in the neighbourhood of the town. This practice was therefore prohibited in 1705, and the epidemic ceased to appear. The steeping of flax being productive of paludal fever is a fact the knowledge of which is not limited to Italy; for the ancient as well as the new "*coutumes*" of almost all the provinces of France have proscribed the steeping of flax—"la rouissage"—even in running waters, from the fear of infection. In the Netherlands the same belief has prevailed. In July, 1627, the King of Spain passed an ordinance prohibiting the steeping of flax in the streams and canals of Flanders. The prohibition may be explained on another ground—namely, because the flax poisons the water and kills the fish. In Ireland, and perhaps also elsewhere, steeping flax in a running stream is therefore forbidden by law. The experience of the indigo-planter is to the same effect. In India, after the colouring matter has been extracted from the indigo plant, it was formerly the custom to throw the detritus into large heaps or masses in the immediate neighbourhood of the works, which, at the end of three or four years, became manure of an excellent quality. It was found, however, that these heaps, wetted from time to time by the heavy rains, and afterwards heated by the rays of a burning sun, rapidly decomposed, and at length emitted *miasmata*, which produced all the effects of those extricated from the marsh. The workmen who lived near, and more especially those to leeward of these masses, were found to be very commonly attacked by fever, chiefly of the remittent type, and similar to those which prevail in the paludal districts of that country. This consequence is now so well established, that the most intelligent indigo-planters no longer allow these heaps to be formed either near the works or in the immediate neighbourhood of the cottages of their workmen. Ships also afford additional evidence of the truth of the hypothesis of vegetable decomposition and growth being the remote cause of malaria. All intertropical regions, where the nature of the locality admits only of a rice cultivation, are well known to be unhealthy.

These facts render it highly probable that the noxious agent must be a product of vegetable development, growth, and propagation, evolved on the soil, and moving in the lower regions of the atmosphere. If we consider the paludal poison to be a product of vegetable decomposition, or of vegetable growth, it follows that heat and moisture, quantity of vegetable matter, and nature of the soil, though not the essential agents, must have a sensible influence on its formation, must vary its intensity or quantity, and also must limit paludal diseases to particular localities, seasons, and latitudes. A certain temperature, for example, under certain conditions as to moisture, is evidently necessary to its extrication and development. It is certain also that a given quantity of moisture is as necessary to vegetable decomposition or growth as a given temperature, and that the extrication of the paludal poison will be most abundant from that soil which contains no more moisture than is necessary for that process; for an excess in quantity, by dividing and separating the

particles, and by preventing the access of atmospheric air, will either retard or altogether put a stop to putrescency. Hence in some countries frequent and heavy rains will render marsh fevers prevalent, by saturating the whole of the open country; while privation of rain will in others produce exactly the same effect in other instances, merely by diminishing the superfluous quantity of water. Thus, in the West Indies, an uncommonly rainy season seldom fails, in the perfectly dry and well-cleared Island of Barbadoes, to induce for a time general sickness; while at Trinidad, whose central portions are described as a sea of swamp, and where it rains nine months in the year, an excess of moisture is a preservative from sickness; for should at any time rains fall only eight months in the year instead of nine, the swamps become dry and bared to the sun, and remittent fevers of the worst kind are sure to make their appearance. The same result follows on the subsiding of the waters of rivers that have overflowed their banks, as those of the Nile, the Rhone, the Danube, the Tigris, the Ganges, and many others. From these data it appears that the swamp, on its approach to dryness, is the source of disease and death; while an excess of rain has a preservative power so long as moisture is in excess. On the contrary, on the rich and dry plains, and even on the hills of tropical countries, rain is the cause not only of vegetable decomposition, but also of disease; while absence of rain tends to preserve health. In estimating, however, the dryness of a country, its superficial appearance is often deceitful. In the years 1748 and 1794 the summers were dry, and our troops took up the encampments of Rosendaal and Ousterhout in South Holland. The soil in both places is a level plain of sand, with a perfectly dry surface, and where no other vegetation existed, or could exist, but a few stunted heath plants; yet in both years fever became epidemic among the troops in each place. On digging for water the cause was discovered, for the soil was found to be saturated with water to within a few inches of the surface. It is probable, therefore, that the country was originally formed of vegetable and other detritus, brought down by the Rhine and the Waal, and afterwards covered with sand thrown up by the sea, which, heated by the summer's sun, became the powerful cause of the extrication of marsh miasmata. From the exceeding malignity of the salt marshes, it has been supposed that a mixture of salt and fresh water renders a marsh more pernicious than either of them alone. It has been found, however, that on coasts where these marshes have been kept up to one uniform level by means of floodgates, the surrounding country is healthy; and it has therefore been inferred that the sickness produced is a consequence of the perpetual alteration of the level of the waters of the marsh, and not owing to the admixture of sea and spring water.

It is probably owing to a great excess of temperature that rocky countries, as Gibraltar and the Ionian Islands, are so often and so severely attacked with malarious fever. It is on the summits of these rocks that springs arise. The slightest frost produces fissures, into which fungi, as "*mould*," and other vegetable matters, insinuate themselves, while the bare rock becomes heated to an intense degree. Humboldt, on ascending the Orinoco, found the station at the great fall depopulated by fever, which the natives attributed to the bare rocks of the rapids. He determined the heat of these rocks to be 118.4° Fahr., while the temperature of the

air immediately around was only 78·8° Fahr. Again, the rock of Gibraltar is known to be percolated with water, so that we can hardly conceive a more pestilential focus of disease when the causes necessary to the formation of miasm or fungi are combined. The existence of paludal fever in dry and rocky districts, therefore, although it may appear extraordinary and unexpected, is not necessarily an exception to the general law of paludal diseases being generated by miasmata, the result of vegetable decomposition or of vegetable development. In many hot climates the most deadly sites for encampments have been the dried-up beds of rivers, or their immediate vicinities (MARTIN).

These facts seem, therefore, unquestionably to prove that heat and moisture, though not the primary cause of paludal disease, are conditions essentially connected with the development and specially putrefactive changes of vegetable growths and of the noxious miasmata, and consequently furnish additional argument in favour of the hypothesis of vegetable decomposition generating the remote cause which produces or propagates miasmatic disease. It is certain, however, even when the conditions of heat, moisture, and vegetable matter most abound, that paludal diseases do not always assume their severest forms; and there seems reason to believe that differences of geological formation, by favouring or otherwise influencing vegetable putrefaction and growth, may variously affect the health of countries similarly situated in other respects.

The belief that *malaria* can spread through the agency of drinking water as well as by air, is of very ancient origin; but has evidently been overlooked of late, since Lancisi fixed attention on the air of marshes as the sole source of malaria. Dr. Parkes, however, brings us back to the days of Hippocrates for a statement of the fact that the spleens of those who drink the water of marshes become enlarged and hard; and Rhazes not only asserted this, but affirmed that it generated fevers. It has also been a very general belief among the inhabitants of marshy districts, that the water could produce fever. The Cinghalese attribute fevers to impure water (HENRY MARSHALL); and Dr. Parkes states that on making some inquiries of the inhabitants of the highly malarious plains of Troy, during the Crimean War, he found the villagers universally stated that those who drank marsh water had fever at all times of the year, while those who drank pure water only got ague during the late summer and autumnal months. The same belief is prevalent in the south of India; and in Western Candeish, Canara, Balaghut, and Mysore, and in the deadly Wynaad district, it is stated by Mr. Bettington (*Indian Annals*, 1856, p. 526) of the Madras Civil Service, that it is notorious that the water produces fever and affections of the spleen. He gives some extraordinary evidence on this point—such as, villages placed under the same conditions as to marsh air, but in some of which fevers are prevalent, in others not; the only difference is that the latter are supplied with pure water, the former with marsh or nullah water, full of vegetable *débris*. In one village there were two sources of supply—a tank fed by surface water and marsh, and a spring; those only who drank the tank water got fever. In a certain village (Tulliwaree) no one used to escape the fever till Mr. Bettington dug a well, when the fever disappeared; and during fourteen years it has not returned. Another village (Tambatz)

was also "notoriously unhealthy;" but a well was dug, and the inhabitants became healthy. In Marseilles, paroxysmal fevers formerly unknown have made their appearance since the supply to the city has been taken from the canal of Marseilles. The natives of India, too, have a current opinion that the use of river and tank water in the rainy season (when the water always contains much vegetable matter) will almost certainly produce fever; and Dr. Townsend believes that in this way the prevalence of ague in dry elevated spots is often to be explained. The people also who use water of streams draining forest lands and rice-fields suffer more severely from ague than the inhabitants of the open plain drawing their water from a soil on which wheat grows. In the former case there is far more vegetable matter in the water. The Upper Godavery tract is said to be the most aguish in the province, yet there is not an acre of marshy ground; but the people use the water of the Godavery, which drains more dense forest land than any river in India. In the "Landes" of South-west France the water from the extensive sandy plains contains much vegetable matter, obtained from the vegetation which binds together the siliceous particles of the subsoil. It has a marshy smell, and produces intermittents and visceral engorgement (FAURÉ). In the march from Massowah to the highlands of Abyssinia, Dr. Blanc and Mr. Prideaux, who drank water only in the form of tea or coffee, escaped fever, while others, less careful, suffered. In this country similar facts have been noticed. More than twenty years ago Mr. Blower of Bedford mentioned a case in which the ague of a village had been much lessened by digging wells; and he refers to an instance in which, in the parish of Houghton, almost the only family which escaped ague was that of a farmer who used well-water, while all the other persons drank ditch water (SNOW). At Sheerness, the use of ditch water, which is highly impure, from vegetable *débris*, has been considered to be one of the chief causes of the insalubrity of the district; and the late Dr. Parkes suggested the question,—“Is it not possible that the great decline of agues in England is partly due to a purer drinking-water being now used?” Formerly, there can be little doubt, when there was no organised supply, and much fewer wells existed, the people must have taken their supply from surface collections and ditches, as they did till lately at Sheerness. Surgeon-Major Faugut also records of the artillery quartered at Tilbury Fort (in the Gravesend district), that they have generally suffered more or less from ague; whilst the people at the railway station, and the coast-guard and their families in the ship lying just outside the fort, never suffer from malarious poisoning. The troops were supplied with drinking-water from two underground tanks, which received rain-water from the roof of the barracks; while the other persons mentioned drew their drinking water from a spring near the railway station. From December, 1873, to July, 1874, the troops were supplied from the same source, on account of the barrack tanks being out of repair, with the result of a marked diminution in the number of cases of ague. An analysis of the water showed that the tanks were exposed to soakage from the surrounding salt marsh, for the so-called rain-water yielded 41·3 grains per gallon of total solids in the one case, and 145·25 in the other—the chlorine being respectively 12·8 and 33·9. On the other hand, the station water gave

38 grains total solids, and only 3·3 of chlorine. As regards organic matter, the tank water showed actually less impurity than the station water, by the ammonia method; but by the permanganate method, they were three times as impure (*Army Medical Reports*, Vol. XVII, p. 212; PARKES' *Hygiène*, Fifth Edition, pp. 44–46). It is therefore quite clear that the infection of malaria may be conveyed into the system, as much by the stomach imbibing marsh water, as by the lungs inhaling marsh air.

By numerous observations it has thus been established that some aëri-form material (probably of an organic vegetable nature, but specifically poisonous) is exhaled from marshy or wet grounds in the progress of drying, which is capable of impregnating water as well as air. Agues have always been observed to be the diseases of moist or marshy districts; and to prevail most in low, swampy, and humid countries, where seasons of considerable heat occur. The vicinity of marshes, or of a district that has at some recent time been under water; the banks of great lakes, and the shores of great rivers and seas, where the water flows slowly, and in some places stagnates, in shallow rivers, over land alluvial, low, and flat; extensive flat tracts of wood, where much moisture is constantly present, where the process of drying is uninterrupted, and yet the surface constantly exhaling humidity,—these are some of the terrestrial physical conditions in which the paludal and the littoral fevers are found to abound. It must also be admitted, however, that these diseases do not prevail in *all* marshy districts, and they cannot, in some cases, be traced to a residence in the vicinity of marshes.

The concurrence of circumstances under which paludal and littoral fevers have been observed to become developed may be shortly stated as follow:—

- (1.) A certain degree of heat. A high temperature is especially favourable to the production of malaria, and the more so when acting on moist alluvial soil.
- (2.) A certain relation as to season, variable with the geography of the locality in which such fevers prevail. The season of the year most marked in tropical climates is that which immediately succeeds the cessation of the rains, or, as it is called, “the drying up of the rains.”
- (3.) Low swampy grounds and extensive rice-fields are well-known sources of malaria. In such districts clouds of mist are often seen, wafted along the earth's surface for miles; and it is believed that *malaria*, whatever be its nature, clings to such mists. And, although it has been observed that absolute marshes do not always produce agues, nor that agues are always due to obvious marshes, yet it is generally found that in districts where such paludal fevers abound the surface is porous, penetrable, and yet retentive of moisture, although it does not appear on the surface of the ground; that the district had been at one time submerged, and that it continued slowly but constantly to undergo the process of desiccation; or while at certain seasons it imbibes moisture from local or meteorological sources, at other seasons it undergoes the drying process under intense solar heat. Such are some of the most sickly and febriferous districts in Europe, India, and America. For example, the Maremma of Italy; the sandy shores of Walcheren; the parched water near the Douro, so fatal in the Peninsular War; the district of the lakes near Varna, in Bulgaria, so fatal to our soldiers during the Russian War of 1854–56; and many parts in the south of Spain; many districts in Burmah; also Hong Kong; and

many newly cleared tracts in North America. In most of these places the conditions of the surface of the ground are very much alike. While no obvious appearance of a marsh exists, the vigour of vegetation is extreme, amphibious animals of the batrachian kind abound, plants and cephalopodous mollusca of notoriously marshy regions find a habitat; and the rich alluvial soil is so imperfectly cultivated that the process of vegetation is not adequately exhausted, and a surface of humid ground is exposed to the solar heat, and so exhales a material which exercises a persistent deleterious influence on the human frame. It is believed that the number of insects and reptiles with which a place abounds, is more significant of its insalubrity than almost any other circumstance; and that a mixture of animal and vegetable matters undergoing decay give rise to miasms much more noxious than those resulting from vegetable matter alone. The drying of all porous soils, from which watery fluid readily evaporated, is the genuine source of exhalations capable of producing the paludal fever; and the febriferous activity of these exhalations is influenced by the character of the season, the moisture, the temperature, and the aerial movements of the atmosphere (FERGUSON, *The Edinburgh Philosophical Transactions*, Vol. IX., p. 273).

The evidence regarding the geological nature of soil as a cause of ague is somewhat conflicting. It is a fact that the usual localities in which paludal fevers abound, are those in which the soil consists of mineral, vegetable, and animal matters, mixed together in such proportions, and of such constituents chemically, as tend to absorb moisture and retain it, and subsequently to decompose. Such soils are known as *alluvial*. Paludal fevers abound, however, where soils of a different nature predominate. Level plains of sand, or dry, loose, open gravel, are soils where malaria fevers have prevailed—e.g., Rosendaal, in South Holland, in 1794, when after a dry and very hot summer our troops in the month of August took up an encampment there.

High grounds near exposed marshes are often more unhealthy than the places immediately adjoining which are on a level with them. Rocky places, such as the black basaltic rocks on the Rio Negro in Peru, Ciudad Rodrigo, Gibraltar, and Malaga, have now and then been ravaged by epidemics of littoral paludal fevers, and the rocky shores and islands of the Mediterranean—for instance, Minorca, Sardinia, Sicily, Cephalonia, Cyprus, and all the Cyclades—abound as much in these fevers as the most level parts of Holland; and the West India Islands, most of which, although coralline rocks, are the native soil of these diseases. Soil composed of tenacious or stiff clay (argillaceous) is highly retentive of moisture, and is difficult either to dry or to drain. The basin of the Thames, comprehending Middlesex, Essex, Surrey, and Kent, is almost entirely clay land, and is the district of England where agues most of all prevailed before drainage became common and efficient,—especially along the banks of the Medway and the Thames. In the days of Sir Gilbert Blane agues had almost entirely ceased to occur in London, and the cases which he treated he believed to have been imported from malarious districts around. The same may be said of those of the present day. An hundred years before the time of Sir Gilbert Blane, however, we find that agues prevailed in situations in the town of London where they are

now wholly unknown, such as Russell Street, Covent Garden, Fleet Street, Fetter Lane, Newgate Street, Paternoster Row, Cheapside, Smithfield, and Fenchurch Street. At the present time ague rarely occurs in London, except on the south side of the river, especially in Bermondsey and Rotherhithe, and chiefly in persons who have recently been exposed to malaria in Kent or Essex, or who have come from marshy districts either quite recently or within a few months. The malarious influence still in the metropolis seems, however, sufficiently powerful to imprint a periodic character upon various local affections, and occasionally to give rise to fevers of a remittent type. Recently (in 1856) such affections have been unusually prevalent; but the forms of ague now met with in London are more tractable and milder than those which formerly prevailed (Dr. PEACOCK). The diseases usually attributed to malaria, which were formerly so destructive, have almost disappeared from this country. The reason of this may fairly be ascribed to the improved drainage both of the towns and of the agricultural districts. The fact may be proved, did space permit; and the practical inference leads one to hope for still more immunity from diseases arising from this source, if the "proper authorities" direct further efforts in that direction. "Within the last half-century land-draining and town-sewering have ripened into sciences. From rude beginnings, insignificant in extent, and often injurious in their first effects, they have become of the greatest importance. Land has, in many instances, doubled in value; and town-sewering, with other social regulations, have not unfrequently prolonged human life from 5 to 50 per cent., as compared with previous rates in the same district." "Agues (and malarious cachexiæ) are reduced. Since 1840 an annual mortality in English towns of 44 in 1,000 has been reduced to 27; an annual mortality of 30 has been reduced to 20, and even as low as 15; and human life has now more value in England than any other country in the world—a result entirely due to better sanitary arrangements" (RAWLINSON, *Journal of Society of Arts*, March 21, 1862, Vol. X., p. 276). Of towns that have been drained and remain healthy there are many examples in ancient and modern history. Hippocrates tells us that the city of Abydos had been several times depopulated by fever; but the adjoining marshes having been drained, it became healthy. London, in the time of Sydenham, was infested with epidemic intermittent fever and dysentery, the mortality from the former alone averaging, in a comparatively small population, from one to two thousand persons annually. In the present day, a case of ague contracted in London is unknown. Many other towns, both of this country and of France, as Portsmouth, Rochefort, and Bordeaux, from being the constant seat of paludal fevers, have been, from the same causes, rendered in like manner comparatively healthy. Dr. Wood, of Pennsylvania, relates an interesting fact regarding the neutralisation of miasmatic effluvia. He tells us they are in some way rendered innocuous by the air of large cities. This fact is notorious in relation to the city of Rome; and it is abundantly confirmed in the larger towns of the United States, in the neighbourhood of which these diseases have prevailed.

It is observed that the surface of the earth may be dried either by the direct rays of the sun, or by currents of hot dry air wafted over it, or

by both combined ; but it is principally by the direct rays of the sun that the deleterious material of the soil is liberated ; and it seems to be at a certain period of this "drying up" process that the exhalations are more potent than at another time in developing paludal fevers. The exposed grounds, after clearing off the copious vegetation from dense jungles, so as to admit the influence of the sun's rays in the "drying up," is known to be a fertile source of malaria. There appears also to be a certain state of the human frame which renders it more than usually susceptible to this disease. The natives of warm and tropical climates are much less frequently and less violently attacked with paludal or littoral fevers than settlers or visitors from other lands. In the Mediterranean, along the coast of Africa, in the East Indies, in the West India Islands, and in the Southern States of the Union, newcomers from the northern latitudes are almost invariably attacked, and suffer much more severely from the fever than those who have been long in the country. It has been also noticed that those who, after residing in a territory where paludal fevers abound, have been out of it for some time, an augmented susceptibility to renewed attacks of the fever becomes manifest on their return. Other causes predispose to those fevers, and none more than laborious or fatiguing duty in military or naval operations, labouring in the sun, exposure to chill, or from great heat suddenly to cold, excess in eating or drinking, intellectual exertion combined with bodily fatigue, and a crowded state of the population. A great deal has been written of late as to the influence of a chill being potent to cause all the phenomena usually described as due to the poison we call malaria. Fever from exposure to cold or "chill" is usually the result and symptomatic of a pneumonia, a bronchitis, or an attack of rheumatism,—not by affections of the spleen and liver, which are peculiar to malaria poisoning. "Chills" and exposure to cold may bring on paroxysms of ague, but only in those who have been already poisoned by malaria ; but they never cause the disease originally. It may also be noted that the daily ranges of temperature are greatest at places where malarious fevers are absent and cold nights are common, than in places like Hong Kong, where fevers are endemic, and sleep nearly impossible from the heated air in the summer months. Indeed, *sunstroke*, or heat-apoplexy, is regarded by some as a form of remittent fever (JOHNSTON, MARTIN, HILL).

A question of much interest in connection with *malaria* has given rise to considerable speculation. It relates to the *varieties* of the malarious poison. Is the poison which gives rise to a *quotidian ague* different from that which produces a *tertian*? and is this, in its turn, different from that which produces a *remittent fever*? and this, again, different from that which gives rise to the malarious form of *yellow fever*? Our knowledge is not yet sufficiently precise and extensive to settle these points. But when we see a large body of men placed under the same circumstances as to food, drink, and clothing, and labour, and exposed to the same causes of disease in the same way and in the same place, some of whom are seized with *quotidian ague*, others with *tertian*, and others with *remittent fever*, the presumption is that the same cause has produced different forms of one disease, according to its intensity, the constitution of the individual, and the predisposing causes to which he may be subjected. Other facts, also,

favour this presumption—namely, *remittent fevers* are known to pass into *quotidian agues*; and these again into *tertian agues*.

Infecting Distance of Miasmata.—As a general law, the danger of infection is in proportion to the proximity to a marsh. But there are many disturbing causes which produce remarkable exceptions to this law. These disturbing causes are, the extent of surface which generates the miasmata, their intensity, the direction of the wind, its force, the season of the year, the time of day, the attracting influence of surfaces over which the miasmata pass, and the existence of ravines as channels or conduits. Dr. Parkes has put the following practical question relative to the *vertical* ascent of malaria:—A marsh or malarious tract of country existing at any point, what altitude gives immunity from the malaria, suppose there is no drifting up ravines? It is well known that even a slight elevation lessens danger, a few feet even, in many cases; but complete security is only obtained at greater heights. At present the elevation of perfect security in different parts of the world is not certainly determined, but appears to be, as to Italy, 400 to 500 feet; America (Appalachia), 3000 feet; California, 1000 feet; India, 2000 to 3000 feet; West Indies, 1400 to 1800, and up to 2200 feet. We know that marshes can be active at an elevation of 6000 feet; but whether the emanations from a marsh will ascend that height without drifting up ravines, has still to be investigated; but 1000 to 1200 feet will generally give security. The Monte Mario, which adjoins Rome, is, according to Breslack, about 165 yards perpendicular height above the Pontine Marshes, and is extremely unhealthy. Tivoli, which is about 230 yards above the level of the same marshes, is infinitely more salubrious; while at Serre, 340 yards perpendicular height, the inhabitants enjoy an entire exemption from the paludal diseases which prevail below. Different latitudes may account for these differences. In towns partially freed from marsh miasmata by extensive drainage, the difference of a few feet perpendicular height makes an almost inconceivable difference in the liability of persons to paludal disease. The barracks of Spanish Town, the capital of Jamaica, for instance, consist of two storeys, or of a ground floor and of a first floor; but it being found that two men were taken ill on the ground floor for one on the first floor, it was at length ordered that the ground floor should be no longer occupied. Dr. Cullen remarked a similar result at Portobello; Dr. Fergusson in St. Domingo; and Sir Gilbert Blane in the expedition to Walcheren. This law is so well understood in the West Indies that in Demerara, and in many other parts, the houses are built on dwarf columns, after the manner of corn stacks, in order that a stratum of air may be interposed between the house and the ground. In Rome, and in other towns of Italy, it is also so well known that the lower rooms of the houses are abandoned, the family occupying the upper rooms, as affording a greater protection from the paludal poison. As regards *lateral horizontal spread*, Levy has recorded his belief that malaria will spread 700 to 1000 feet horizontally from the central point of the marsh. Currents of air will take it greater distances, but seldom beyond one or two miles, unless the air currents are rapid and strong. The precise limits are unknown, but it is very doubtful if the belief in transference of malaria by air currents for 10, 20 or 100 miles is correct.

^ 295 ft

^ 1020 ft

A few precise observations show that the *spread of malaria over water* varies in different countries. The cases least complicated for investigation are those when water alone intervenes between the marsh and the recipient. In the year 1746-47, while our troops lay in Zealand, the sickness was so great among four battalions quartered there that some had hardly 100 men fit for duty, or less than a seventh part of a battalion. In one corps, the Royals, only four men escaped. At the time, however, of this remarkable prevalence of fever on shore, Commodore Mitchell's squadron lay at anchor between South Beveland and the Island of Walcheren, and the fever raged at both places; but, nevertheless, in the midst of all the sickness that reigned around, the seamen were neither affected with fever nor flux, but continued to enjoy perfect health. These observations of Sir John Pringle were fully confirmed by those of Sir Gilbert Blane during the last disastrous expedition to Walcheren. "I had," says this physician, "the opportunity of observing the extent to which this noxious exhalation extended, which was found to be less than was generally known. Not only the crews of the ships in the road of Flushing were entirely free from this epidemic, but also the crew of the guardship, which was stationed in the narrow channel between this island and Beveland. The width of this channel is about 3,000 feet; yet, though some of the ships lay nearer to one shore than the other, there was no instance of any of the men or officers being taken ill with the same disorder as that with which the troops on shore were affected." It appears, therefore, that in Europe the horizontal spread of marsh miasmata over fresh water is less than 3,000 feet. With respect to the spread of the miasmata over salt water, Sir Gilbert Blane wrote that in tropical climates ships at a distance of 3,000 feet from a swampy shore—a distance to which the miasmata did not extend in Zealand—and even farther, were affected with the noxious exhalations. Dr. John Hunter considered a few miles to be a necessary interval for a ship lying to leeward of a swamp, in order to insure a complete exemption from the disease. When, however, the swamp or other source of the poison is of small extent, a much less space is sufficient to assure an exemption. In the epidemic on the coast of Spain, the fisherman living with his family on board his boat has been rarely attacked, though lying at anchor close in shore. Also, during the epidemics at Gibraltar, it was not unusual for the richer inhabitants to hire a Moorish vessel, and to live on board in the bay; and there was scarcely an instance of those persons having been affected, though keeping up a free communication during the day, either directly or indirectly, with the town. The extent to which the marsh miasmata may spread from its source over land in a horizontal direction is a much more complicated question. In China and the West Indies a farther distance is necessary. In China three-fourths of a mile; and in the West Indies one mile has been sufficient (GRANT, quoted by Dr. PARKES, *Practical Hygiene*, 5th Edition, p. 644).

The effect of trees in intercepting the paludal poison is remarkable, and appears to have been known to the ancients, who surrounded their temples with groves, presumedly on account of their protecting influence. Pope Benedict XIV. ordered a wood to be cut down which separated Villatri from the Pontine Marshes, and for many following years there raged

throughout the whole country, and in places never before attacked, a most severe and fatal fever. The same effects were produced from a similar circumstance in the environs of Campo Santo. In the West Indies it is quite wonderful how near the marsh the planter will venture to place his habitation, provided he is protected by trees.

Different soils also affect the transmission of the paludal poison. The spot, for instance, on which the new National Dock and Arsenal at Woolwich are built was a marsh of about 700 acres, and on either side of it are the villages of Greenhithe and of Northfleet. The peculiarity here is, that the inhabitants of these villages rarely suffer from intermittent fever, whilst those on the hills beyond were most greatly afflicted with that disease. Dr. Maton mentions a similar fact in the neighbourhood of Weymouth, and the same circumstance is observed in the vicinity of Little Hampton and the marshy districts in Sussex. It is well known that different soils radiate heat with different degrees of intensity, and consequently are, under the same circumstances, of different temperatures, having very different powers of attracting moisture; and possibly, also, they may have other and more direct properties favourable to the generation of the paludal miasm. Nothing, for instance, is better determined in husbandry than that the *carbonate of lime*, mixed with the ordinary matters of a compost, greatly forwards the processes of molecular decay, so that the mass thus prepared is fit in a much shorter time for the purposes of manure. There are some soils peculiarly favourable to the decomposition and growth of vegetable matters, and consequently to the more abundant extrication of marsh miasmata. It is remarkable also that those countries most celebrated for paludal fevers, have been found very similar in their geological formation to each other, and to those artificial conditions which most favour rapid vegetable decomposition and growth.

To predicate all the facts connected with paludal diseases is not yet possible; for the variations of atmospheric temperature, the changes in the quantity and nature of the electric fluid, the quantity of water, the nature of the soil, the amount and character of the vegetable matters and their growth, form a problem extremely complicated, and one whose smallest variation as to quantity or time may occasion marked differences in the result. As a general rule, however, it may be stated, that in no climate do paludal fevers prevail to an equal degree all the year round. In the winter, much of the vegetable matter has already undergone decomposition and further growth, while the dryness of the season and the diminished temperature are little favourable to its further development. When the spring, however, arrives, and the rain falls, and the heat of the sun increases, the earth again evolves a miasm of mitigated intensity. In summer the products of vegetable decomposition are used up in affording nourishment to the growth of many vegetable forms; and this season, like the winter, is in general healthy. But in the autumn, and after the harvest has been gathered, when the ground is covered with vegetable *débris*, when the rain falls in torrents now and again, and when the solar heat has acquired its greatest intensity, all the conditions for the greatest amount of growth of vegetable matter, of moderate moisture, and of highest temperature, change and are united; so that the season which realises the hopes of the husbandman is also the period of pestilence and of his

greatest danger. There are two other facts, also, which are too prominent to be mistaken. The one is, that the miasmata vary greatly in intensity in different countries, and in different parts of the same country; again, the diseases they produce, though annually *endemic* in certain districts, yet become, in certain years, and from the action of causes not yet determined, *epidemic*. In the same countries, also, it is determined that difference of altitude is equivalent to difference of latitude; and, as a general law, it may be stated that in the Antilles, on the continent of America, from Boston to Rio de Janeiro, and also on the continents of Asia and Africa, while in the low country severe remittent or yellow fever prevails, still in the higher country, though immediately contiguous, the type is changed to intermittent and mild remittent. The circumstance of intermittents passing into remittents, and remittents into malarious yellow fever; and conversely, of remittent and malarious yellow fever often terminating in intermittent—facts observed not only in the East and West Indies, but on the continents of America and of Africa—demonstrate a unity of cause as firmly as the best established facts in medicine. That paludal diseases, like many diseases produced by morbid poisons, are annually endemic, and only occasionally epidemic, is unquestionable. A few years ago intermittent fever was epidemic in particular districts in this country; but of late years the cases of ague have been comparatively rare. In Demerara it has been observed that malarious yellow fever is epidemic about every seventh year. At Gibraltar, although sporadic cases of paludal fever occur annually, still malarious yellow fever is only occasionally epidemic, but so irregularly, that after it assumed that character in 1804, in 1810, in 1813, and again in 1814, from that period the garrison suffered no similar visitation till 1828. The physical causes on which this greater virulence and greater spread of the disease depend are not determined. In temperate climates it has been observed that paludal fevers have been most prevalent when a hot summer has succeeded a wet spring.

The specific action of malaria poison is only made apparent in its effects upon the human body. After a period of latency, more or less long, functional disorders of the great nervous centres are brought about, terminating in the phenomena either of *intermittent*, *remittent*, or *yellow malarious fever*. These fevers may exist without any alteration of structure being set up, and the patient often dies from the severest forms, with hardly a trace of disease being discoverable. In the milder forms of these fevers, however, a greater number of organs and tissues are morbidly altered than perhaps in any other disease, as the liver, spleen, lungs, heart, brain, and the serous and mucous membranes of the body generally. The specific action of the malarial poison, within certain limits, may be said to be in the inverse ratio of the intensity of the fever which attends its action. The affections of the liver and spleen also vary greatly according to the country. In some parts of India the spleen is the organ chiefly affected, while in other districts it is the liver, the nature of the country, perhaps of the soil, impressing evidently some peculiar character on the poison. Patients labouring under intermittent fever in this country generally recover without any manifest derangement either of structure or of function of any organ or tissue. When, however, the disease is neglected, the liver and

spleen almost always suffer, the disordered function of the liver being generally indicated by jaundice; or inflammation of the liver may ensue, of which jaundice may or may not be a symptom; and this inflammation may be acute or chronic, diffuse or limited to one place. If a liver, previously healthy, becomes the seat of diffuse malarious inflammation, it is of the deepest hepatic tint, and loaded with blood. Or we find it greatly hypertrophied, filling the abdominal and pelvic cavities; and, according as the inflammation is acute or chronic, either greatly indurated or so softened as to be easily broken down. In a few instances this inflammation may terminate in abscess, generally of phlegmonous character. On the contrary, if the liver be previously diseased, its colour, even when the seat of abscess, or otherwise most acutely inflamed, may be of the palest yellow, and its texture sometimes so soft and broken down that the larger blood-vessels may be dissected out with the fingers, or so indurated as to form a shapeless mass of varying magnitude. When abscess forms, it may rupture into the duodenum, or into the cavity of the abdomen, or it may point externally. The paludal poison also often produces structural alteration of the spleen. In these cases that organ has been found sometimes so enlarged as to weigh from ten to thirty pounds (ague cake), greatly exceeding the liver in size, while in other cases it is sometimes even less than natural. In consistency, also, it varies from a state of almost fluidity, a mere bag of blood, to a hardened mass with a distinct indurated edge. It is sometimes the seat of abscess; or its parenchyma is transformed into a soft mass of pigment of an extremely dark colour. The functions of the peritoneum may be alone deranged, so as to produce dropsy; but every form of peritoneal inflammation may precede or accompany the *ascites*,—as the serous or the purulent, with diffuse or partial local adhesions. These are the most usual alterations of function and of structure in the mild paludal fevers of the present day; and in estimating the relative frequency of these secondary affections, *ascites* is the most common, then *jaundice*; while *peritonitis*, *hepatitis*, and *splenitis* are less frequent, and occur, perhaps, in nearly equal proportions. The pathological phenomena which attend severe *intermittent* and *remittent* fever are much more severe, and extend over a greater number of organs. The information afforded us by the dissections of Davis, and the observations of Sir Gilbert Blane, in the cases of the Walcheren remittent; of Jackson in those of the West Indies; of Burnett in the Mediterranean, enable us to understand at least the tendency of the morbid action. Sir Gilbert Blane, in his observations on the Walcheren fever, remarks that the structural derangements were more frequent (especially swelling of the liver and spleen), which then occurred in a very few weeks. Such results seldom occur in England, except under a long continuance of the disease, or after frequent relapses. The morbid changes also extended to the mucous membrane of the stomach, which, in a few instances, became inflamed and ulcerated, and the ulcers had generally a sharp perpendicular edge, as if made with a punch. In cases which died dysenteric, the large intestines, and more particularly the sigmoid flexure and the rectum, were always much contracted, thickened, inflamed, and ulcerated, the ulcers being often so numerous and so confluent that the whole inner surface of the gut appeared in a state of granulation. There is a marked tendency in the phenomena of

these paludal fevers to become inflammatory, the congestion of some organ proceeding at once to exudation from the blood-vessels into its parenchyma, which appears to be the cause of prostration and of fatal results. "The significant term *bilious*, as applied to these fevers of the East, is not an accidental or a misapplied term, as modern statistics fully show. A severe disturbance of the hepatic function is almost universal in the progress of the remittent fevers in the East" (Sir R. MARTIN).

There is another remarkable specific tendency to be noticed in the persistent effects produced by *malaria*—namely, that it impresses a character of intermittence and periodicity to subsequent ailments, especially neuralgic affections; and the disposition to the recurrence of these diseases seems to last for life. Susceptibility to the action of the paludal poison does not diminish, but rather increases by continued residence where it prevails, so that a chill or the opposite, such as exposure to great heat, will alike bring on the specific expression of malarious fever in some of its typical forms in those already poisoned by it. The returns published by the War Office and Army Medical Department show such a result in the West Indies. Thus, while the annual mortality among the troops resident one year in Jamaica was 77 per 1,000, mean strength, in those resident two years it was 87 per 1,000, while of those still longer resident it was no less than 93 per 1,000. "In making calculations of efficient force," writes Sir James Macgrigor, "this description of men could not be relied on for operations long continued in the field" (speaking of men who had suffered from an attack of paludal fever), for "we found that in those who were convalescent or lately recovered from ague, the causes next prone to reproduce the disease were exposure to a shower of rain, or wetting the feet, full exposure to the direct rays of the sun, or to cold, with intemperance, irregularity, or great fatigue." There are many instances also of the same person being repeatedly attacked with the West Indian malarious fever. Sir Ranald Martin writes, with regard to himself, that "after a residence of ten years in Europe, I happened to pass three nights at the best hotel in Strasburg, at a time when ague prevailed in the garrison amongst the French soldiers who had served in Algeria; and two days after quitting the town I was seized with ague at the hour of eleven A.M. (the hour at which ague used to commence with me in India), and I was the only person of the party who was so affected." Although the fever of malaria is characteristically intermittent, it is often irregular in its intermissions. The seizure is not always attended, as in temperate climates, by three stages—the cold, hot, and sweating. At times the cold stage is violent, or it may be altogether wanting. The hot stage is seldom absent, and often constitutes in itself the entire and only access of fever. The sweating stage is often scarcely perceptible, or may be altogether wanting. The type of fever may be also irregular, and it may change rapidly, although periodical intermission constitutes the generic type of the fever. The periods when the attacks come on vary greatly, and are often irregular. The continued or remittent types do not characterise different species of marsh fever; they are only devious or modified types derived from intermittence. The attacks which mark the onset of remittent fever might pass unnoticed by reason of their slight intensity, and the rapidity with which they pass one into the other—sometimes only

one irregular access ushers in remittent fever; occasionally, but rarely, under the influence of quinine, the continued and remittent fevers pass into intermittents. The first attacks of malaria are regular, but soon, after repeated attacks of great violence and long duration, they may lose their regularity, and the short period of intermittence, which passes away, often changes the type into remittent or continued.

All the different types of fever—quotidian, tertian, &c.—including the modified types—remittent and continued—constitute a group. So do also pernicious fevers of the tropics, which are really only intermittent, accompanied by some rapid and formidable change, or by the predominance of some symptom dangerous to life, as algidity, excessive sweats, attacks of dysentery, coma, &c. These pernicious tendencies are always ready to start up, and thus aggravate the malarious condition. Marsh miasm does not affect all persons in a similar manner: some, but very few, resist its influence; others suffer from attacks of intermittent, remittent, and masked fever; while in others the poison remains dormant until some cause from without brings it into play, when intermittent will declare itself. Others, again, fall into a state of cachexia; some there are, who, after having removed to a healthy climate, suffer occasionally from attacks of fever, even of a pernicious character. There are others, again, who, although they may have changed climate, should they by chance receive a wound, or suffer from some other inflammatory affection, will reveal, by the intermittence of the symptoms, the malarious influence which lies latent in the system. This prolonged incubation, this slow evolution, these manifestations far from the scene of the morbid cause, establish the existence of a malarious diathesis, which ends in cachexia. Dr. John Sullivan of Havana especially calls attention to the prevalence of “masked” forms of malarious fever in tropical regions where malaria is endemic (*Med. Times and Gazette*, Feb., 1876).

“They are but too well calculated to deceive the medical attendant, so variable are the symptoms, so changeable, and so capricious; sometimes accompanied with fever, at others fever may be altogether absent. They nevertheless derive their origin from a common malarious cause, which is indicated by three intimately connected phenomena:—First, their intermission; second, the decided influence of quinine in all masked fevers; and third, the frequent transformation of an access of masked fever into one of an active and violent nature. It is in the very ease and rapidity of this change that the great danger from masked fever consists; indeed, this may be followed by attacks of pernicious fever, which, if repeated and not traced to its original source, may lead to death. The marsh poison introduced into the system may lie in a masked form, for a longer or shorter period; and although it may not manifest its presence as a frank intermittent, it will excite, complicate, and aggravate any disease to which the body, from peculiar temperament—whether nervous or sanguineous—may be predisposed. Hence we find in these masked fevers attacks of hysteria and neuralgia in the nervous, of hepatitis and phlegmasia in the sanguineous. In districts where marsh fever is endemic, diseases may declare themselves simple, uncomplicated, and independent of malaria; but when they show a tendency to periodicity, or appear to yield to the administration of quinine, or pass into intermittence, then we can no longer entertain a doubt respecting their malarious origin. These masked

fevers may affect every type—quotidian, tertian, quartan, &c.; and sometimes return periodically, after a lapse of time far greater than that of intermittent fever (after an interval of three weeks, sometimes of a month), and when they do return they are irregular. The attacks may come on in the morning, the evening, or at night, and, whenever masked fever takes on the tertian or quartan type, we must apprehend great danger, and guard against the possibility of an attack of pernicious fever. Sometimes some slight symptom indicative of its malarious origin will develop itself in the course of the fever, as headache, intolerance of light, shivering, a sense of cold and alternating heat and cold, and perspiration; but it happens more frequently that no such indications present themselves. The diagnosis becomes obscure. Now, quinine is the touchstone in this case,—it reveals the true nature of the disease; and in our uncertainty we had better take the safe side, from the fear of pernicious fever—that is, an attack of intermittent, accompanied by some prominent symptom dangerous to life.

"This masked fever, however varicoloured may be its form, manifests itself usually by some functional derangement—by a lesion in some organ or another, some hæmorrhage, or inflammation in some part. It will sometimes take the form of neuralgia, at others of a neurosis: a supra-orbital or infra-orbital neuralgia, a dental neuralgia, are some of the many forms in which masked fever is often disguised. Should these pains occur in the night-time, they must not be confounded with pains of a syphilitic origin. In children you will sometimes find the voice become hoarse, or nearly lost, with a cough which comes on periodically. These attacks may often simulate and be mistaken for hooping-cough. Hiccough, coming on at certain hours, I have often noticed as a manifestation of masked fever. This masked fever, in infants, often assumes a convulsive form. In tropical climates convulsions in children are very frequent. They may be owing to worms—so common in the Antilles—to teething, or to some malarious influence. Here we must be careful in making a proper diagnosis, as a mistake might be fatal to the life of the child,—by administering quinine improperly, or by not giving it when required. In women it may exist under the mask of hysteria. Blood in the urine (so frequent in the Brazils) will often betray a masked malarious fever. If we find that its return is periodical, we must employ the febrifuges; or, in plain language, if we find that a tendency to decomposition of the blood is manifested by hæmaturia, we must make use of an antiseptic capable of counteracting that tendency. Although masked fever is described as varied and capricious in its manifestations, we must bear in mind that the nature and essential attributes of malaria never vary. The poison, when introduced into the blood, powerfully depresses the nervous system, and leads to congestion of internal organs. The most complete expression of its effects is to be seen in the cold stage of intermittent, and, when still more intensified, in the algidity of pernicious fever. When we say that the symptoms of masked fever are varied and capricious, we mean that it is as varied in its symptoms as are the subjects to its influence varied in constitution and susceptibility. Latent malaria always detects the weak spot in her victims, and develops the irritability of that spot, but with a masked periodicity and intermittence. This masked fever requires a larger dose of quinine than does simple intermittent,—a dose such as may be administered in pernicious fevers. In hot climates, where malaria is endemic, we may take it for granted that any symptom, with or without fever, characterised by intermittence, ought

always to excite the careful suspicion of the medical attendant, who must always remember that slight febrile attacks are often followed by attacks of a pernicious nature, and that pernicious fever may rapidly, and when least expected, usurp the place of masked fever."

As regards *Pernicious* fever the same authority writes:—

"The poison of malaria, in proportion to the degree of its intensity or destructive power to the susceptibility of the bodily condition of the recipient of such poison, may generate a fever, which, from its tendency to paralyse all nerve-power and to promote a dissolution of the elements of the blood, may justly be denominated 'pernicious fever.' No poison has ever introduced itself into the human system so capable of producing effects so varied, of assuming such questionable shapes, of wearing so many masks and disguises, as the poison of marsh fever,—effects which vary in degree from a slight depression of nerve-power to a partial extinction of sense, and even to the complete destruction of life. But the poison of malaria will often lie concealed in the system ready at any moment to complicate and exaggerate any disease to which the body may be subject, if it do not completely control and subject such body to its undivided influence; or it will take upon it so many Protean forms that its victim will be thrown off his guard, and probably die from its masked effects. But under no form does malaria show itself with so great intensity as under the form of pernicious fever. This fever may appear under the form of algide or of comatose fever, with all their varied complications—*febris algida* being the most complete expression of the true nature of the poison, paralysing all nerve-power, determining the blood to internal organs, and thus producing that intense algidity as of death; while *febris comatosa* is marked by an especial malarious determination to the brain, producing an anæmic contraction of arteries, as in a fit of epilepsy. It sometimes happens, while attending upon a case of intermittent fever, when we imagine that the case is progressing satisfactorily and with all due regularity, on a sudden the symptoms alter greatly for the worse; one or more of its stages may become present in great intensity, complicating or placing in jeopardy one or more of the organs essential to life. In the tropics, pernicious fever may be seen complicating the various types of intermittent, and tending to aggravate a variety of morbid states which have no necessary connection with the poison of malaria. The prognosis must depend upon several conditions. Suppression of urine is an alarming symptom, but should the secretion be restored, an improvement is sure to follow. Convulsions, frequent in children, are perhaps, next to black vomit, the most dangerous symptom; when they make their appearance at the close of an attack of fever, there is always danger to life. Vomiting of black matter is fatal to adults; I have seen children occasionally recover. There are two symptoms which, when they do occur in the course of an intermittent, always occasion me great alarm and anxiety. The one is an acute pain, which fixes itself in some part of the body—sometimes in the side, like an attack of pleurisy, at other it strikes upon the hip-joint. Now, from long experience I have always found the accessions of these pains to be a very ominous symptom. The second symptom, of still rarer occurrence, is the vomiting of blue (not black) matter; it may occur in the adult who has suffered from marsh cachexia, or from chronic diarrhoea. Pernicious fever, the reverse of yellow fever, may repeat itself. I have known persons whose life was always a burden

to them from the fear of a fresh attack. All races suffer from it. Old people often die from it. I have seen children attacked by it two or three times almost imperceptibly, then begin to vomit up a blackish fluid, followed by convulsions and death. In them when fever is ushered in by general or partial convulsions, the symptom is generally fatal, as indicating a tendency to the same manifestation in every succeeding attack.

"All chronic diseases in tropical climates, all deep-seated causes of debility, privation, and suffering, the emotions from grief or anxiety, and alcoholism, predispose to attacks of pernicious fever, since malaria directly attacks nerve-power; and when this has been shattered from the above causes, it becomes more sensitive to its influence. The varieties of pernicious fever characterised by some violent predominant symptom may be classed into two principal groups—*febris algida*, comprising the fainting, dysenteric, and choleraic varieties; *febris comatosa*, the tetanic, lethargic, and convulsive.

"(1.) *Algide Pernicious Fever.*

"*Febris algida* would appear to be the most faithful manifestation of the *modus operandi* of the poison of malaria on the living organism—perhaps the only pathological base upon which all other pernicious fevers rest, so that it will sometimes appear in its naked uncomplicated form as a termination of pernicious fever. The progress of algidity is usually insidious; it often shows itself in the middle of an apparently simple attack of intermittent. The patient appears in his usual health; presently the face turns pale, the features become sharpened and drawn in, the lips turn blue, the eyes hollow, the pupils become dilated, the pallor and coldness gradually go on increasing, and cold, clammy sweats pervade the entire body; you might fancy you were gazing on a corpse. The hands and feet first turn cold, next the extremities; the abdomen alone preserves some heat. If you touch the skin it feels like marble, and yet the patient complains of a sensation of heat internally. The tongue is the last to become cold, and when the air expired is colder than that inspired, the danger becomes imminent. The urine is often suppressed as the algidity increases; the skin becomes more and more bathed in a cold perspiration. The heart beats slowly, and is scarcely audible; the pulse thready, not to be felt at the wrist. The intellect is unimpaired. The patient does not complain of any particular pain. Respiration, from being calm and slow, becomes anxious and rapid. To a calm succeeds a vague sense of uneasiness, which is soon replaced by a feeling of approaching death. In the present year (April, 1875) I was called to visit the wife of D. B. M., a well-known sugar planter in Cuba. She had been delivered of a child about two months before, at which time she complained of slight chills and fever; since then severe intermittent had set in. I was greatly surprised, on my arrival, at the consternation and dismay depicted on the countenances of the patient's friends and attendants. I decided upon taking a view of the patient, unseen by her. She was seated in an arm-chair, the arms of which she firmly grasped with both hands; the body slightly inclined forwards, rigid, erect; the face was pale and cadaverous, the countenance expressive of anxiety and terror, mixed with a certain degree of sadness. She looked as it were on vacancy, and took no notice of those who surrounded her. On my entering the

patient's room, she directed an anxious and inquiring look towards me, as if conscious who I was, and desirous to be relieved. I took her hand, which, as well as the entire body, was bathed in a cold, clammy, icy sweat; no pulse at wrist. She could only reply to my questions in a whisper, but a whisper deep, cold, and unearthly. The heart's beat was strong and even tumultuous, as though labouring hard to upheave and dislodge the mass of blood by which it was oppressed. The right heart communicated its impulse to the jugular veins,—hence strong venous pulsation. I found, on examination, considerable congestion and enlargement of the spleen, and induration of an atrophied liver. I ordered a hot mustard foot-bath, ten grains of calomel, with fifteen grains of quinine, to be taken immediately,—the dose to be repeated in two hours' time; frictions of quinine in brandy (two to six drachms) to be rubbed over the extremities and down the spine every half-hour. About four hours after my first visit the heat of the body returned, and the patient was enabled to lie down in her bed, which she had not been able to do during the last three weeks. After the second dose, I followed up the calomel and quinine in smaller proportions—two grains of calomel to four of quinine every four hours. I ordered besides, in order to promote the partially suspended secretion of urine, two tablespoonfuls of a strong diuretic mixture every two hours, and placed over the loins (a custom peculiar to the country people) some digitalis leaves wetted with vinegar. After eight hours had elapsed, considerable relief was obtained and all the symptoms improved: the secretion of urine was restored, circulation became less impeded, and some sleep was procured. The algidity returned daily about 3 p.m. for three successive days, but in a gradually milder form, of less duration; and the patient finally completely recovered. This case I regard as very instructive and interesting, as it is a complete manifestation of the effect of malaria upon the human body. It would seem that the miasmatic poison availed itself of the new inlet into the system by the medium of the raw and dilated surface of the vagina and uterus after delivery—a medium through which, under similar circumstances, other septic poisons have been introduced, and with fatal effect, into the system. This intensified dose of malaria would appear to have generated algidic pernicious fever. Large doses of quinine in this case proved most triumphantly its value and its nature as an antiseptic.

“The diagnosis is easy when the pernicious fever is simple, but when complicated with dysentery or cholera it is rather obscure. Between pernicious choleraic fever and Asiatic cholera it is almost impossible to diagnose, especially when the two endemics exist at one and the same time. But between simple dysentery and pernicious dysenteric fever the diagnosis is more easy. The stools are not so bloody; they are more abundant and liquid, and provoke less tenesmus. The antecedents—attacks of fever, algidity, cold sweats, &c.—make the diagnosis more easy. We must not confound the fever of pernicious dysenteric fever with that which accompanies simple or true dysentery, which is symptomatic; while the fever of pernicious dysenteric fever, being of malarious origin, is independent—a foreign element superadded to the principal malady, which it aggravates and often renders mortal. This is an important fact worthy to be remembered in our practice in tropical climates. Hence, in the complicated dysentery, all remedies directed to the cure of dysentery will prove useless, so long as we do not neutralise the foreign morbid element by large doses of an antiseptic.

“The attack of *febris algida* consists of one long stage. The cold increases

gradually, with cold and clammy sweats; the voice falls to a whisper; the pulse falters, finally ceases; and the patient dies in a swoon. Should reaction take place, the pulse rallies; the heat returns gradually to the surface from the abdomen, in which it would appear to have concentrated itself, radiates to the chest, and thence to the whole body. The cold stage, or state of algidity, may last forty-eight hours or longer. Recovery is often insidious, and may be complicated with symptoms of an ataxic or typhoid nature. The mortality from *febris algida* is not so great as that from *febris comatosa*. In the algide state the powers of absorption do not appear to be annihilated, as in the cold access of Asiatic cholera.

"(2.) *Comatose Pernicious Fever.*

"Insolation is occasionally a most powerful cause. A man who leaves a marshy district, and travels a long way exposed to a burning sun, is very subject to an attack of malarious apoplexy. The fit may seize him in the evening when the sun has set, or even at night-time when in bed. Like other pernicious fevers, the *comatose form* especially attacks those who, after a long residence in a marshy district, remove into another free from malaria. It is usually preceded by some derangement of the health—by two or three irregular attacks of fever, which may be trivial or scarcely perceptible. The invasion of coma is violent, its progress rapid, its stages of intense severity; if the patient be seized when standing, he falls down as if struck by lightning; if when lying down, it comes on unperceived, and only to be revealed by his stertorous breathing. I have never known delirium or convulsions precede an attack of pernicious malarious coma. I remember one patient, who had recovered, told me that he had experienced during two or three days before the attack a creeping sensation up the back, with a sense of suffocation and constriction about the chest. The cold feeling is absent, but the hot stage is soon developed; and, unlike the skin in *febris algida*, it is covered with a hot perspiration—a frank intermittent is seldom observed. The fit may last from four to five hours, or even to as many days. The characteristic symptoms are,—loss of motion, sensation, and intelligence, with impaired function of respiration and circulation; the patient answers pettishly and in monosyllables; the pupils are insensible to light; some low murmurs of complaint may be often heard, indicating severe headache. The frequent return of motion proves the intermittent character, when the symptoms generally improve. But in cases which threaten to end fatally, we often observe muscular contractions, as trismus strabismus (complete abolition of sense and motion). There may be violent tonic and clonic contractions; and death often closes the scene, after one or two attacks of black vomit.

"The diagnosis is not difficult. The antecedents—previous attacks of intermittent, the paludinous diathesis,—the absence of all plethoric symptoms, will clear the way for a correct diagnosis. The only difficulty is to diagnose between the apoplectic form of pernicious fever and true apoplexy. This occurs but very rarely in the tropics, but may occur to strangers who have lately arrived. We must be careful not to bleed in pernicious apoplexy, as distinct from true apoplexy. Bleeding would inevitably hasten the fatal termination. By not interfering too hastily, by waiting a little, by adopting the maxim *festina lente*, so as to allow the disease to clear up, we may be enabled to form a just diagnosis. The

heat and moisture of the skin, the hurried pulse, indicate that the apoplectic fit derives its origin from marsh poison. *Post-mortem* examination reveals no inflammation of the brain or of its membranes, nor any lesion to which might be traced the severity of the symptoms. We may find, as in all fevers of a certain duration, the membranes, especially the pia mater, somewhat injected; the cerebral substance redder than usual; some serum infused into the ventricles; and when black vomit has occurred, we shall find the mucous membrane of the stomach of a yellowish tint, streaked with violet-coloured stains.

"The almost impossibility of administering proper medicines adds to the danger of this form of pernicious fever. Quinine can be introduced into the stomach with difficulty, by reason of the spasm of the pharynx or œsophagus. We must, therefore, have recourse to injections of the drug per rectum, to frictions of quinine in alcoholic solution, so as to produce absorption. In these cases the subcutaneous injection of quinine is a valuable method of its introduction into the system. It possesses the advantage of economy, is easily applied, however severe may be the vomiting, difficulty of swallowing, or coma. Mr. W. Moore, of Bombay, has tried the hypodermic method on a large scale. He assures us that he has injected quinine successfully in 100 or more cases of intermittent and remittent fevers. The quinine must be used in solution, not in suspension. The best time to inject is before the cold stage.

"Several French, German, and American physicians have advocated the subcutaneous method in all cases of intermittent, especially where quinine cannot be administered by mouth. I cannot, from my own experience, bear witness to the great advantages to be derived from the subcutaneous method. I have tried the plan eleven times, three times with a certain beneficial result; the effect was always a shorter duration than when quinine was introduced by any other method, and I had to renew the injection frequently. I would have injected more frequently had I not been deterred in my practice by the inconvenience from little accidents arising from the puncture made by the injecting-syringe, some swellings, and often superficial abscesses, which were looked upon by my Spanish patients in Cuba with a most ridiculous degree of anxiety. For my part, I have trusted more, in the treatment of *febris comatosa*, to revulsives—hot mustard foot-bath, blisters to the calves of the legs, strong injections of quinine per rectum, having previously administered a purgative enema, strong frictions of quinine, &c. It is seldom that this plant fails to develop the malarious origin of this comatose form of pernicious fever; but in no case, however strong the indication may appear, however intense the symptoms may be, must we bleed. When the tendency towards convalescence becomes established, should speech appear to be somewhat embarrassed, should we observe dulness and uncertainty in the gait, in such cases I have always derived advantage from the combination of calomel and quinine."

CHAPTER VII.

DETAILED DESCRIPTION OF THE GENERAL DISEASES.

SECTION A.

SMALL-POX.

LATIN EQ., *Variola*; FRENCH EQ., *Variole*; GERMAN EQ., *Blattern*—Syn., *Menschenpocken*; ITALIAN EQ., *Vajuolo*.

Definition.—*Small-pox in man is the product of a specific and palpable morbid poison, which is reproduced and multiplied during the course of the malady. It is contained in the contents of the pustules, and in the cutaneous and pulmonary excreta of small-pox patients. After a definite period of incubation (of about seven to nine days, in cases of inoculated, and from ten to thirteen days by ordinary infection) a remittent fever is established, and followed by an eruption on the skin on the third day of the fever after forty-eight hours' illness, and sometimes on the mucous surfaces. The eruption on the skin passes through the stages of papule, vesicle, pustule, scab, and leaves marks or cicatrices on its site. The disease runs a definite course, and, as a rule, exhausts the susceptibility of the constitution to another attack.*

Pathology.—The theory regarding the origin and development of small-pox is, that a specific poison of a material nature is absorbed and infects the blood, and after a given period of latency gives rise to “primary fever,” which lasts from two to four days, till the eruption appears, when the fever for the most part *remits*. (See Chap. I., p. 356, *ante*.)

Morbid Anatomy.—As the eruption, or formation of the small-pox pustule, is undoubtedly a marked characteristic of the disease, it requires particular description first. It has certain definite stages in its development. It runs a given course of about eleven days, and in its progress undergoes many mutations. It is at first a papule, then a vesicle, then a pustule, and lastly it forms the scab or crust. These various changes form so many *stadia* of unequal duration. The first, or stage of papule, lasts from twenty-four to forty-eight hours; the second, or vesicular stage, four days; the pustular stage, three days; while the last stage, or that of scabbing, lasts three days more, making the whole duration of the *normal* pustule ten or eleven days. There are varieties, however, of this disease, in which the formation of the pustule is irregular, as in the *confluent* and *horn* small-pox. In the latter the two last stages are singularly shortened, or absent altogether. When the eruption in small-pox is of the “*distinct variety*” (Syn., “discrete”), its first appearance consists of a number of small red papules, about the size of a pin's head, more or less numerous, but separate and distinct from one another, and scarcely salient. They commence with a circumscribed hyperæmia of

the true skin (*variolous dermatitis*), extending, more or less deeply, into the subcutaneous tissue. The cells of the *rete* Malpighii swell up, the papillæ elongate, and the red spot of skin becomes a sharply-defined nodule, perfectly solid, and having a flattened top (NIEMEYER). On the second or third day of the eruption the second stage towards the development of pustules commences. A small vesicle, which gradually enlarges, bound down and depressed in the centre, or *umbilicated*, forms on the apex of each pimple, by elevation of the outer layer of the epidermis, and contains a clear whey-coloured fluid. This vesicular stage lasts about four days, when the vesicle matures or "ripens" into a pustule, the characteristic "*pock*." This process is so gradual, that, if you examine the pock closely about the fifth or sixth day, you may see, at least in many, two colours—viz., a central whitish disc of lymph set in, or surrounded by, a circle of yellowish puriform matter. "In truth, there is in the centre a *vesicle*, which is distinct from the pus, so that you may puncture the vesicular portion, and empty its contents, without letting out any of the pus; or you may puncture the part containing the pus, and let *that* out without evacuating the contents of the vesicle. The vesicles have even, by careful dissection, been taken out entire" (WATSON). The adherence of the altered cuticle to the cutis at some points, and its separation at others, produces the little compartments or dissepiments spoken of by some writers. These cavities are usually irregular in shape; and all who have examined these multi-locular cavities agree in describing the existence of a white substance in them, of the consistence of pulp or thick mucus, which at first was supposed to be the specific exudation of small-pox. It is now ascertained that it is no pseudo-membrane, but is composed of the deeper and softened layers of the epidermis. This "disc" of softened epidermis covers the interior of the pustule, and extends from the centre to the raised circumference of the pustule in diverging rays, forming part of six or eight fan-like chambers of nearly equal size. In the structure of this disc the following elements are distinguishable from without inwards,—(1.) Large flat cells; (2.) Large cells not so flat, but more globular, with nuclei; (3.) Nearest the cutis are the cells and tissue of the *rete mucosum* (GRUBY, GLUGE, RAYER, GUSTAV SIMON, besides other observers of more early date). The poison is believed to be most active just at the period when the clear contents of the vesicles begin to turn cloudy. While the maturation of the vesicle into a pustule is going on, a damask red areola forms around each pustule; and as each vesicle fills, the whole face swells, often to so great a degree that the eyelids are closed. While the maturation is complete, the "*bride*"—the name given to the part which binds down the centre of the vesicle—ruptures, and the pustule now becomes *spheroidal* or *acuminated*. About the eighth day of the eruption a dark spot is seen on the top of each pustule. At that spot the cuticle ruptures, allowing matter to exude, which concretes into a scab or crust; and during this process the pustule finally shrivels and dries up. The crust is detached between the eleventh and fourteenth days, leaving the cutis beneath of a dark reddish-brown hue—a discolouration which lasts many days, or even weeks. On the face, however, the pustule often burrows, so as to cause ulceration of the *rete mucosum*,

leaving a permanent cicatrix in the form of a depression or "pit." The cicatrix thus formed, though at first of a reddish-brown colour, ultimately becomes dead white.

The eruption does not appear over the whole body at once, but in three successive crops. It almost invariably appears first on the face, generally the forehead, and the first crop covers the face, neck, and upper extremities, the second the trunk, while the third appears on the lower extremities. In rare instances it commences about the wrists. There is usually an interval of several hours between each crop; and the later the papules are in appearing on the trunk and lower extremities than on the face and neck, by so much the later they are in maturing and in disappearing from those parts. When the eruption on the face is declining, that upon the extremities has scarcely yet arrived at its height, so that the hands and feet are then considerably swollen. This is to be regarded as a favourable sign, in so far as it indicates a certain vigour of constitution. The number of pustules may not exceed five or six over the whole body; more commonly they number from one to three hundred, and occasionally amount to several thousands. It has been calculated that if ten thousand pustules be counted on the body, two thousand at least will be found on the face; and accordingly, the number of pustules on the face being in proportion, those on the other parts of the body furnish a fair estimate of the severity of the disease, and of the danger of the patient.

The pustule is subject to many irregularities, both as to its form and course; which give rise to two marked varieties of the disease—namely, the *confluent* and the *horn* small-pox. The *confluent small-pox* differs from the *distinct small-pox* in the papules being small, less prominent, and so numerous that even on the first appearance of the eruption there is hardly any distinct separation between them. The vesicles which form on their apices appear earlier, and their diameters increase more irregularly than in the distinct forms, and often they run one into the other. The eruptive stage is usually shorter, and spreads over the body more rapidly, the contents of the vesicles become purulent sooner, and their confluence on the face makes it look "as if it were covered with one large bladder of matter." The pustules, likewise, which are confluent, either remain flat, and do not rise, or, the areolar tissue rupturing, they form large bullæ or bladders in clusters like a bunch of grapes—a rare variety of the disease (*variola corymbosa*)—and are not encircled with the usual red areola round their base; neither do their fluid contents always acquire the yellow colour and thick purulent consistency of the milder disease. Their crusts, moreover, are soft, and do not fall off till many days after the usual period—not till the eighteenth or twentieth day, or even later. When the desiccation is completed and the crust detached, a deep scar or pit, sometimes an extensive seam, remains, and shows the loss of substance that has taken place, and how destructive to the tissue of the true skin has been the process beneath these crusts. When the pustules remain separated by intervals of healthy skin, or just touch each other, the varieties are known respectively as *distinct* or *discrete* small-pox (*variola discreta*) and *coherent* small-pox (*variola coherentes*). The *horn small-pox* is a variety of the pustule, and is by much the mildest form of the disease. The pock in

this variety passes through the stages of papule and of vesicle; but on the fifth or sixth day of the eruption, instead of maturing, the pock shrivels, desiccates, and crusts, and the disease terminates three or more days earlier than in the usual course, and without the occurrence of any secondary fever. This is the very mild and modified form of the disease which so usually follows after vaccination. Where the pocks do not pass beyond the first stage of papule, so that no vesicles form, the variety is known as *variola verrucosa*. Many other varieties have been described by the older authors, which are seldom if ever now seen—for instance, *black small-pox* (SYDENHAM); a *blood small-pox*, *variola cruenta* (MEAD), or *hæmorrhagic*, in which “blood is effused into the vesicles or pustules,” the period of incubation of this variety being from four to eight days (ZUELZER, quoted by Dr. MURCHISON); a gangrenous small-pox (*variola gangrenosa*), when the vesicles fill with an ichorous matter, accompanied with gangrene of the skin; a *siliqueous small-pox* (FRIEND), in which the pustule resembles a small hollow bladder, but contains no fluid (*variola siliquosa seu emphysematica*). There is one variety, however, which is not uncommon, called the *crystalline* or *pearl pock* (*variola crystallinæ*), in which the vesicle continues transparent, seldom matures, and has a tendency to become confluent (*variola lymphatica seu serosa*). Every variety of the eruption, when the disease is severe, may be intermixed with petechiæ. Such are the chief features of the disease, so far as the development of the eruption is concerned.

Varieties and Symptoms of Small-Pox.—The species of small-pox recognised in the Nomenclature of the College are:—

Group A, Unmodified; *Group B*, Modified: Definition,—*Pustules cut short in their development, by vaccination or previous attacks of small-pox.*

The varieties applicable to both groups are,—

- (a.) *Distinct*; Synonym, *Discrete*. Definition: *All the pustules separate.*
- (b.) *Confluent*. Definition: *Pustules running together over the greater part of the body.*
- (c.) *Semi-confluent*. (d.) *Abortive*; Synonym, *Varicelloid*. Definition: *Comparatively few pustules, the general eruption scarcely passing beyond the stage of vesicle.*

The subordinate varieties are,—

- (e.) *Petechial*. (f.) *Hæmorrhagic*. Definition: *Blood effused into the vesicles or pustules, with a tendency to hæmorrhage from the mucous surfaces.*
- (g.) *Corymbose*. Definition: *Some of the pustules assume the form of clusters, like a bunch of grapes (corymbus).* This is a rare variety of the disease.

The varieties of small-pox are here much more elaborated than those of other similar diseases in the Nomenclature of the College, chiefly to meet the wants of public institutions for the treatment of small-pox.

Group A.—Unmodified or Natural Small-Pox.

Distinct Small-Pox.—In this form of small-pox all the pustules are separate, but sometimes may partly touch (*coherent—variola coherentes*). The symptoms and development of *variola discreta*, or of distinct small-pox, are traceable through four stages. The first comprises the period of incubation or of latency—a period of time which varies according as the poison

has been introduced by the mucous or cutaneous tissues. In the former case the more usual time of latency is from ten to thirteen days; while in the inoculated small-pox the period of latency is from seven to nine days, the extremes, taking both forms of the disease, being from five to fourteen days. Bärensprung, of Berlin, has lately recorded a most interesting fact, which demonstrates, in a more striking and definite manner, the period of latency. He observed seven cases of small-pox, *all* of which were infected from the same source on the same day. In *all* of them the outbreak occurred between the thirteenth and the fourteenth day. Some of them had been vaccinated and others had not (*Annalen des Charite Kranken*, Vol. XIX., p. 103). But Dr. Murchison has recently shown (p. 385, *ante*), that the incubation period of small-pox is far from being fixed. The *second stage* comprises the primary fever, the earliest expression of constitutional disturbance, which generally terminates with the appearance of the eruption (*stadium prodromorum*). The *third stage* commences with the eruption, and terminates with the appearance of the secondary fever (*stadium eruptionis*). The *fourth stage* commences with the secondary fever, and includes the suppuration and maturation of the pock, with all the subsequent phenomena of desiccation, scabbing, and convalescence (*stadium suppurationis seu maturationis et exsiccationis*).

In the adult the symptoms of the second stage are to be distinguished from those of the first stage of typhus, or other febrile affections, by the characteristic ranges of temperature. There is, however, nausea. The brain, too, is oppressed, as indicated by drowsiness, stupor, or coma, followed occasionally by convulsions, especially in children. The ordinary duration of this fever is four days; and it may be sudden in its attack, or preceded by some days' illness, in which case the most prominent and characteristic symptoms in the adult are severe muscular pains simulating rheumatism, especially in the small of the back, the *sacral* and *lumbar* regions, and the frequent occurrence of obstinate vomiting, foreboding a severe form of the disease. The primary fever commences with a chill or repeated well-marked rigors, followed by the sensation of great heat; and the thermometer rising rapidly, may indicate a temperature of 104° to 106° Fahr., attaining its maximum when the eruption can be felt. The face is suffused with redness, and the carotids pulsate strongly. There is usually much thirst, loss of appetite, and pains in the limbs. The tongue is coated, and the secretions of the mouth are slimy. Sometimes there is epistaxis. During the fever, and prior to the eruption, there is one symptom of great importance in small-pox, as especially pointing to what is about to happen,—that is, the pain in the back. It is a peculiar and striking symptom, and more intense than in any other form of fever. It is distinguished from lumbago by its position. In lumbago the muscles on each side of the spine are affected, it is greatly aggravated by movement; while the pain of the back in small-pox is in the central part of the *sacrum* and the lumbar region, and is not influenced by movement. There are also other characteristic symptoms, namely,—pains and fulness over the epigastrium, with nausea and sometimes obstinate vomiting; pains general over the body, and tremulousness of the muscles.

On the evening of the third or morning of the fourth day, after the commencing chill, the fever is usually at its height; and on the fourth day

(sometimes sooner, but seldom later) the eruption appears, and the third stage commences. The phenomena of the third stage are as a calm succeeding a storm; for, on the appearance of the eruption, the fever remits, the heat abates, the affection of the head subsides, the vomiting ceases, and the pulse returns to its natural standard. The febrile phenomena seem to have altogether disappeared for the time, and the patient may think himself well. A temporary defervescence is thus well marked, the temperature falling, from perhaps 106° Fahr., progressively downwards to 100° Fahr.

The eruption appears first in minute bright red specks on the face,

TYPICAL RANGE OF TEMPERATURE IN A CASE OF NATURAL SMALL-POX, COMMENCING WITH THE THIRD STAGE; THAT IS, FROM THE PERIOD OF THE ERUPTION, ON THE EVENING OF THE FOURTH DAY FROM THE BEGINNING OF THE SICKNESS. THE RECORDS INDICATE MORNING (A.M.) AND EVENING (P.M.) OBSERVATIONS (Wunderlich).

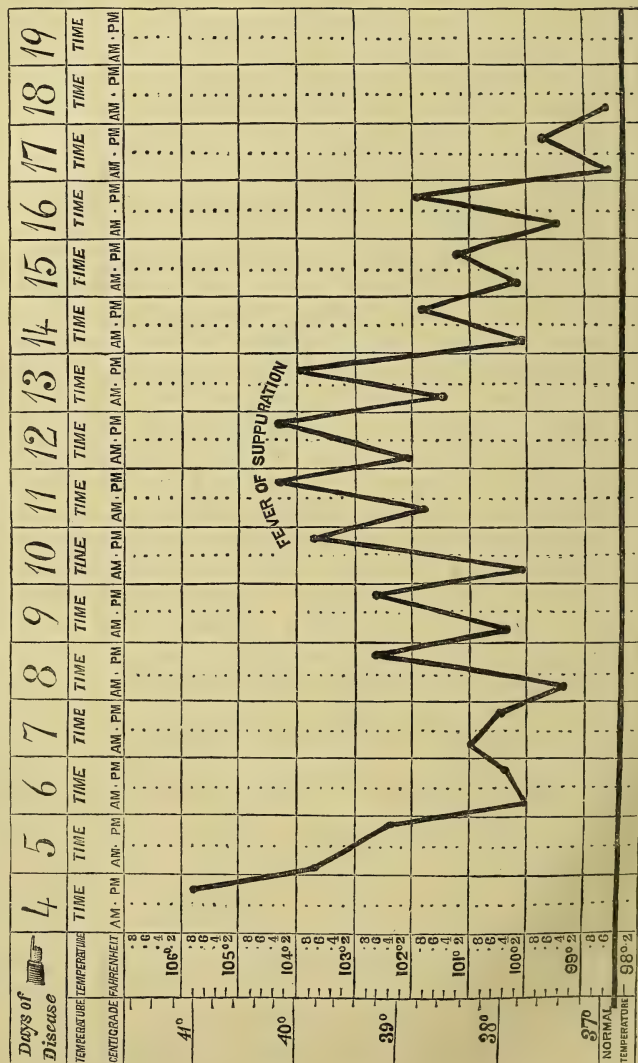


Fig. 80.

neck, and upper extremities, then on the trunk, and lastly on the lower extremities. It runs its course in a succession of crops, developing late as they appear late; which undergo the various mutations of papule, vesicle, and of pustule already described. The eruption on the mucous membrane commences at this time also, but it is not generally recognised till its occurrence in the mouth increases the flow of saliva; or in the pharynx, causing a difficulty in swallowing; or in the air passages, causing cough, hoarseness, or altered *timbré* of the voice; or the conjunctiva, causing the tears to flow, and light to be avoided. There may be also difficulty of making water, and severe pains in the external genitals, symptoms which come on later after the others may have subsided. About the eighth day of the disease, however, or when the eruption is fully out over the whole body, and the pustules on the face begin to mature, the whole face, head, and neck swell, particularly the eyelids, which often close and blind the patient. The swollen parts throb, and are painful when touched. The intumescence of these parts lasts three days, during which the spaces between the pustules inflame, and are of a deep red or damask-rose colour. It is during this period of intumescence, simultaneously with the renewed hyperæmia of the skin, and introductory to the change taking place in the contents of the pustule, that the fever, which had remitted, returns, and the *fourth stage*, or that of secondary fever, commences, sometimes called the—

Fever of Suppuration.—This stage, in cases of ordinary intensity, is marked by a rise of temperature to a considerable height, by a frequent pulse, sometimes by a rigor, or repeated chills, and by slight delirium, from which the patient is easily aroused. If, however, the disease be of greater intensity, hæmaturia, hæmoptysis, or a hard dry cough, are added in proportion to the implication of the mucous membrane. In favourable cases the swelling of the face, the redness of the intervening spaces, and also this secondary fever, having lasted from the eighth to the eleventh or twelfth day, subside, and the pustule, now fully ripe, bursts and discharges a thin yellow matter, which, concreting into a crust, falls off on the fourteenth or fifteenth day, and the disease terminates. During this somewhat protracted defervescence, the temperature sinks gradually, to rise, perhaps, for the third time, when the final desiccation takes place.

In the very mild variety of distinct small-pox which was wont to be named the "*horn-pox*," the primary fever is little more than a febricula; the pustules may not exceed half a dozen to two or three hundred; and, having passed through the stages of papule and of vesicle, they will on the eighth day—*i.e.*, about the usual time of maturation—shriveled, desiccate, and crust. The secondary fever, often so fatal in unmodified small-pox, when vaccination has not been done, does not recur; so that the convalescence usually commences on the eighth day, and the disease is terminated on the eleventh. It was once supposed that in such cases the pus of the eruption was absorbed; but it appears that pus does not form, the fluid always remaining serous. Sydenham and Frank observed in every variolous epidemic, that some few persons who have not previously had the small-pox, or, according to Frank, have neither had the small-pox nor been vaccinated, are seized during the time the small-pox is raging with all the symptoms of primary variolous fever, which, having subsided, they

have afterwards been found insusceptible of the disease. Sydenham records fatal cases of this kind attended with purple spots and bloody urine,—and hence the “*variola sine eruptione*” of authors,—which, when it occurs in the present day, is more usually regarded as a modification of small-pox, probably depending on the influence of vaccination, the existence of which may have been overlooked; but when occurring in the unvaccinated, is generally suggestive of a fatal issue.

In cases of any degree of severity, even in the *distinct small-pox*, the poison acts on the mucous membrane as well as on the skin as already indicated, and produces an exudation in several parts of that surface. This additional lesion, however, does not appear to aggravate the fever, or to occasion other inconvenience than what arises from the local mischief. The buccal eruption is usually preceded and accompanied by soreness of the throat and difficulty of swallowing, and sometimes salivation; but these symptoms do not usually exceed those of a common sore throat. The exudation upon the mucous membrane is generally resolved without the formation of ulcers, or anything that can be considered a scab or cicatrix. The exudations which form within the eyelids are not attended with much pain; and it is only when the swelling has subsided that the mischief which sometimes takes place is discovered.

A peculiar faint and sickly odour, of a “greasy, disagreeable” kind, and quite *sui generis*, emanates from the small-pox patient during the period of maturation of the pustules. So much is this the case, that Dr. Watson says, “one might name the disease at once by the smell.” When, however, the case assumes an unfavourable character, and threatens a fatal termination, the face, which ought to have been intumescent on the eighth day, remains without increase of size, and the spaces which ought to have inflamed are pale and white. The pustules also, says Sydenham, look red, and continue elevated (even after death). The saliva, which flowed freely up to this day, suddenly ceases; and at this critical period the secondary fever, instead of its usual sthenic character, may assume one of two forms,—namely, either a form like the second stage of typhus, with brown tongue, frequent pulse, and delirium; or the patient may be overwhelmed with the depressing influence of the poison, and sink almost without experiencing a reaction, the pulse being hardly increased in frequency, the heat of the body natural, and the intellect unimpaired. But the patient suffers from an indescribable restlessness, an inexplicable anxiety, some cough, with sickness, a frequent desire to pass urine; and with such symptoms as these he dies. In the regular typical course of small-pox the vesicles become larger at the period of maturation, and the skin around them swollen, and of a bright red colour. With diffuse redness and swelling, the face becomes greatly disfigured, and the pulsating pain in the skin becomes severe, and the tension distressing. It is during the suppurative stage that the lesion of the mucous membrane causes the greatest distress, swallowing may become almost impossible, the saliva flowing from the mouth, and the nose being stopped, the voice inaudible, and the cough distressing. The eyes burn, and are extremely sensitive to light, being of a dark red, and full of muco-purulent secretion.

The intensity of the fever of maturation seems to be in proportion to the intensity of the dermatitis, on which it seems to be secondary, and

not directly due to the variolous poison, although, doubtless, the influence of the poison on the blood must tend to aggravate the fever.

Confluent Small-Pox is that form in which the eruption is abundant and the pustules run together over the greater part of the body. It is described by Sydenham as beginning with symptoms similar to those of the distinct small-pox, but more violent; the second stage, or primary fever, being attended with more sickness and vomiting, with a higher temperature, with rigors, with more severe muscular pain, with more considerable delirium, and in children often, on the evening before the eruption, by convulsions. The fever is not only more intense than in the distinct form of small-pox, but is of shorter duration, and more tumultuous—the eruption appearing more generally on the third day, or even earlier; and the sooner the pustules appear, so much the more confluent is the eruption likely to be. It is often preceded by an extensive erythematous or erysipelatous inflammation, and the pimples come out irregularly, or in small clusters, like measles, and are less eminent than in distinct small-pox.

When the third or eruptive stage is formed, the primary fever remits, but not so completely as in the distinct kind. The pulse often continues frequent (110 to 120 in a minute); the temperature does not fall so distinctly; the tongue continues white; and even the delirium may recur in the evening. The eruption also has some remarkable characters; for the pustules, especially those of the face, do not rise; they are more irregular and flatter in form; and, from their greater number and contiguity, run into each other, sometimes forming bullæ as large as a hen's egg, and sometimes scarcely a portion of healthy skin is visible. Other symptoms, sometimes seen in distinct small-pox, never fail to accompany the second stage of confluent small-pox—namely, *sore throat* and *salivation*. The tonsils and the fauces become tumid and red, the face begins to swell, and then the salivary discharge begins either with the eruption or within a day or two afterwards. The discharge of saliva is at first thin and copious, resembling the ptyalism of mercury. About the eighth day, however, it becomes viscid, and is expectorated with difficulty; while in bad cases it either ceases for a day or two, and then returns, or it disappears altogether abruptly; and if the swelling of the face also subsides suddenly, the danger is great. Children are not so liable to this salivation as adults. In them, however, a vicarious diarrhoea often appears, but not constantly; neither does it occur so early in the disease. It is frequently profuse, and often proceeds till the disease terminates. Not unfrequently the larynx and trachea are implicated, even to the larger divisions of the bronchia. There is then cough, with hoarseness, painful expectoration, and sometimes complete extinction of the voice. These are most dangerous symptoms when the eruption is confluent. It has been stated that, on the appearance of the eruption and the commencement of the third stage, although the fever is mitigated, it does not altogether subside, defervescence is incomplete, and the affection of the head, the frequency of the pulse, and greater heat of the surface, often continue. With these ominous symptoms still present, on the eighth day of the eruption, or the eleventh day of the fever, the fourth stage, or secondary fever, commences, bringing with it new sources of anxiety to the physician and of danger to the patient. Drs

Gregory and Watson (of the Foundling Hospital) both consider the eighth day of the eruption as the most perilous day of the disease. Blood often appears in the urine in slight and sometimes in large amount. Renal cylinders are not uncommon. The bladder is affected in a great number of cases, and there is increased mucus in the urine, which, if it be retained in torpid and semicomatose cases, soon becomes ammoniacal, as in cases with catarrhal cystitis (PARKES, *On the Urine*, p. 262). "The confluent small-pox," says Sydenham, "does not in the least endanger life in the first days of the illness, unless there happens a flux of blood from the urinary passages, or from the lungs. Yet, on the decline of the disease, or on the eleventh, fourteenth, seventeenth, or twenty-first days, the patient is often brought to such a state that whether he will live or die is equally uncertain." He is first endangered on the eleventh day by high fever (and the highness of the temperature may indicate the danger), attended with great restlessness, and other symptoms which ordinarily prove destructive, by *adynamia* and fatal paralysis, not unfrequently accompanied by an acute hæmorrhagic condition, the contents of the pustules becoming bloody, and petechial on such parts of the skin as may be free from eruption—*epistaxis*, *hæmaturia*, *hæmoptysis*, or *hæmorrhage* of the uterus may also supervene. "But should the patient outlive this day, the fourteenth and seventeenth are to be apprehended, for a very vehement fit of restlessness comes on every day towards evening, and there is the greatest difficulty in saving him." The disease is apt to prove fatal by way of apnoea after the eighth day; but after that period the characters of asthenia supervene.

The unfavourable symptoms of the fourth stage are, the absence of the usual redness in the intermediate spaces, the non-intumescence of the face, the suppressed salivation, cough, with hæmoptysis, or hæmaturia, and great restlessness. Sometimes other symptoms are added to these, as a brown tongue, delirium, petechiæ, or a black spot in the centre of each pock, scarcely so big as a pin's head; or a disposition to gangrene round the larger vesicles. When these symptoms are present, few patients survive such a crisis, dying early with symptoms of great prostration; or if life continue longer, pleurisy or pneumonia may be eventually fatal. In some cases, however, the event is favourable, and the patient is restored; but the struggle is sharp, and the convalescence long; even in the most favourable cases a slow and protracted convalescence is the rule. An endless series of abscesses may form, or inflammation of a joint may take place, and produce lameness; ulceration of the cornea, blindness, otitis, or deafness may also ensue; while the deeply-scarred face is a lasting record of the severity of the disease, and of the great danger the patient has survived. It is the most malignant form of small-pox. A dark crust sometimes forms, which covers the face like a mask, which is constantly being added to by the continued suppuration beneath, until it falls off, leaving a cicatrix like that of a burn, and equally tending to induce deformity by contractions.

The Inoculated Small-Pox.—The phenomena which result from the introduction of the variolous poison by means of inoculation into the cutis differ in many respects from those that occur in the natural small-pox. They are as follow:—On the day after the operation is performed little

alteration is discovered in the punctured part. On the second day, however, if the part be viewed with a lens, and the operation has succeeded, there generally appears an orange-coloured stain around the incision, while on the fourth or fifth day the part is hard, slightly inflamed, and itches. A vesicle containing serum is formed on it. About the sixth day some pains and stiffness are felt in the axilla, symptoms which foretell the near approach of the fever and the favourable progress of the disease. On the seventh day the vesicle becomes more developed, and the red areola forms round its base. The operation having now been performed about seven days (the usual period of latency of the poison when so inoculated), and the vesicle having existed four days, the ordinary symptoms of primary fever appear. This fever lasts three or four days, when the general eruption follows, now called the secondary eruption, the pustules coming out, as usual, in three successive crops, on the face, trunk, and lower extremities. On the day of the general eruption the primary pustule, says Dr. Gregory, is distended with matter, and proceeds on its course, so that it is scabbed when the secondary eruption is only about to mature. The most remarkable phenomena, however, of the inoculated small-pox are the singular mildness of the fever, and the diminished number of the pustules of the secondary eruption. The mildness of the fever is thus instanced by the late Dr. Watson, of the London Foundling Hospital,—“Of the seventy-four persons whose histories I have related, though inoculated with variolous matter in different states, although prepared in so different a manner, and a great number not otherwise prepared than by an abstinence from animal food, not one of them were disordered enough during the whole progress to occasion the least anxiety for the event; not one of them had their eyes closed a single day, from the pustules being upon the eyes or near them; none continued in bed an hour longer than they would have done in their best health.” The number of pustules from inoculated small-pox is subject to great variations, but, with very few exceptions, is much less than in the natural small-pox. In some cases not more than two or three appear; occasionally only the primary pustule is seen; but more generally the number varies from ten to two hundred, the mean being thirty or forty. Such is the general course of the inoculated small-pox. In a few instances, however, the disease that follows this operation is extremely severe; in a still smaller number it is confluent; and in either case the patient is perhaps destroyed. Many theories have been propounded to explain the singular mildness of the inoculated small-pox, but none of them are satisfactory.

Complications of Small-Pox and Special Morbid Tendencies.—About the eighth day in the distinct small-pox, and the eleventh day in the confluent small-pox, a secondary fever is established, and at the same time a new series of phenomena (already indicated, but requiring more special notice) may present themselves in a few severe cases,—as affections of the lungs, of the pleuræ, or of both; of the urinary organs, or of the areolar tissue of the body generally. It is during the progress of this *secondary fever* that the case may assume a fatal type. Complete deservescence is never established; but lesions become developed, whose advent may be appreciated by careful records of morning and evening temperature during the progress of this *fever of suppuration*. These are the *tertiary affections*,

the eruptions and the fever being the *secondary effects* of the specific poison.

The most frequent indication of lung complication is hæmoptysis. Occasionally inflammation of these organs takes place, generally as a pleuro-pneumonia, indicated by a sharp pain in one side, increased dyspnoea, harassing cough, and bloody expectoration. The mucous membrane (for instance, of the trachea) is often found covered with a thick semi-purulent, muciform matter, peculiar to small-pox, irregular or honey-combed at its free surface, which, being removed, the subjacent tissue is found diffusely inflamed. The pleura also is peculiarly disposed to inflammation—suppurative or hæmorrhagic—which comes on about the eleventh or twelfth day, for the most part very suddenly, and proceeds rapidly to empyema, sometimes destroying the patient in thirty-six hours. The tertiary action of the variolous poison on the urinary and genital organs is seen in the frequent occurrence of hæmaturia, in the occasional formation of abscess of the kidney, in the occurrence of peripheric and parenchymatous orchitis, and in ovaritis; while its action on the uterus is manifest from menorrhagia in the unimpregnated state, and by frequent miscarriage when the patient is pregnant. The areolar tissue of the body generally is also frequently implicated. In some cases examined a few hours after death, the bodies can with difficulty be laid on the table, the skin being detached by the pressure necessary to raise them; and the serous coat of the intestines separates from the mucous and muscular coats with the greatest facility for many feet, and apparently might be entirely peeled off. In some cases the finger can be thrust through the walls of the heart with ease, the tissue of that organ having become unnaturally soft and broken down from fatty degeneration. The affection of the areolar tissue generally is seen in the great tendency to the formation of abscesses on the subsidence of the eruption. Twenty, thirty, and even more small boils or abscesses will sometimes form on a limb or other part of the body, in most formidable succession, which, on being opened, are found to contain sanious, or, only in a few instances, laudable pus. In a case of *septicaemia* occurring during the course of confluent small-pox, examined by Dr. Parkes, the disease ran its course well till the eleventh day, when there was shivering; on the following day there was bilious vomiting; on the fourteenth day there was sudden pain in the right wrist, and swelling of many joints; and on the following day there were all the well-marked symptoms of pyæmia—pyogenic fever of small-pox. A daily examination of the urine showed the remarkable fact that the amount of sulphuric acid passed continued progressively to increase daily, rising from 23·8 grains to 44·4 grains (PARKES, *On the Urine*, p. 267).

Sequelæ.—The different lesions that have been mentioned are not the only miseries from which the patient may suffer. These are often followed by sequelæ even more formidable than the preceding phenomena, such as blindness, deafness, or lameness. With respect to blindness, although “the eye seems to possess a complete immunity from the small-pox eruption, and although it sometimes extends to the inner margins of the eyelids, the particular local affection that causes the destruction of the organ of vision in variola begins generally on the eleventh or twelfth day, or later, from the first appearance of the eruption, and when the pustules in every other

part of the body are subsiding. It comes on after the secondary fever has commenced, with redness and slight pain in the part affected, and very soon an ulcer is formed, having its seat in the ocular conjunctiva, almost invariably at the margin of the cornea. This continues to spread with more or less rapidity, and the ulceration passes through the different layers of the cornea, until the aqueous humor escapes, or till the iris protrudes. In the worst cases there is usually hypopion, and when the matter is discharged, the crystalline lens and vitreous humor escape. In some instances the ulceration proceeds very rapidly. More than once Mr. Marson has seen the entire cornea swept away within forty-eight hours from the apparent commencement of the ulceration; and now and then the mischief goes on without the least pain to the patient, or his being aware that anything is amiss with his eyes. Hence the *hypopion* may be overlooked. Further, Mr. Marson calculates that in 1,000 cases 26 had ophthalmia, or about 1 in 39; and of these 11 lost an eye each, or 1 in about 100. The inflammation of the buccal membrane may extend to the Eustachian tube, causing suppuration of the ear, and sometimes permanent deafness. It may spread to the glottis, so that the patient has been known to die suffocated by effusion into the areolar tissue around it, and so closing the aperture. Sometimes it has terminated in ulceration, with the loss of a portion of the nose, or in caries of the jawbone, or in enlargement of the glands of the neck. The soreness of the fauces and tonsils is often associated with pustules on these parts; and the tongue, the roof of the mouth, the inside of the cheeks, the uvula, and the *velum palati* may be covered with an eruption like pustules; and it has been much disputed whether the eruption forms on any other part of the mucous membrane. As a general rule it does not; but Martinet found, in a man that died on the eighth day, the rectum covered with what he supposed to be variolous pustules. Rostan has seen the alimentary canal garnished with pustules similar to those of the mouth, from the œsophagus to the rectum. Sir Gilbert Blane met with pustules on the mucous membrane of the intestines in two persons who died in the West Indies; and Rayer has given a plate representing pustules on the mucous membrane of the trachea. Dr. Mead recorded that he had "seen subjects in which the lungs, brain, liver, and intestines were thickly beset with pustules." Dr. Pitzholdt, in the *Morbid Anatomy of Small-Pox*, writes that he has seen the peritoneum covering the liver and the spleen presenting appearances which he felt justified in regarding as the product of small-pox. May not some of these have been the results of pyogenic fever (p. 454, *ante*)?

The pustules which form on the mucous membrane of the intestine, however, have not been very distinctly studied either as to their course or phenomena. Rayer terms them *rudimentary* pustules. A case of small-pox recorded by Dr. George Patterson, of Edinburgh, was examined by one of the most learned and discriminating pathologists of the day, Professor W. T. Gairdner. He observed pustules on the mucous membrane of the colon identical with the pustules on the skin (*Edinburgh Monthly Journal*, 1849, p. 549). Still it appears to be doubtful whether such eruption on the mucous membrane of the intestine is not the same as that seen in cholera cases, extending (as I have frequently seen it do in cases I examined in the hospitals at Scutari, in 1855) throughout the whole

intestinal tract. The appearance of eruption in such cases is due to the solitary mucous glands, which are filled with exudation, not of a purulent kind, but having all the external appearance of pustules. Such are the pathological phenomena which occasionally complicate small-pox. Death, however, not unfrequently anticipates their action, and destroys the patient during the primary fever, and before any of these lesions, not excepting the eruption, are set up.

Group B.—Small-Pox after Vaccination—Varioloid, or Modified Small-Pox.

Modified Small-Pox.—*In this form pustules are cut short in their development by vaccination or previous attacks of small-pox.* It has been already noticed that during the epidemic prevalence of small-pox, even before vaccination was known, cases of small-pox occurred in a very modified form: such as the occurrence of variolous fever without the eruption (*variola sine variolis vel eruptione*); or the occurrence of small-pox in which the eruption continued vesicular (*the crystalline pock*); and lastly, the occurrence of small-pox in which the vesicle dried up instead of becoming a mature pustule. These cases were known as stone-pock, horn-pock, and wart-pock (*variola, verrucosa, vel cornea*). Modern pathology now regards these varieties as the result of the modifying influence of vaccination where vaccination has been performed; and they may now all be described and classed under the common name of "*varioloid*." Comparative mildness of symptoms and course is their great characteristic, the pustules being "cut short in their development by vaccination or a previous attack of small-pox." There appears to be every variety of the modification, of which the principal are:—(1.) A fever of three days, without eruption, affecting people during variolous epidemics. (2.) A high and severe fever, followed by a very mild eruption, sometimes only a single pock; the slight proportion which the amount of eruption bears to the severity of the preceding fever is perhaps the most marked characteristic of varioloid. (3.) The occasional appearance of a scarlet efflorescence like that of scarlatina or roseola, preceding the appearance of the proper pimples, which occur as a very scanty crop. (4.) In some rare instances the eruption is confluent, but does not advance beyond the development of a pimple or vesicle, and begins to dry on the fourth or fifth day of the eruption, forming a small hard tubercle, which soon disappears. (5.) Sometimes the eruption is pimple, vesicle, and pustule at one time in the same case. (6.) Sometimes the eruption runs its regular course, but stops sooner, sometimes on the sixth or seventh day, instead of the eighth or ninth. In general, it may be stated that the severity and fully developed state of the disease is in proportion to the length of time which elapses from vaccination (COPLAND). (7.) The varioloid eruption wants the peculiar odour of natural small-pox, and secondary fever is very rare. (8.) Other eruptive affections—such as measles, scarlatina, purpura—materially modify the course and symptoms of small-pox. Generally, it may be stated that after an intense continuous fever, lasting a few days, a final exacerbation terminates the fever suddenly and simultaneously with the development

TYPICAL RANGE OF TEMPERATURE IN A CASE OF SMALL-POX MODIFIED BY VACCINATION. THE RECORDS INDICATE MORNING (A.M.) AND EVENING (P.M.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE SECOND DAY (Wunderlich).

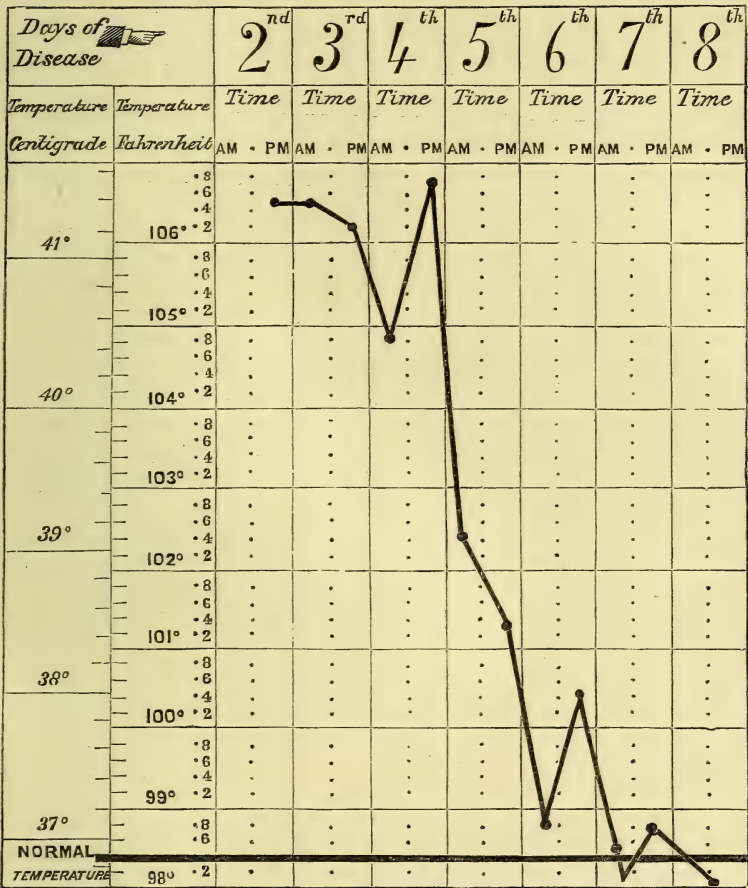


Fig. 81.

of the small-pox pimples. A rapid and perfect defervescence then ensues, the temperature decreasing seven or even more degrees (Fahr.) within thirty-six hours. From this event the patient remains entirely free from fever—*provided there exists no serious complication*—in spite of the continuous and progressive development of the small-pox pimples into pustules, and even in spite of the successive eruption of new pimples.

Exhaustion of Susceptibility.—Small-pox has the property, in common with measles and scarlet fever, of exhausting, after one attack, the susceptibility of the constitution for the rest of life to any future action of the poison. The nature of this protection is still unexplained (see p. 391, *ante*). The disease, artificially induced by vaccination with cow-pox matter, has a similar effect upon the constitution, and furnishes the basis

of its protective influence. This law, however, is not without some exceptions, and in an epidemic at Marseilles, Bosquet considered that one person in one hundred was attacked a second time with small-pox. In some few instances even a second attack has had no protective influence (see previous edition). Hence also, vaccination, while it invariably modifies small-pox, does not always absolutely prevent its occurrence in the person vaccinated, especially if they have been vaccinated early in life, and exposed late in life to infection. Hence vaccination ought to be repeated in adult life (eighteen to twenty years of age) after having been performed in infancy.

Co-existence of Small-Pox with other Morbid States.—The variolous poison is capable of *co-existing* with many other poisons, and also of influencing their actions, and of being reciprocally influenced by them. Dessessarz has seen variolæ co-exist with scarlatina and with whooping-cough; Cruickshanks with measles; Frank with psora; and Dimsdale with syphilis. A patient was admitted into St. Thomas' Hospital with tertian ague, writes Dr. Robert Williams; the ague subsided and small-pox appeared; small-pox having run its course, the ague immediately returned. Ring mentions a case of triple disease co-existing—namely, small-pox, measles, and whooping-cough—and that they all ran their course together. In the *British Medical Journal* for May, 1868, an instance of the co-existence of small-pox and scarlet fever is related by Dr. Sansom.

Cause.—The same obscurity hangs over the cause of small-pox as over that of many other diseases, such as of measles and of scarlatina. There is every probability, however, that these diseases have now no other mode of communication than from one person to another. The source of contagion or infection in small-pox can always be traced where there is no peculiar obstacle in the inquiry. It spreads solely by a material *contagium* or *virus*. There are some grounds for believing, however, that small-pox, in common with some other distempers, originated in the lower animals, and extended from them to the human species by infection or contagion. Sheep, we know, are liable to a distemper of the nature of small-pox; and there is every reason to infer that the disease is perpetuated by its own specific poison or virus, which spreads it about by the media of impalpable substances technically called "*fomites*," which are capable of receiving, preserving, and carrying the germs of the disease. By such impalpable means the disease has been propagated since its first appearance in the world. The poisonous material of small-pox is given out from the mucous and cutaneous surfaces of a patient, especially from the lungs and skin, from the exhalations, the secretions, the excretions, the matter in the vesicles and pustules, and the scabs. These contain the noxious germs of the disease, which may attach themselves to bed-clothes, body-clothes, and especially to woollen, cotton, and felted articles. Such stuffs retain the specific poison for a very long but undetermined period: any number of years, so far as is known—just as the hat, cap, and coat worn in a dissecting-room retain the peculiar effluvia of that place for a very long period. The public cabs in large towns are also frequent means of propagating small-pox. The poison is most tenacious of vitality. Drying preserves it; and if protected from air it remains active for an unknown

number of years. It is not yet determined at what period this poison is first generated by the patient's person, whether during the primary fever, or not till after the eruption has appeared; but it probably begins to form and multiply in the patient during the primary fever. There are, indeed, some facts which render it probable that a person in whom the disease is incubating, and who has no symptoms, may impart the disease (MURCHISON). Generally, it may be stated that the poison is most powerful when it is most manifest to the sense of smell, and at the period when the clear contents of the vesicles begin to turn cloudy that the dried crusts of the pustules or scabs possess the power of communicating the disease, and retain this power for a very long time. It is unsafe for a susceptible person—*i.e.*, a person who has not been vaccinated, or has not had small-pox—to be in the same room, or in the same house, with a patient labouring under the disease. It has been caught by passing a child ill of small-pox in the street; and “to expose a person in the public highway, infected with this contagion, is considered a common nuisance, and indictable as such.” The dead body of a variolated person is equally infectious, and students who have been near it when brought into the dissecting-room have in consequence had the disease communicated to them, although they may not have touched the body (CÆSAR HAWKINS). In my own experience I have known students take small-pox from dissecting small-pox subjects. The *infecting distance*, therefore, must be many yards around the subject of small-pox lesions. Indeed, with every precaution, there is great difficulty in preventing it spreading from ward to ward in large hospitals during the prevalence of the disease. “There is no contagion so strong and sure as that of small-pox; none that operates at so great a distance” (WATSON).

The fact that small-pox is communicable from person to person has been fully demonstrated by the once general practice of inoculation. The poison by this operation has been proved to exist in the serum, in the pus, and in the crusts of the small-pox pustule. There is no law more singular and unexpected, in the whole range of morbid poisons, than that the introduction of the variolous poison by means of the cutaneous tissue, should produce an infinitely milder disease than when the same poison is absorbed by a mucous tissue. The poison then seems to be much more uncontrollable in its operations, as in the case when it affects a person who breathes an infected atmosphere, compared with one who has been *inoculated* with the small-pox poison inserted beneath his cuticle through a puncture of the skin.

The causes which predispose to small-pox or increase the susceptibility of infection are,—(1.) A very early age. (2.) Not having had the disease before. (3.) Not having been vaccinated: such are called “unprotected persons.” (4.) Peculiarity of constitution—*e.g.*, the negro and dark races. (5.) Fear of infection. (6.) Epidemic influence.

It is gratifying to know that of recent years the prevalence and mortality of small-pox in this country are greatly less than were wont to be. Dr. Farr tells us that, for the three years previous to 1855, out of every 1,000 deaths from all causes, only 7·607 were from small-pox. At present (1879) an epidemic has prevailed and extended in London during the past eight years, after having prevailed in Paris; and which

not improbably had its commencement from the influx of Parisians to London, in consequence of the Franco-Prussian War and the siege of Paris by the Germans.

Prognosis and Cause of Death.—The prognosis in cases of natural small-pox is always most grave. The danger may be measured, to a certain degree, by,—(1.) The quantity and confluence of the eruption; (2.) The state of the circulating fluids; (3.) The presence and nature of the complications, especially those of the respiratory organs and nervous centres; (4.) Age and habit of body of the patient; (5.) Nature of the epidemic, as to its virulence, which may prevail. Natural small-pox in unprotected persons is generally very fatal. The deaths average one in three. The fully-formed confluent small-pox is always a very dangerous disease. About ONE in TEN PATIENTS die of distinct natural small-pox; and ONE to THREE PER CENT. OF PATIENTS only of small-pox after inoculation or after vaccination. The calculation of the proportionate number of deaths, however, appears to have greatly varied in different years, without any cause that can be found out. There are certain signs regarded as unfavourable,—for example, excessive lumbar pains continuing; the persistence of high temperature and of vomiting after the appearance of the eruption; the occurrence of delirium, convulsions, or coma in adults during the primary fever; great confluence and simultaneous appearance of the eruption over the whole body. Such unfavourable signs are not necessarily fatal; but unfavourable signs which appear during secondary fever forebode, with greater probability, a fatal end. These are,—the absence of the usual redness of the skin in the intermediate spaces between the pustules; the distribution of petechiæ in the interstices; the development of a black spot hardly so large as a pin's head in the centre of each pustule; a livid or purple colour of the pustule; a disposition to gangrene in the larger vesicles; imperfect development of the pustules, or their sudden subsidence, without remission of symptoms; sudden suppression of salivation; sudden suppression of urine; hæmaturia; cough with hæmoptoe; absence of swelling in the hands and feet when the eruption is copious; tendency to the formation of abscesses (*pyogenic fever*) after desquamation has commenced; congestive pneumonia, or bronchitis, with livid lips, face, or extremities, with hoarseness or complete aphonia. Recovery may take place even although the first-mentioned of these unfavourable signs exist; but convalescence is likely to be retarded by ulcerations of the cornea, asthenic ophthalmia, purulent deposits in the joints, ulceration of cartilages, otitis, abscesses and suppuration in the areolar tissue under the skin. The development of scrofula and phthisis is apt to follow small-pox, even though no unfavourable symptoms occur. In pregnant women the disease is always dangerous, often fatal, almost always produces abortion; and the foetus so parted with not unfrequently bears evidence of the eruption upon the skin. The most common causes of death are due to combinations of the unfavourable signs already noticed; and the greatest number die on the eighth day of the eruption; or the eleventh day of the fever is the most fatal period (GREGORY). In private practice, Dr. Wood, of Pennsylvania, considers the period between the twelfth and eighteenth day as the most dangerous to life. The greatest mortality from small-pox is at early ages,—for example, before

the fifth year. Dr. Farr estimates that out of every 100 deaths from small-pox, 75 are below that age. It is next most fatal in persons who have passed middle life; and least fatal from 10 to 15 years of age.

Diagnosis.—It is not possible to distinguish, except by careful records of the temperature, the primary fever of small-pox from that incident to many other diseases with eruptions, or from the first stage of continued fever. It is for the most part characterised by excitement rather than depression; and in the adult the muscular pains and pains in the back and loins are more severe and intense than in ordinary fever. The pain of the back is central in its position—a *spine-ache*—and is less affected by change of posture than the pain which is characteristic of lumbago, which affects the muscles at the side of the spine (often on one side only, and which is much aggravated by movement) (BARCLAY). If vomiting occurs, which cannot be ascribed to any obvious cause, and persists till a papular eruption appears on the third or fourth day, with a remission of the febrile symptoms, little doubt can exist as to the variolous nature of the disease.

The diseases with which small-pox may be, at first, confounded, are *petechial eruptions*, *measles*, *chicken-pox*, and the *secondary pustular eruptions of syphilis*. Small-pox is to be distinguished from *measles* by the symptoms, as well as by the form and successive changes of the eruption. Crescentic patches, terminating in desquamation on the fourth day, characterise *measles*, as compared with small-pox, the eruption of which, even although it may be at first in efflorescent patches, never fails to become vesicular and pustular, proceeding to suppuration or blackening on the eighth day—a process which never fails to be attended by secondary fever. It is more difficult to diagnose between varioloid and varicella, or chicken-pox. The chief difference consists in the eruption of chicken-pox presenting a vesicular character, which it retains; and it does not proceed to suppuration, but completes its course in five or six days, with a mild and short symptomatic fever.

The combination of mercurial poisoning, scrofula, and syphilis may give rise to cutaneous eruptions attended with fever, which may, in the first instance, be mistaken for commencing small-pox. The eruption, however, is more tedious in its development, irregular in its course, and is persistent. It is recognised by the history of the case, the long duration of the eruption, and the deep red or copper colour it generally presents.

Treatment.—Since the first accounts by the Arabian physicians of the ravages of small-pox in Mecca, the history of this disease may be considered in three great eras, each of which is characterised by remarkable epochs, and a fourth may be said to be running its course now. The first of these eras is marked by a commencing epoch of improvement in the treatment of small-pox. In few diseases has medical opinion undergone a more obviously beneficial change, and to Sydenham is due the merit of this revolution in medical practice. The epoch of the second era is marked by the discovery of the singular and beneficial phenomenon that the virulence of the poison of *small-pox* was greatly mitigated by introducing or engrafting the disease into the system, through the cutaneous tissue, thereby causing the transference of the disease from one person to another, by *inoculation*. To

Lady Mary Wortley Montague is due the merit of having introduced the practice of inoculation into this country in 1722—a deed which must be considered as one of great heroism, when measured by the knowledge possessed by the physicians of those days. The third great era in the history of small-pox is marked by the epoch of that remarkable discovery which has rendered the name of Jenner immortal—namely, the modifying and protecting influence of *vaccination*. He found that a certain disease in the cow, known as the *cow-pox*, could be transferred to the human subject by inoculation; and that, having been so transferred, it modified, to a considerable extent at least, the course of the disease, if it did not altogether prevent in the human subject the occurrence of small-pox in its natural state. A fourth era may be said to have commenced in this country almost imperceptibly. It may be described as marked by an epoch of transition, in which doubt and scepticism have arisen as to the efficacy of vaccination; tending to propagate an erroneous popular belief; and consequently, leading to the ineffective adoption of means which practically have been proved to be sanative in the highest degree. In other countries, on the contrary, and especially in Central Europe, this present period is marked by implicit faith in the virtues of vaccination, and the successful legal enforcement of this sanative measure. The most plausible objections to vaccination must now give way before the one great fact that, during the last century, ONE-TENTH of the population died of small-pox, while *another tenth* were disfigured for life by the disease; and that since the introduction of vaccination, the general mortality from all diseases is less, and that of small-pox is reduced to a minimum.

An account of the treatment of small-pox resolves itself, therefore, into a consideration of two topics, namely,—(1.) The usual therapeutic, curative, or sanative treatment of the disease; (2.) The sanitary treatment—*i.e.*, the means of protecting individuals from the *small-pox*; or of modifying the influence of the malady by inoculation or by vaccination. Of these in their order,—

I. Therapeutic, Curative, or Sanative Treatment of Small-Pox.—The main object, in the first instance, is to prevent, if possible, a copious eruption; for the severity and danger of the disorder may be measured, in some degree, by this. The vulgar belief, that “better out than in,” does not apply in the case of small-pox. The great object is to reserve the strength of the patient; and the attentions of an experienced nurse are demanded. A third indication is to watch for and deal vigorously with intercurrent inflammatory action, which is apt to be set up. It has hitherto been the belief and orthodox teaching, that the disease is not under the influence of any specific or antidote; that there is no remedy which will *cut short* the disease; that it must run its course; and that it is the business of the physician to assuage the untoward symptoms and avert the dangerous accidents which may arise, by all the most approved methods of treatment, in accordance with the science of medicine of the present day. That there are grounds for modifying this belief and dogmatic teaching, the following statements will show:—The chief physician of Iceland, Dr. J. Hjaltelin, believes that it may be stamped out by the action of *sulphurous acid gas*; and relates how, in its malignant form, it was so treated, and stamped out of that island without a single person of the inhabitants

becoming infected. The disease was imported into Iceland by French fishing vessels coming into the harbour of Reykjavik, to ask for medical aid, having on board men sick of small-pox. Vaccination and revaccination were immediately instituted; and government at once erected a quarantine hospital, to which the cases were taken from the ships. The epidemic showed a high degree of malignancy; and Dr. Hjaltelin, being a firm believer in the power of disinfectants and antiseptic remedies, resolved to try such remedies, one after another; the only question being whether to use those compounds which acted by *oxygenising* or by *disoxygenising*. He ultimately decided to use the *disoxygenating* antiseptics, as being the most safe; and fixed upon *sulphurous acid* as less irritant for the respiratory organs than chlorine. He used it externally by fumigation, by burning refined *sulphur* in the sick rooms, and internally by giving *sulphurous acid* mixed with pure water—the usual dose being ʒi , mixed with one ounce of water, and repeated every third hour. The patients at first disliked the fumigation, from the strong and pungent odour of the sulphur; but they soon felt its good effects and desired to have more. These good effects were,—the heat of the eruptive fever diminished, and the vesicles dried very quickly, leaving the skin covered with thin brown scales, which soon fell off. Out of 22 patients so treated he only lost one, who came to the hospital moribund, and who died thirty-nine hours after arrival. Seven had confluent small-pox; and of these three were in the suppurative stage. Others were cases of discrete small-pox having the eruption of vesicles abundant. He thus sums up the good effects of the disinfectant method of treatment by *sulphurous acid* externally and internally:—(1.) It mitigated the primary and secondary fever; lowering the temperature, and thirst was diminished, even when fever ran high. (2.) The general symptoms were also lessened in severity, such as the headache, backache, and pains in the joints. (3.) He believed the sulphurous acid and vapours had a destructive influence on the small-pox virus. Its destructive effects on parasite life are undoubted (*Brit. Med. Journal*, Nov. 4, 1871, p. 519).

Treatment by Vaccination.—There are also some grounds for believing that *vaccination* and the hypodermic injection of vaccine lymph “may curb the small-pox,” even when performed at a period *subsequent* to the reception of the variolous poison; and the observations and practice of Mr. R. C. Furley, detailed in the *British Medical Journal* for June 8, 1872, show that vaccination will modify small-pox *after the eruption has appeared*. An impression prevailed that it was dangerous to be vaccinated while small-pox was prevalent; and also that it was of no use to vaccinate so long as the former vaccination mark was quite distinct. Both these ideas are opposed to facts. On the contrary, Mr. Furley’s cases show that vaccination, and the injection of vaccine lymph, especially if done about the second or third day of the disease, possess valuable curative powers, so that a disease which promises to be most severe is curbed or modified. Mr. Furley uses a hollow needle for injection, the bone of the tube of which is large enough to admit a vaccine tube, from which the lymph is forced directly into the circulation. He injects hypodermically in both arms.

Dietetic and General Treatment.—In the first instance, the course to be pursued is for the physician to act on the defensive, and simply protect his

patient from certain injurious influences to which he may be exposed,—such as, heating drinks to force out the eruption, which are apt to be given by ignorant and officious friends. Throughout the whole course of the disease, the diet should be strictly limited to slops, sago, arrow-root, and ripe fruits, with cold water or lemonade for drinks, with or without ice. The chamber in which the patient lies should be cool, not above 60° Fahr., and freely ventilated. The bed-clothes should be light, the body-linen daily changed; and, when the disease is long, the patient's back should be often examined to prevent sloughing. The scalp, likewise, should be examined, and, if full of pustules, the hair should be cut off, to prevent its matting. If the disease be recognised early, however, it is proper to shave the scalp, because the irritation which attends the suppuration of the pustules is thereby diminished, and cold may be more efficiently applied to the head, if necessary. In the early stage of the primary fever, in severe cases more especially, it is necessary to have the bowels well opened in the first instance, and to keep them regularly moved daily by saline medicine, or an enema of *three parts water to one of vinegar*. A cathartic pill, composed of the following ingredients, will be found to be efficient in most cases, especially if aided by a Seidlitz powder, given six or eight hours after the pill; namely,—Two grains of *calomel*, one grain of the *compound extract of colocynth*, one grain of *gamboge*, and one grain and a half of *scammony*, made consistent with a little aromatic oil. The bowels must be daily attended to, and *castor-oil*, or *rhubarb*, or *magnesia*, may sometimes be required. Saline diaphoretics, in the form of James's powder; or the *aqua acetatis ammoniæ*, to which a grain or two grains of *tartar emetic* has been added, so as to have $\frac{1}{8}$ or $\frac{1}{16}$ of a grain in every tablespoonful of the mixture, is an efficient and cooling diaphoretic. *Spirit of nitric ether*, or the *nitrate of potass*, may be added if required. The surface of the body, over the hands, face, and feet, ought to be sponged several times a day with tepid water mixed with *Condyl's fluid*, or with *sulphurous acid*, with a view to relieve the intolerable itching; but caution is necessary to prevent exposure to cold. *Cold-cream*, or a liniment of *olive oil*, *glycerine*, and *lime-water*, or of *Carron oil* (*linimentum aqua calcis*), smeared from time to time over the itching surface, by means of a camel-hair pencil, may be found to afford relief; and *chlorine lotions* are highly spoken of by Eisenmann. With regard to the occurrence of convulsions in children, it is *not* found that opiates, as recommended by Sydenham and Cullen, are expedient. When the children are robust, or previously in good health, *local bleedings*, by means of one or two leeches to the temples, are more beneficial. Delirium, violent screaming, intolerance of light or sound, heat of head, all of which indicate a tendency to meningeal congestion, still more clearly warrant the application of *leeches*. With regard to the propriety of bleeding (general) in adults, it is now well ascertained that it will neither eradicate the fever nor diminish the amount of the eruption; moreover, experience has now amply shown that loss of blood is badly borne in all infectious diseases. The persistent application of cold to the head, and repeated affusion of the body with cold water, is to be preferred, in cases where there is much cerebral excitement. When delirium, with restlessness, wakefulness, and a frequent pulse, is continuous, *an opiate*, or the free use of alcoholic stimulants, are indicated; and *opium* is most advantageously given combined

with *tartar emetic*. A draught, composed of *thirty minims of the solution of muriate of morphia*, with *half a grain of tartar emetic*, will be found beneficial in such conditions, especially when given at bed-time. If the temperature rise higher, large doses of *quinine*, 3 to 5 grains, every three or four hours, are to be given. Cooling drinks, with or without ice, of *lemon-juice*, *tamarinds*, *neutral effervescing powders*, are always agreeable to the patient, who ought, for the sake of coolness, to be very lightly clothed. After the eruption has fully appeared, this is all which in ordinary cases requires to be done, and if, towards the tenth or eleventh day, there is much restlessness or sleeplessness, *an opiate* may be given.

In the complications which sometimes ensue, such as *inflammation of the throat and base of the tongue*, opiates are found to be injurious. The general treatment must be by *cathartic or purgative clysters*, if swallowing is difficult. In the other inflammations, however, opiates are of the greatest service, provided the symptoms be not those of cerebral oppression or bronchitis. In bronchitis, nauseating doses of *antimony* every hour sometimes procure relief; and if relief does not follow in the course of thirty to thirty-six hours, stimulant doses of *mistura ammoniac* with *squills* ought to be given every second hour to promote free expectoration.

In the advanced stage of the secondary fever the strength of the system requires maintenance and support; because the abundant suppuration and extensive cutaneous irritation combine to exhaust the strength, as shown by the weakened pulse, the dark and dry tongue, blueness, paleness, or coldness of the extremities. Tonics, stimulants, and generally nutritious diet, are now called for. *Quinine*, *mineral acids*, *malt liquors*, especially the *light bitter ales*, *light porter*, *wine*, and even *brandy*, may be demanded. The diet should consist of *milk*, strong *animal broths*, *eggs*, raw or lightly boiled, according to the discretion of the physician, and regulated by the digestive powers of the patient.

The development of a severe eruption on the conjunctiva is to be especially guarded against, by the assiduous employment of cold water applications, or by compresses moistened with a weak solution of *corrosive sublimate* (*one grain to six ounces of water*, with or without the addition of *muriate of ammonia*) (NIEMEYER).

To prevent the face from being seamed, scarred, or "pitted" by the suppuration of the pustules, has taxed the ingenuity of physiologists and physicians. It has been stated that the influence of the atmospheric air is essential to the development of the pustules, and, accordingly, anything which would effectually exclude this influence would prevent the occurrence of a scar. But it is evident that the chance of scars can only be diminished by those means which are calculated to allay the general violence of the disease. When the eruption is severe, it is almost impossible to prevent the formation of "pits," because the depression results from the expulsion of a small slough; and the more mild the suppurative inflammation can be rendered, so in proportion will the chance of sloughing, and therefore "pitting" be diminished.

The local means which have been adopted to prevent "pitting" may be shortly stated as follow:—(1.) To open each individual pustule after suppuration has commenced. (2.) To cauterise the pustules with nitrate of silver. (3.) To employ both methods—that is, to open each of the

pustules when it becomes vesicular, and introduce a strong solution of nitrate of silver into the cavity of the vesicle. At the end of a week scales fall off, and no pit is left. Or, lastly, to paint the face with a solution of nitrate of silver, in the proportion of one drachm of the nitrate to the ounce of water. (4) The application of a mercurial plaster, with the view of producing resolution of the *papulae*. The preparation in use for the purpose at the Children's Hospital in Paris consists of twenty-five parts of mercurial ointment, ten parts of yellow wax, six parts of black pitch. With this plaster the face is to be covered, and so to remain for several days. (5.) Sulphur ointment, applied several times a day. (6.) Calamine mixed with olive oil, to form a coherent crust (BENNETT). (7.) Tincture of iodine, painted over with a brush. (8.) Saturated solution of gutta percha in chloroform (Drs. GRAVES and WALLACE). (9.) To smear the face over with common olive oil. (10.) Skoda prefers compresses moistened with solution of *corrosive sublimate* (*two to four grains in six ounces of water*) to mercurial plaster, which induces too much elevation of temperature. (11.) Hebra recommends cold-water compresses only.

All of these applications have been, for the most part, applied to the face, the hands, and the arms only.

The severity and the mortality of small-pox have led many to think of means by which the disease might be completely extirpated. This leads to the consideration of—

II. The Prophylactic, Sanitary, or Preventive Treatment of Small-Pox.

—More than half a century ago it was generally taught, among English physicians, that small-pox attacked the same individual only once in the course of life, and that its double occurrence in the same person was either very rare or next to impossible. The observations of Drs. Willan, John Thomson, Mr. Cross, Dr. Barnes, Dr. Craigie; and others since the time of these eminent physicians, lead to the following general conclusions:—(1.) Small-pox, though in general attacking the same individual only once during the course of life, may, however, affect him a second, and even a third time. (2.) This happens much more commonly when the first attack has been one of mild distinct small-pox, than when it has been severe; and if the first attack has been one of confluent small-pox, it is rare for the same individual to have a second attack. (3.) It is established by numerous observations, that an attack of any one of the varieties which have been named spurious small-pox, or abortive small-pox, or chicken-pox, by no means secures the same individual from an attack of confluent small-pox at a subsequent period. (4.) Small-pox produced by inoculation does not necessarily secure the individual against an attack of small-pox induced in the natural way. (5.) Every previous attack, however, of small-pox, whether natural or inoculated, exercises some modification on that which succeeds. This modification may be various in degree, from very slight and almost imperceptible to very conspicuous and remarkable. In this modification the symptoms of eruptive fever may be mild and of short duration; and the eruption may consist of vesicles or hard pustules, which disappear without suppuration. (6.) The most powerful modifying agent on the course of small-pox is the action of the cow-pox on the constitution, or the disease produced by the appli-

cation of vaccine lymph to the exposed skin. The specific disease so induced, in a large portion of cases, not only renders the individual less likely to be affected by the variolous effluvia, but, if he is affected, changes very much the characters of the disease which may supervene. Though the fever which precedes the eruption in cases of this class be similar in form and equal in degree to that by which the inoculated small-pox is attended, the eruption is either papuliform or tuberculated, without much surrounding inflammation. A similar eruption is produced when vaccine and variolous matter are inoculated at the same time in the same individual; or when a person who is exposed to the variolous contagion has been inoculated with vaccine lymph early enough to mitigate, but not wholly to supersede, the eruption of small-pox. In such circumstances the vaccine lymph and variolous matter restrain and counteract the operation of each other on the system and on the skin. To these eruptions of modified small-pox the general name of *varioloid* eruptions has been applied. (7.) Cow-pox destroys the susceptibility to inoculated small-pox almost entirely; but the susceptibility to the natural disease, or that by inhalation, it does not entirely extinguish. This susceptibility, however, it diminishes in a much greater degree, and much more effectually, than inoculated small-pox does. (8.) The susceptibility to second attacks of small-pox,* and attacks of small-pox after vaccination, is principally favoured by the existence of an epidemic constitution of the atmosphere, and by the circumstance of early life, or the age below ten years. If no epidemic influence exists, the occurrence of second attacks of the disease may not be observed for a long series of years. But if, on the other hand, the atmosphere should possess or acquire an epidemic or variolous constitution, then neither the circumstance of a previous attack of small-pox, nor vaccination, can insure many of those under ten years of age, and not a few between that and thirty, from attacks of small-pox.

The preventive management of small-pox consists,—(1.) In the artificial production of the disease by *inoculation*, or *artificial variolation*; (2.) In the modifying and protective influence of *vaccination*.

Inoculation consists in the application of small-pox matter or *virus* to the surface of the corium, exposed by a puncture or scratch. The result is a local inflammation similar to small-pox, attended with an eruption and a fever, generally milder in form than small-pox acquired by breathing an atmosphere contaminated with the specific poison of the disease. The poison thus passes through the mucous membrane to infect the blood; and hence this is called the “natural way” of contracting small-pox. For obvious reasons, the operation of *inoculating* the poison of small-pox has been rendered illegal in this country, and the practice of vaccination has been attempted to be enforced by law. What remains to be said about vaccination will be considered under the next topic, namely:—

* The average number of second attacks of small-pox seems to be one per cent. (R. Acad. of Med., Marseilles, 1828; and *B. and F. Med.-Chir. Review*, Jan., 1848, p. 74).

COW-POX.

LATIN EQ., *Vaccinia*; FRENCH EQ., *Vaccine*; GERMAN EQ., *Kuhpocken*;
ITALIAN EQ., *Vaccina*.

Definition.—*Cow-pox is the product of a specific and palpable morbid poison, which causes an acute specific disease in cows as a malady natural to them, attended with the formation of vesicular eruption or pocks near the udder or in the teats, which runs a definite course. The poison is reproduced in the contents of the pocks, and is thus multiplied during the course of the malady in the cow as it is in the human being. After a definite period of incubation (from the time that the specific virus is artificially implanted in a child), a specific nodule or papule forms, which, at the point of inoculation, passes through the stages of vesicle, pustule, scab, and desiccation. On the fifth or sixth day it changes to a vesicle, which, by the eighth day, has attained the size of a lentil, having a central depression—an umbilication with dissepiments. On the ninth day the contents become cloudy and whey-like, and a broad dark-red halo becomes developed around the vesicle. On the tenth day the contents are purulent; after which the pock begins to dry up, and the scab falls off towards the end of the third week or later, leaving a white cicatrix, with characteristic round pits over its surface. During the maturation of this specific lesion the adjoining lymphatic glands swell; a febrile state is induced, denoted by increase of temperature (104° Fahr.), constitutional disturbance, acceleration of the pulse (which, to a certain extent, has been observed to continue persistent in some cases); and a general lichenous, roseolar, or vesicular eruption may make its appearance on the trunk and limbs. The disease runs a definite course, affords immunity from another attack (for a considerable time at least), and exercises (during that period) a protective influence from human variola.*

Pathology.—The importance of a comprehensive knowledge of the pathology of variolous diseases generally, and of *cow-pox* in particular, lies in the relation of *cow-pox* to *small-pox* and to *vaccination*. Dr. Jenner named the disease "*variola vaccinae*," implying thereby that one genus at least of the animal creation is liable to a disease of a kindred nature with that which attacks man. The disease in the cow was observed to be generally mild; in man it was observed to be most pestilential and sometimes fatal. It was observed, also, that *cow-pox* was communicable from the cow to man, and that persons so affected were protected from subsequent attacks alike of *small-pox* and of *cow-pox*. Dr. Jenner believed that the two diseases were in reality identical. It has now been shown, by unquestionable evidence, that cattle and horses have for centuries been known to be affected with a species of *small-pox* or *variola*. Every different writer who has seen the disease has given it a similar name. Previous to 1745 it was known and described in Italy (FRACASTORIUS, LANCISI, RAMAZINI) as a malignant disease which destroyed cattle almost as extensively as *small-pox* did the human race. It was first observed in this country in 1745, and again in 1770, when it appeared among the horned cattle with so much severity that His Majesty George III., in his speech from the throne, on the 9th of January of that year, called upon the Houses of Parliament to take the subject

into their serious consideration. The disease continued with more or less violence till 1780; and it was no doubt the expiring embers of this epizootic which Dr. Jenner found in Gloucester, and made the basis of his investigations during that and subsequent years. (See also TROUSSEAU'S *Medicine*, Vol. II.) Dr. Layard described the disease amongst the cattle in England in that year, in a paper communicated to the Royal Society; and he mentions that inoculation from cow to cow was successfully practised to mitigate the severity of the disease; just as Mr. Simonds, of the Veterinary College, London, in 1862, successfully practised inoculation of the variolous disease from sheep to sheep, or lamb to lamb, throughout the counties of Wilts, Hants, and Dorset, when *ovine small-pox* was epizootic. The great increase of mortality from small-pox among human beings which occurred during the latter part of the last century is a fact of some importance in connection with the epizootic disease; for at other times and places it has been observed that when the cattle were scourged by the variolous disease, mankind were in like manner great sufferers from a similar epidemic. In the interesting lectures "Introductory to the Study of Fever," by the late Dr. Andrew Anderson, of Glasgow, we are told that while small-pox was raging with great violence at St. Jago, on the west coast of New Granada—near to which a town named David, in Chiriqui, was situated about sixty or seventy miles to leeward—a few days (four or five) before the disease appeared in this latter town the small-pox had attacked and destroyed many monkeys in the forest. Dying and dead monkeys were seen on the ground covered with the perfect pustules of small-pox; and several sick monkeys were seen on the trees, moping or moving about in a sickly manner. In the course of a fortnight one-half of the inhabitants of the town of David were stricken with small-pox (ANDERSON, p. 70). It is also within the experience of many medical men, that during the prevalence of small-pox cattle are apt to become affected with cow-pox. Horses, as well as cows and sheep, are liable to the affection; and the countries where the disease of late years has been found are those where it has formerly been known to have existed among cows or horses in its most virulent form. During the epidemics of small-pox previous to 1840, the variolous affections among the cows of the country were more observed than at any period for many years. In the dairies of Suffolk, of Gloucestershire, Dorsetshire, and Buckinghamshire, the disease has prevailed not only during epidemic small-pox, but when no cases of variola were known to exist in the immediate neighbourhood. There are good grounds for the belief that the impalpable emanations—the specific effluvia—from cases of small-pox in human beings, have been sufficient to communicate the variolous disease to cows. Mr. Ceely gives a most interesting history of such an occurrence in the tenth volume of the *Transactions of the Provincial Medical and Surgical Association*. At the village of Oakley, about sixteen miles from the town of Aylesbury, small-pox had been epidemic from June to October, 1840. Two cottages, in which three persons resided during their illness, were situated, one on each of two sides of a long narrow meadow, comprising scarcely two acres of pasture land. One of these three patients, though thickly covered with pustules of small-pox, was not confined to her bed after the full development of the eruption; but frequently crossed the meadow to visit the

other patients—a woman and a child—the former of whom was in great danger, from the confluent malignant form of the disease, and died. She was buried the same evening, according to custom; but the intercourse between the cottages across the meadow was still continued. On the day following death the wearing apparel of the deceased, and the bed-clothes and bedding of both patients, were exposed for purification on the hedges bounding the meadow; the chaff of the child's bed was thrown into the ditch; and the flock of the deceased woman's bed was strewed about on the grass over the meadow, where it was exposed and turned every night, and for several hours during the day. This purification of the clothes continued for eleven days. At that time eight milch cows and two young heifers (stirks) were turned into this meadow to graze. They entered it every morning for this purpose, and were driven from it every afternoon. Whenever the cows quitted the meadow the infected articles were again exposed on the hedges, and the flock of the bed was spread out on the grass, and repeatedly turned. These things remained till the morning, when the cows were readmitted and the contaminated articles were supposed to be withdrawn. It appears, however, that the removal of the infected articles was not always accomplished so punctually as had been enjoined, so that, on one occasion at least, the cows were seen in the midst of them, licking the flock of the bed which lay on the grass. These cows were in perfect health when first put out to graze; but in twelve or fourteen days *five* (out of the eight) milch cows appeared to have heat and tenderness of the teats. The teats became swollen, and small hard papules could be distinctly felt upon them, as if imbedded in the skin. These papules daily increased in magnitude and tenderness; and in a week or ten days they rose into *blisters* (vesicles), passing into brown or blackish scabs. When the teats were in this condition, and very tender, constitutional symptoms of ill-health became developed. Sudden *sinking* or loss of milk, drivelling of saliva from the mouth, frequent inflation and retraction of the cheeks, staring of the coat, "tucking up of the limbs," "sticking up of the back," and rapid loss of flesh, were the appearances which even the peasants themselves were able to appreciate. By the middle of the third week the pustules were mature, and the crusts and loose cuticle began to be detached. The simultaneous occurrence of the disease on all the animals increases the probability of the operation of one common cause. The whole of the cows were certainly affected within less than three days of each other; and another circumstance requires particular notice, namely, the occurrence of the disease in a young heifer (stirk), to which of course the disease could not have been communicated by those casualties which commonly propagate the vaccine variola amongst milch cows. The cause which originated the disease amongst them at the same time affected the young heifer, which hitherto had not been considered liable to the vaccine disease, simply because no one had seen the animal affected by it. Now it is known, both in this country and in Germany, to be liable to cow-pox. The proprietor of the animals referred to in this narrative had the disease communicated to himself. He had never suffered from small-pox nor the vaccine disease; and it was his own spontaneous conviction "*that his cows had been infected from human small-pox effluvia*," to which undoubtedly they had been exposed. He had not the

remotest idea of the medical theories concerning the nature of the disease, and consequently had no prepossession in favour of the opinion he thus spontaneously expressed. His cattle had hitherto been in good health, and no *vaccine variola* had been known in the vicinity.

Human small-pox has also been communicated to the cow by direct implantation of the specific virus from man. The efforts at first were numerous and unsatisfactory to inoculate directly the cow with human small-pox; and the experiment is said to have first succeeded at the Veterinary College in Berlin, so early as 1801. M. Viborg, of Copenhagen, about the same period communicated the disease to dogs, apes, and swine. In 1807, Gassner imparted small-pox to the cow by inoculation. In 1830 or 1831, Dr. Sonderland, of Bemen, communicated the disease to cows, by simply covering the animals with sheets and blankets on which persons suffering from small-pox had lain. In 1836, Dr. Basil Thiele, of Kasan, in South Russia, successfully inoculated some cows on the udder with the *virus* of human small-pox. Vesicles were produced bearing all the characters of the true *vaccine vesicle* in those animals. The lymph so produced from the *variola* of the cow continued to retain the specific properties of the *vaccine variola* throughout seventy-five successive transmissions in the human subject. In 1838, M. Thiele repeated this interesting experiment with a similar success. It would therefore seem that *the constitution of the cow has the power of assimilating, of modifying, and of mitigating the human variolous virus, and of stamping it with the properties of the vaccine variola*. Dr. Ceely, of Aylesbury, twice succeeded in accomplishing this object (so important pathologically), after many fruitless trials. The interesting papers by him in the eighth and tenth volumes, and the Reports of the Vaccination Section of the Provincial (now British) Medical Association in their *Proceedings* for 1839 and 1842, should be studied by every student of Medicine. Very recently (1860) Martin inoculated some variolous matter, taken from a pock upon the body of a man who died of variola, into a cow's udder, and subsequently vaccinated about fifty persons with the matter derived from the cow. Most of those so inoculated were attacked with variola, and three died (*Boston Med. Journal*, 1860; *New Syden. Society Year-Book* for 1860). Mr. Ceely has also often recommunicated the vaccine disease from man back to the cow (*retro-vaccination*, as it has been called); and he has observed that good human lymph, when re-transmitted in this manner, loses some portion of its activity. The phenomena appear later, smaller vesicles are produced, but ultimately, *after successive re-inoculations on man*, it regains its activity. Human small-pox has also been transmitted through the horse to the cow, and so to the child in the form of cow-pox (FLETCHER).

As the first origin of these specific poisons is unknown, it cannot be now definitively determined whether man first had *variola* communicated to him from the animal creation, or whether the lower animals, such as horses or oxen, had the disease communicated to them from man. The existence of small-pox in man is recorded in China as early as 1122 years before Christ (MOORE). And it is certain that when variolous disease appears among the lower animals in a malignant form, it is capable of producing, by inoculation, a disease of similar severity in man, if he has not already suffered from a similar affection; and that the direct inoculation

of the cow with human small-pox produces a mild and mitigated form of disease; that such disease being again reproduced in man by inoculation from the mitigated disease of the cow, accords entirely in its character, in its progress, and in its protecting influence with the *variola vaccinae*, as described by Dr. Jenner. These and similar facts seem to lead to the conclusion that small-pox and cow-pox are not dissimilar diseases, but are identical in their nature.

There are some remarkable circumstances which must at once arrest the attention of the student who carefully studies the accounts given of the experiments on men and animals, from which many of these statements are deduced. *First*, there seems to have been great uncertainty and difficulty attending the actual attempts to transfer the specific virus of these eruptive or variolous diseases from one animal to another. The very interesting experiments of Ceely, Thiele, and others, demonstrate this in a remarkable manner. *Second*, These experiments show the *marked increase or improvement which sometimes takes place in the energy, and therefore in the quality of the specific virus, by subsequent removes or inoculations, in animals of the same kind, after the virus had been successfully implanted in one of them* (see p. 375, *ante*). This increased energy, and therefore improved quality, was shown in the more perfect development of vesicles, and in the more active manifestations of the primary and secondary symptoms. The subsequent inoculations of such improved lymph seem to produce less severe and less dangerous *local* results—the virus seems less acrid, less virulent, and less mischievous—having apparently acquired increased specific activity combined with mildness of action, and a greater susceptibility of transmission from one animal to another of the same kind.

Keeping, therefore, these facts in view, the history of the remarkable epizootic of *variola ovinae* which made its appearance in August, 1862, in some of the largest breeding flocks of sheep in the West of England, is of great interest to the pathologist. This variolous disease of the sheep allies itself very closely with small-pox in man, with cow-pox amongst cattle, and with the vesicular eruptive diseases of the horse; but undoubtedly modified by the constitution of the sheep, just as the variola of man is known to be modified or transformed by the constitution of the cow into the *variola vaccinae* of that animal. The ovine variola is known as the *clavelée* of the French (*tag sore* or *rot*); and although this kind of *rot* was not observed in this country till 1847, when it was imported from Spain, yet it is a disease by no means uncommon as an epizootic in the flocks of Italy, France, and Moravia. In 1803 the mortality was considerable in Moravia; but by a timely inoculation with the *virus* of the disease, the remaining parts of affected herds were preserved. The artificially affected animals seemed to pass through a milder disease. To this kind of inoculation the name of "*clavelisation*" has been given. This variolous disease in sheep assumes one of two forms, namely,—(1.) A virulent or malignant form; and (2.) A benign form. The virulent form (which would seem to have been the form epizootic in England) never produces pustules; and specific virus for safe inoculation (*clavelisation*) can only be got from the benign form of the disease. When the disease is virulent, the sheep lose their eyes, their wool falls off, and their skin

cracks in a zig-zag manner. Their nostrils are so full of a fetid discharge that the shepherds are under the necessity of constantly syringing them with medicated lotions, to prevent suffocation. When the disease is benign, genuine pustules form, and every pustule, after the scab falls off, leaves a cicatrix in the form of a pit. On this cicatrix the wool never grows again. Hence it can always be told what sheep have undergone the variolous disease, as easily as it can be seen that a human being has had small-pox, from being marked with the pits or cicatrices. The prevention or mitigation of this disease among sheep is a most important object in a sanitary point of view. In 1803 Dr. De Carro, of Vienna, tried the effects of the inoculation of *variola vaccinae*, but without success. The inoculation only produced small local sores. It is said, also, that this sheep-pox cannot be communicated directly to the cow, nor to children (CEELY, SIMONDS). Other observers state, however, that it is so communicable, and that *ovination* is protective against small-pox (SACCHO). The *ovination* or inoculation of the disease from sheep to sheep, was first proposed by Chalette in 1762, and has been yearly practised since that time in many parts of Italy, Prussia, Austria, and France. The practice of inoculation from sheep to sheep was practised in England by Professor Simonds during the recent epidemic. Results accrue to sheep, from the communication of the disease to them by inoculation, not less beneficial (compared with the fatal effects which followed when they became affected with the disease in the "*natural way*") than the beneficial effects that accrued to man when small-pox was communicated to him by inoculation—as it *rightly* was—before the protective powers of vaccination were known. The ovine variola has been ascribed by some farmers to the communication of the virus from an eruptive disease of the horse (DE CARRO, RING). Fontan relates that some mares being affected with a pustular eruption, the matter from the pustules was inoculated on the teat of a cow, where it produced several fine pustules. From these several infants were vaccinated, with the result of producing perfectly characteristic vaccine vesicles. Thirty infants were vaccinated from this source at Toulouse, and in all the result was most satisfactory (*L'Union Méd.*, 1860; *New Syden. Society Year-Book*, 1860). If this can be definitively established, then the successful inoculation of some animal, *other than the sheep*, with the virus from the specific eruptive disease of the horse, may give such energy and, at the same time, mildness to the morbid poison, by subsequent removes, that the implantation of the new virus (*equination*) may perhaps be followed by the same beneficial results to sheep, in respect of the malignant variolous disease to which they are liable, that *vaccination* has conferred on man in respect of small-pox. The question, then, at once suggests itself: "Has human small-pox ever been communicated to sheep, with the view of obtaining a modified lymph, which may confer protection on them from the variolous disease to which they are liable?" From analogy, may we not indulge the hope that the practice of inoculating sheep from the small-pox of man might induce as mild and modified a disease in them, and prove as protective to them as vaccine variola, through vaccination, has been to man? Or, having communicated the human variola to cows (since the experiments of Ceely and Thiele demonstrate that such inoculation may be effected), might not sheep be tried with the

resulting virus as a protective agent? The vesicular eruptive diseases of dogs, as well as of horses, should be similarly inquired into and experimented with, seeing that dogs are so much associated with sheep. The outbreaks of the variolous diseases amongst cattle and sheep seem to follow similar inexplicable paths to those which small-pox amongst human beings is observed to follow. Occasionally the disease is *epizootic* (equivalent to *epidemic* amongst men), or prevalent at the same time in several farms at no great distance. Cases spring up like small-pox, now and then, which appear to be solitary, and the source of which cannot be traced. It is rare indeed that every solitary case of small-pox in man can be traced to a communicating source. In oxen it may be seen sometimes at contiguous farms; at other times, one or two farms, apparently similarly circumstanced amidst the prevailing disease, entirely escape its visitation. Sometimes it is introduced into a dairy by recently purchased cows. On the other hand, it has been undoubtedly communicated to cows from the vesicular disease of the horse, through the hands of the common attendant on both animals. There can be no doubt, also, that the disease exists, although it is not observed; for being mild, and the temper of the animal good, little notice is taken of tenderness in milking, and so its existence escapes detection.

There are *spurious* forms of cow-pox, which it is necessary to be able to distinguish. In the true cow-pox in the cow there is very slight manifestation of fever or constitutional disturbance. The secretion of milk may diminish; but the animal continues to feed and to graze very much as usual. The local affection may be so mild that a single vesicle only may appear upon the udder; but where the udder is voluminous, flabby, pendulous, and uncovered with hair, with a corrugated, thin, or fissured skin, then there is apt to be a copious eruption. The disease is very readily propagated from cow to cow by the milkers. It is also said to be communicated in the natural way; but the evidence on this point is not sufficient, at least there is no evidence to show that a healthy human being, or a cow, ever becomes infected with cow-pox by entering the atmosphere where a person or beast is suffering from vaccinia. The local symptoms of cow-pox in the cow are evinced by heat and tenderness of the teats and udder for three or four days, followed by irregularity and nodular hardness of the surface, especially about the basis of the teats and the adjoining part of the udder. The nodules or papules assume a red hue when about the size of a vetch or pea, and are quite hard. In three or four days more they increase to the size of a horse bean; milking becomes painful to the animal, and the pimples become vesicles (pocks), which are then apt to be broken by the hands of the milkers, giving rise to troublesome and dangerous sores on the udder and teats of the cow; and communicating the disease to the milker, if he is not already protected by having had the disease before. If the vesicle remains unbroken, it becomes a globular, oval, and ultimately a pointed (acuminated) pustule. A central depression, with a marginal induration, is the form ultimately assumed; and when punctured towards the centre, the pock yields a more or less viscid amber-coloured fluid. The period of incubation, after casual communication of the disease, seems to be from six to nine days, although it is said papules may be felt under the

cuticle about the fifth day. When the vesicles are fully mature, they may measure from eight to ten lines in the largest diameter; the centre and edges of the intumescent margin being of a deep blue or slate-colour, and the surrounding areola of a pale rose-colour, and seldom more than four or five lines in depth, the integuments under it being deeply indurated. The lymph contained in the vesicle is now so copious that the cuticle over the central depression appears raised up by it, and so gives rise to a globular or cone-like vesicle; or it spontaneously ruptures, when the lymph freely flows out, and concretes into a clear amber-coloured crust or scab. Dark-brown or black, solid, uniform crusts eventually form on the site of the pocks. Some of these crusts may be seen semi-detached, others entirely so, and exposing a raw surface with a slight central slough. The forms of the crusts are either circular or oval, some flatter, and others unguiform, some thin, and more or less translucent. These varied appearances are seen in all stages at the same time, indicating the formation of new crops of vesicles at different periods. If undisturbed, the crust or scab gradually becomes thicker, darker, and more compact, till the thirteenth or fourteenth day, and spontaneously separates about the twentieth or twenty-third day. A cicatrix or pit is thus left, which is shallow, smooth, oval, or circular, of a pale rose or whitish colour, with some traces of induration surrounding it. The anatomical structure of the vesicle seems to be precisely similar to that of small-pox in man, as shown now by many observers (GENDRIN, CEELY, and others).

Primary Vaccine Lymph.—To procure *primary* liquid vaccine lymph direct from the cow, in a condition fit for use, is a task of no ordinary difficulty. *Primary* crusts should be sought for on the lower part of the udder, and around the base of the teats; and during a search for these it is not improbable that smaller vesicles of later growth may be found to yield efficient lymph. The best lymph is to be obtained from perfect vesicles before they begin to point. After this period it is less to be depended on, particularly if very abundant, thin, or discoloured. Pointed vesicles, when broken by violence, are rarely to be relied on. Entire unpointed vesicles, or vesicles with central crusts, should be sought for on parts where they are least exposed to injury—namely, on the lower and naked parts of the udder and adjoining bases of the teats. It is impossible to exercise too much delicacy in the proceeding. The puncture to liberate the lymph should be made with a sharp lancet, as *near the centre* of the vesicle as possible; and the epidermis may be gently raised to a moderate extent around the discoloured or most depressed part. Slight pressure with the blade of the lancet, or between the thumb and finger, will enable the operator to charge a few points or capillary tubes with the slowly exuding lymph. Punctures at the elevated and indurated margin of the vesicle are utterly useless. They only give vent to blood. Vesicles on which the central crust has begun to form are the most productive, particularly if the crust be small, and the margin of the vesicle be tender, hot, and tumid. Small superficial vesicles are often more yielding than contiguous larger vesicles, which are more deeply seated or confluent.

Useful substitutes for liquid lymph, capable of communicating the vaccine disease, are—(1.) Amorphous masses of concrete lymph, found

upon or in close proximity to broken vesicles. They ought to be colourless, like crystals of white sugar-candy; or of a light amber hue, resembling fragments of barley-sugar. (2.) Central crusts, irregular, rough, and more or less conical—the more transparent and nearer a dark-brown hue the better. (3.) Vesicular crusts or desiccated vesicles. These crusts should be carefully removed by the milkers before they are casually removed or spontaneously fall; and those only of primary formation, which are, as it were, the mould of a vesicle, of a dark-brown translucent appearance, should be retained. These three dry conditions of the specific vaccine virus may be reduced to a liquid state at any time for use. Glycerine is the best solvent for such solid conditions of the lymph, which ought to be reduced to powder before the glycerine is added (COLLINS, *Boston Med. and Surg. Journal*, 1858).

Vaccination.—It is now (1879) at least seventy-nine years since Jenner first promulgated his discovery to the world, that the eruptive vesicular disease which has now been described as occurring on the udders and teats of the cow, and which he named the *cow-pox*, was communicable directly to the human being, and conferred protection from the small-pox, so fatal to man. The operation for thus engrafting the cow-pox on the human being has been named “VACCINATION;” and its discovery still remains one of the most interesting facts in the history of Medical Science. Dr. Jenner first caught a glimpse of these great truths when an obscure apprentice to a surgeon at Sodbury, near Bristol. Amongst the gossip of the cow-herds he had heard of the vague, obscure, but popular belief regarding the possible communication of cow-pox to the milkers of the cows, and the protection from small-pox which the cow-pox conferred—a belief which undoubtedly prevailed in the rural districts of Gloucestershire, and, at the same time, curiously enough, on the continent of Europe, in some districts round Göttingen. These things Jenner mentioned to the famous John Hunter, when he became an apprentice to that eminent surgeon; but John Hunter, otherwise sagacious and far-sighted, pooch-pooched Jenner’s notions as vague and improbable. Nevertheless, Jenner had determined to examine into the truth of the tradition. He thoroughly investigated the facts amongst the expiring embers of that epizootic disease which laid waste the herds of this country towards the end of the last century. He did not suffer the spark to be lost in the flame it had served to kindle; but commenced his earnest and painstaking investigations as soon as he had established himself as a surgeon at Berkeley, in Gloucestershire. In June, 1798, he published his observations in the form of a thin quarto, of scarcely more than seventy pages, dedicating it to his friend, the celebrated Dr. Parry of Bath. Jenner seems to have felt almost a holy reliance in the truth of his great discovery; and in the face of much foolish opposition he modestly continued to prosecute his inquiry, “encouraged,” as he said, “by the hope of its becoming beneficial to mankind.” Its importance to the welfare of the human race has since been clearly demonstrated; and the acute observation of Jenner himself has been abundantly fulfilled—namely, that the keenest of all arguments for or against the practice of vaccination will be *those which are engraved with the point of the lancet*. We have, indeed, in this country, paid but tardy homage to his memory; nevertheless, he has made for himself imperishable “footprints on the sands of time,”

which wave after wave of scientific research appears only to deepen and render more distinct. He has not only pointed out the means of subduing a loathsome disease, but the health of all civilised communities has improved since vaccination was introduced; and, in proportion as vaccination has been efficiently carried on, the frequency of small-pox epidemics has been diminished, and the duration of human life has been extended.

The subject of vaccination is one which demands a careful study, alike in its pathological and in its sanitary relations, involving, as it does, questions of great national importance. In 1841 the Vaccination Act was passed in the Houses of Parliament, which rightly made the practice of *inoculation unlawful*. In 1853 another Act was passed, with the view of rendering the practice of *vaccination compulsory*—an Act which is known as Lord Lyttleton's Vaccination Act. During the interval between the first and second reading of the Bill in the House of Commons, "The Small-Pox and Vaccination Committee of the Epidemiological Society completed a report on the prevalence and mortality of small-pox, and of the means taken to guard against it through vaccination." The conclusions they arrived at were deduced from the largest and most accurate mass of statistical evidence which had ever been brought to bear upon the question, and were eminently calculated to encourage Her Majesty's Ministers to pass an efficient measure to *compel* vaccination. A most valuable pamphlet was afterwards published by Dr. Seaton, which demonstrates, in a still more forcible manner, the protecting and modifying influence of vaccination as regards small-pox. To this belief, indeed, the general assent of the medical profession appears to have been given more than half a century ago. *Then*, it would seem to have been all but unanimous; and *now*, one would think, at first sight, that it were almost an insult to human understanding to be obliged to collect statistics to *prove* that vaccination confers a large exemption from attacks of small-pox, and almost absolute security against death from that disease. But so it is; and independently of the information which such statistical inquiry is calculated to convey to those who advise our Lawgivers and Public Administrators, the inquiry is eminently useful in relation to everything which bears on the nature of vaccine and variolous disease. The general ignorance of the community, especially of the lower orders, and of not a few educated persons (even medical men) who ought to know better as to the value, the aim, and the object of vaccination, is lamentably great. Much unfounded prejudice has also still to be overcome. Moreover, the highest medical authorities of late years recommend that all views and facts put forward as objections to vaccination should be rigorously inquired into, and that there should be published from time to time a true account of such inquiries, with an elucidation of what has seemed doubtful and contradictory (SIGMUND, ALISON). It is now well known that Lord Lyttleton's Vaccination Act (1853) has proved but a very imperfect measure—a piece of legislation which has fallen very far short of accomplishing all that is yet required. The inefficiency and imperfect working of the Act have been fully shown,—(1.) In the Reports of the Registrar-General for 1854; (2.) By the medical profession generally; (3.) By the medical registrars in particular; (4.) By the public, as expressed now and again in the newspapers of the day. To this state of things we owe a most

valuable work on vaccination, written by the indefatigable (late) medical officer (JOHN SIMON, F.R.S.) of the then (1857) General Board of Health. The aim of this publication was to lay before the Board such medical facts and considerations as might assist in estimating the hygienic value of vaccination, and the strength of any objections which may have been alleged against its general adoption. This work is especially valuable, because it brings together a body of evidence down to the day of its publication—evidence of a pathological and statistical kind—records which have been engraved by the lancet's point—such evidence as Jenner would have rejoiced to see. But evidence of the inefficiency of the Vaccination Act of 1853 still continues to be apparent, as may be seen from the yearly reports of the medical officer of the Privy Council. During three or four years previous to 1860, Mr. Simon writes, that "sometimes in one set of places, and sometimes in another, there have been occurring, almost generally throughout England, epidemics of small-pox more or less considerable." To such an extent has this been the case, that, in 1860, the Lords of Her Majesty's Most Honourable Privy Council deemed it necessary, with reference to local outbreaks of this disease, to enter into correspondence with the authorities responsible for public vaccination in several Unions of England where small-pox had been prevalent. Some of these districts were specially visited by competent medical officers, and in two adjoining Devonshire Unions it was ascertained that the *diffusion of small-pox* had actually been to some extent *wilfully promoted by the illegal practice of INOCULATION!* Alarm was of course justly excited among educated persons in the endangered places by the knowledge that this offence was being committed; and in one case, where there was reason to believe that *inoculation had been the cause of death*, Secretary Sir George C. Lewis offered a reward of £50, to be paid to any person not actually concerned in the offence, who would give information and evidence leading to a conviction of the offender. The unsatisfactory working of the Vaccination Act of 1853, led to the promulgation of an Order of the Privy Council (of date December 1, 1859), for the improvement of public vaccination. Their Lordships have seen fit to direct the commencement of a *systematic inspection*, with reference especially to the operation of vaccination, and its efficiency in Unions where the amount of infantine vaccinations, compared with the number of births, appeared to be especially low. These inquiries continue to show that the present law, "to extend and make compulsory the practice of vaccination," is so imperfect as to be almost inoperative; and the *systematic inspections* instituted seem to have been so useful in promoting vaccinations, that their Lordships propose to continue them throughout all the Unions of England. The present position of our knowledge regarding vaccination is based upon evidence which demonstrates,—(1.) The *protective influence of vaccination*; and, (2.) The causes which have combined to *impair its protective power*. Of these in their order,—

I. *Of the Protection conferred by Vaccination; its Nature, and the Evidence of its Existence.*

The main features of the reports and works already mentioned amply illustrate how small-pox diminishes in its mortality *in proportion as*

EFFICIENT measures are adopted to insure PERFECT vaccination. To demonstrate this statement, the progress of vaccination in Great Britain and in Germany has been compared as to its influence on mortality generally; and more particularly, it has been shown, by comparing the statistics of vaccination from various German states with similar statistics from different districts in Great Britain and Ireland, that where vaccination is most perfectly carried out, small-pox is least mortal. The following are the general results which the committee of the Epidemiological Society arrived at:—(1.) To prove the influence of vaccination in England it is shown that out of every 1,000 deaths in the half-century from 1750 to 1800 there were 96 deaths from small-pox; and out of every 1,000 deaths in the half-century from 1800 to 1850 there were only 35 deaths from small-pox. (2.) To prove the influence of vaccination on the Continent, it is shown that in various German states sufficient evidence can be obtained to show that out of every 1,000 deaths *before vaccination was used*, 66·5 were deaths from small-pox; but that out of every 1,000 deaths *after vaccination* came into use, the deaths from small-pox were only 7·26. (3.) To prove that in countries where vaccination is most perfectly carried out small-pox is least mortal, it is shown that—

(a.) In this country, where vaccination has been *voluntary, and frequently neglected*, the deaths from all causes being 1,000, the deaths from small-pox were as follow:—London, 16; Birmingham, 16·6; Leeds, 17·5; England and Wales, 21·9; Perth, 25; Paisley, 18; Edinburgh, 19·4; Glasgow,* 36; Galway,* 35; Limerick,* 41; Dublin, 25·6; Connaught,* 60; All Ireland, 49.

(b.) In other countries where vaccination has been *more or less compulsory*, the deaths from all causes being 1,000, the deaths from small-pox were as follow:—Westphalia, 6; Saxony, 8·33; Rhenish Provinces, 3·7; Pomerania, 5·25; Lower Austria, 6; Bohemia, 2; Lombardy, 2; Venice, 2·2; Sweden, 2·7; Bavaria, 4.

Evidence corroborative of these results has been adduced by Dr. Balfour from the records of the Army and Navy Medical Departments, where every soldier or sailor is protected by vaccination, if he has not previously suffered from *cow-pox* or *small-pox*. (1.) For twenty years, namely, from 1817 to 1836 inclusive, it is shown that in Dragoon Regiments and Guards, with an aggregate strength during that period of 44,611 men, and a total mortality of 627, only *THREE* deaths were from small-pox. (2.) Among the troops at Gibraltar, the aggregate strength being 44,611 men during that period, and a total mortality of 1,291, only *ONE* death from small-pox occurred. (3.) In the West Indies several epidemics of small-pox prevailed during the period, but there were *no deaths* either among the British or white troops, of whom the aggregate

* With regard to the high rate of small-pox mortality in the towns marked by the asterisks, it was clearly shown by Dr. Stark, in Edinburgh, and by Dr. J. C. Steele (the present Medical Superintendent of Guy's Hospital in London), that such mortality was due to the neglect of vaccination. Dr. Steele, then resident in Glasgow Infirmary, called attention to the great increase of small-pox in Glasgow, as mainly coming from the Highland and Irish population, among whom vaccination was rare. Dr. Stark showed that more than 80 per cent. of all the deaths from small-pox happened in children under *five* years of age. No country is now more efficiently vaccinated than Ireland.

strength was 86,661, and with a total mortality of 6,803. Among the black troops on the same station, with an aggregate strength of 40,934, and a mortality of 1,645, there was not *one* case of small-rox. (4.) At Bermuda, Nova Scotia, New Brunswick, Cape of Good Hope, and the Mauritius, not a death from small-pox occurred during the twenty years mentioned; and the white troops of Western Africa wholly escaped this disease, *while the black UNPROTECTED population were dying by hundreds.* (5.) In Malta, from 1818 to 1838 inclusive, the aggregate strength of the British troops being 40,826 during these twenty years, and the total mortality 665, only *TWO* deaths were from small-pox. This is the more remarkable, inasmuch as small-pox raged as an epidemic all over the island in 1830, and again in 1838, destroying 1,169 persons. In 1830 there died of small-pox 1,048 out of a total mortality of 3,407; and in 1838 there were 121 deaths from small-pox out of a total mortality of 2,583. The disease was first introduced by His Majesty's ship "Asia." The mortality among those "*not vaccinated*" was 1 in 4·7; among those "supposed to have been vaccinated" it was 1 in 23·4; and, lastly, among those attacked a second time by small-pox the mortality was 1 in 10·8. The native population of Malta, in 1830, was estimated at 100,839 persons, amongst whom it appears that 1 in every 12·1 persons was attacked with the disease, and 1 in every 85 persons died. Amongst the military, including wives and children, the proportion attacked was 1 in 188, and the mortality only 1 in 682. (6.) The most important observations are those made at the Royal Military Asylum, in which the prevalence of variola among a *vaccinated* and *unvaccinated* population, at the same ages and in exactly similar circumstances, has been traced out by Dr Balfour (See *Med.-Chir. Trans.* for 1852).

In India the Sanitary Commissioner of the Punjaub draws attention to the loss of life by small-pox which occurs annually in that district, as yet practically unprotected by vaccination. With a population of 18,000,000, the deaths from small-pox there are never less than 20,000 a year. In 1869 they numbered 53,195. Any person walking through the streets of a Punjaub city is struck by the immense proportion of persons blind of one or both eyes—a calamity caused in 99 cases out of 100 by small-pox. Europeans, protected by vaccination, suffer very little.

Recent experience still more strengthens these practical conclusions, especially from the epidemic variola of 1870-71, so ably reported on by Dr. Vacher (*Gazette Med.*, September 18, 1875). As the small-pox, which prevailed epidemically throughout Europe from 1869 to 1873, has almost entirely disappeared from the Continent, only showing itself by some isolated cases, Dr. Vacher justly considers it an opportune time to reckon up the results. For some time past the Académie de Médecine and the French medical societies have been urging the importance of revaccination, but vaccination itself has been too much kept out of view; for it seems incredible that, seventy-five years after Jenner's discovery, one-third of the population of France is still deprived of the benefit of vaccination, and that there are some departments, as Aveyron and Corsica, in which scarcely 20 per cent. of the inhabitants are thus protected. It is this circumstance which aggravates the mortality of variola, and renders epidemics so dangerous, even for those who have been vaccinated. It is

not only ignorance and negligence that are the causes of this state of things, but there are unreasoning opposition and prejudices against vaccination there, as in this country, maintained even by persons who belong to the educated classes. Persons of this description point to the late epidemic, which, according to their figures, has in France carried off 200,000 victims, and ask what confidence can henceforth be placed in the efficacy of vaccination, which has evidently become diminished in power, if not radically powerless. The object of Dr. Vacher's paper is to show the erroneousness of these ideas, and for this purpose a most important document, published by the Swedish Government, is cited. It states that the mortality from small-pox fell from 44,184 for the period 1792-1801, to 14,898 for the period 1802-1811, vaccination having been introduced in 1801. In the period 1852-61 it fell as low as 4,175; and, even during the general European recrudescence of the disease, only rose during the period 1862-71 to 8,999. The value of these numbers is, however, best brought out by comparing them with those of the population. In 1760 Sweden had 1,981,000 inhabitants, and in the period of 1762-71 there was one death from variola per annum in every 364 inhabitants, and the deaths caused by this disease constituted twelve-hundredths of the total of deaths. At the present time (1875), with a population of more than 4,000,000, there is but one death for 4,650 inhabitants, and small-pox only constitutes one-hundredth of the total of deaths. The Swedish Government contributes liberally to the expenses of vaccination, thus contrasting favourably in this respect with that of France, where the *conseils* of the departments are often most niggardly in their contributions, and the disease is found to be most prevalent in the localities where such parsimony exists. Recurring to the epidemic in France, this is known to have shown itself at some points early in 1870, and it became generalised as a consequence of the war of that year, the defeated troops spreading it in every direction. The concentration of troops, also, in various places, furnished new aliment to the epidemic. On their arrival at Paris in September, 1870, it took on most disastrous proportions, the deaths becoming suddenly increased in October from 600 to 1,500; and during the four months of investment they amounted to 6,495—being double of those which were caused by the arms of the investing army. The armistice at last put an end to an epidemic which, in fifteen months, had killed 13,093 persons. Although the troops spread the disease in every direction, it is to be observed that it only committed great devastation where the inhabitants had been badly vaccinated. Thus, at Albi, where vaccination and revaccination had been carefully executed in 1869 and 1870, there were only 114 deaths in a population of 25,059; while at Monerat, where vaccination had been grossly neglected, there were 180 deaths and 50 disfigurements among 2,384 inhabitants.

As to the pretended immunity of young children, and the prevalent popular prejudice against their being vaccinated too young, it is to be observed that of 10,331 deaths from small-pox at Paris, 213 took place during the first month; 151 between one and two months; 107 between two and three; 200 from three to six; and 397 from six to twelve,—being a total of 1,153 less than one year of age—a large proportion, when it is

remembered that the number of infants of that age at Paris does not exceed 20,000.

The French epidemic spread to adjoining countries in consequence of the events of the war. Thus, after Sedan it became general in Belgium, producing 21,315 deaths; and the same thing occurred in Switzerland, although no exact figures have been yet published. The disease also broke out in the Prussian fortresses very soon after the arrival of the first French prisoners, and its propagation was greatly favoured by the careless way in which prisoners suffering from the disease were exposed and transported. From early in 1871, in consequence of the distribution of detachments of French prisoners over the whole of Prussia, the disease prevailed in every province. In 1871 there occurred 59,838 deaths from this cause—1,216 of these occurring among the French prisoners. In Berlin there were carried off 5,084 persons, 1,322 being infants of less than a year old. The number of persons who had been revaccinated was 270,000, and of these 1,036 took small-pox, only 162 dying (Dr. GUTTSTADT). Italy, at first free from the disease, was invaded by the epidemic on the return of the Garibaldians, who, in the Côte-d'Or, had suffered as much from small-pox as from the arms of the enemy. The Italian Government has not as yet published its report on the epidemic of 1871-72; but a memoir by Professor Toscani, on the disease as it prevailed in Rome, is of interest. There were 3,149 cases, with 1,219 deaths. Here, as in Paris and Berlin, the infants were the chief sufferers—74 per cent. dying of those aged less than forty days, and 55 per cent. between forty days and one year. Of 521 persons who had been vaccinated, there were but 72 deaths, or 13½ per cent.; while of 2,289 non-vaccinated there died 1,065, or 46½ per cent. M. Toscani believes that many of the vaccinations had been most imperfectly performed. Among the 3,149 cases there were 23 persons who already had had the small-pox, and six of these had been vaccinated before their first attack. One very remarkable case was that of a woman, fifty-five years of age, who had been vaccinated and revaccinated in France before contracting her first small-pox, and then succumbed to the hæmorrhagic form of the disease, which she took at Rome. There were 57 examples of this hæmorrhagic form of the disease, with 50 deaths—i.e., 88 per cent. The disease was confluent in 1,749 cases, with 919 deaths, or 54 per cent.; and discrete or varioloidal in 433 cases, with 217 deaths, or 16 per cent. After adverting to the successful operation of the Scotch Vaccination Act, M. Vacher justly arrives at the final conclusion that the discovery of Jenner will never render the services it is capable of until vaccination be rendered obligatory in all countries (*Med. Times and Gazette*, Oct. 9, 1875).

One very obviously beneficial result of vaccination has not been so much appreciated and noticed as it ought to be—namely, that while the epidemic influence of small-pox greatly increased during the practice of inoculation, it has greatly diminished since vaccination has been adopted. Dr. Hebra, of Vienna, alludes to the fact, and incidentally remarks, "That epidemics of small-pox have been more rare, and are less malignant, since the introduction of vaccination." Definite data are to be gathered from the various reports already mentioned, which fully bear out the statement. The results may be stated as follow:—(1.) During ninety-one years previous

to *inoculation*, there are on record 65 distinct and well-marked epidemics, which is equal to a ratio of 71·4 epidemics in 100 years. (2.) During sixty-three years in which *inoculation* was practised, and that to a very great extent, there were 53 distinct and well-marked epidemics, which is equal to a ratio of 84 epidemics in 100 years. (3.) During the fifty-five years since *vaccination* has been mainly practised, there have been 12 distinct and well-marked epidemics of small-pox, which is equal to a ratio of 24 epidemics in 100 years. This kind of testimony is greatly enhanced by the fact, that epidemics never occur in the army or navy of our own country, nor in those countries where the soldiers and seamen are efficiently protected by vaccination. The details given with reference to the two Malta epidemics in 1830 and 1838 afford a striking proof of the protective power of vaccination when tested by epidemic influence; and there are records of the Danish army and navy having altogether escaped during several epidemics of small-pox in Denmark.

There is still another way in which the protective power of vaccination makes itself manifest—namely, by the *mildness of the disease in the vaccinated compared with the unvaccinated, and the almost absolute security against death from small-pox which PERFECT vaccination confers*. With few exceptions this appears to be the universal belief of the medical profession. At various times the opinions of large numbers of medical men have been specially asked for and obtained on this point. Three distinct and very comprehensive “polls” may be referred to, namely—(1.) That by the College of Physicians in London, eight years after vaccination had been adopted. (2.) An almost national “poll” taken by the Epidemiological Society of London about the years 1852 and 1853. The written opinions of nearly two thousand medical men in this kingdom, as well as Bombay, Bengal, the Mauritius, the West Indies, and various other places, were then expressed; and they concurred in confirming the belief in the protective and modifying influence of vaccination in small-pox. (3.) A very extensive “poll,” of which a list is published by Mr. Simon, comprehends not only members of the medical profession generally, but also the members of the Medical Department of the Army and Navy, together with the opinions of foreign governments. These “polls” may be held as completely decisive of the question, really practically decided in the affirmative sixty years ago. From such evidence the inference is so inevitable, “that he who disputes it is equally unreasonable as he who opposes in like manner any proposition in Euclid” (ALISON). The actual *extent of the security against death from small-pox* enjoyed by vaccinated, compared with unvaccinated persons, has been calculated by Mr. Simon from various sources:—amongst the vaccinated the death-rate varies from an inappreciably small mortality to $12\frac{1}{2}$ per cent.; amongst the unprotected it ranges from $14\frac{1}{2}$ to $53\frac{4}{5}$ per cent. The average percentage mortality from small-pox, stated by Mr. Marson to occur amongst the vaccinated, is 5·24; but when vaccination is known to have been perfectly performed, as shown by the *cicatrices*, the mortality is uniformly found to be reduced to less than half of one per cent.

Another important and interesting phase in which vaccination may be viewed as exercising a protecting influence *indirectly* over the health of the community, may be studied in those statistics which show that the

general death-rates from other diseases have diminished, especially as regards *scrofulous diseases*, since vaccination became more universal. To Dr. Greenhow and to Dr. Farr in this country, and to the statist of Sweden, we are indebted for any accurate knowledge existing on this subject. Not only has the grand total of the death-rates been diminished, but the death-rates of two special classes of diseases have diminished in a remarkable degree. These are,—(1.) *Scrofulous affections*, including *phthisis* or *scrofula*, with the deposit of tubercle in the lungs; and, (2.) *The continued fevers*, especially *typhoid fever*.

Another kind of historical evidence bearing out the protective influence of vaccination is got by comparing the advertisements of old newspapers, especially those during the last portion of the seventeenth and early portion of the eighteenth centuries, with similar advertisements of the present day. At the former period the ravages of small-pox upon the population were beyond conception, testifying to the widespread epidemic influence. The description of every man or woman, as exhibited in "the portraits of the Hue and Cry," showed them to have been more or less marked with small-pox, or "speckled with *pock-holes*." Now-a-days it is the exception, and not the rule, to see such pits and scars amongst the population (*Quarterly Review*, July, 1855).

It has thus been clearly demonstrated how vaccination has thrown the *egis* of protection over the world; and how ample, how great, and how efficient that protection may be. It has been shown to diminish mortality not only from small-pox in particular, but to diminish the mortality generally, both in civil and in military life, at home and abroad, and that just in proportion as it is *efficiently* performed. It has been shown to diminish the epidemic influence; it has been shown to preserve the good looks of the people; it has been shown that it tends to render small-pox a mild disease compared with the same disease in the unprotected; it confers an almost absolute security against death from small-pox; and, lastly, it has been shown to exercise a protecting influence over the health of the community generally. On the other hand, it is no less amply proven that "wheresoever vaccination falls into neglect, small-pox tends to become again the same frightful pestilence it was in the days before Jenner's discovery; that wheresoever vaccination is universally and properly performed, small-pox tends to be of as little effect as any extinct epidemic of the middle ages" (SIMON). Moreover, it has been clearly shown by the *systematic inspections* instituted by Her Majesty's Privy Council, under the direction of Mr. Simon, that it is hopeless to expect to be free from fatal epidemics of small-pox, of greater or less extent, so long as unvaccinated children are allowed to accumulate as they have been found to do. There is therefore the greatest necessity for vigilance on the part of every intelligent member of the community to prevent any re-accumulation of unvaccinated persons.

Several conditions are still absolutely necessary to be efficiently carried out before we can hope to see small-pox eradicated through vaccination, and the aim of Jenner accomplished. It is the executive part of Lord Lyttleton's Act of 1853 which requires special attention. Systematic inspections are necessary to determine whether the results of individual vaccinations are satisfactory; and the local Poor Law Authorities ought to be empowered

to appoint an adequate number of medical officers, (a.) to enforce the Act; (b.) to proceed against defaulters;* (c.) to adopt every known safeguard

* In November, 1876, that eminent statesman, Mr. W. E. Gladstone, in replying to certain anti-vaccinationists, expressed a doubt (not that vaccination itself is expedient, but) as to the expediency of the compulsory and penal clauses of the Vaccination Act, and that he regarded such provisions "with mistrust and misgiving; and were he engaged on an inquiry, he should require very clear proof of their necessity before giving them approval." There may be, perhaps, others of this frame of mind, who have not the slightest doubt of the folly of not vaccinating, but who hesitate because *there are things which would be wrong not to do, that they deem not right to compel others to do*. How far this belief and argument are just to the world at large the records of the previous pages abundantly show; and, if the noblest motive is the public good, it is because "*the unvaccinated*" become common causes of danger to the community that compulsory and penal clauses are necessary and justifiable to enforce enactments for the protection of the public and to secure the common weal. In further proof of this, it may be here noted that on February 13, 1871, Mr. W. S. Forster moved for a committee to inquire into the operation of the Vaccination Act of 1867, and to report whether such Act could be amended, which motion was agreed to. He considered it necessary to provide for vaccination by legislation. Statistics showed that the extent of small-pox epidemics had really gone up and down according as compulsory vaccination had or had not been enforced. In Scotland and in Ireland, where they had ensured almost complete compulsion, the disease was formerly very prevalent, and now it was reduced almost to a minimum. In those districts, too, in England, where vaccination had been enforced, small-pox had almost vanished. Nevertheless, some people in the country entertained conscientious objections to vaccination, on the ground that it would do their children harm; and the evidence which would be adduced before such a committee would, Mr. Forster believed, tend to convince such persons of their error.

The following paper of advice and instruction was issued by the Royal College of Physicians in 1871:—"At the present time, when small-pox prevails extensively as an epidemic, and when much anxiety exists in the public mind respecting it, and questions have arisen as to the efficacy of vaccination as a preventive of that formidable disease, the Royal College of Physicians have thought it their duty to call public attention to the following facts and observations:—Nothing in the history of vaccination, since its first introduction, has occurred to shake the confidence that has hitherto been placed by every well-informed physician in the power of vaccination to diminish the susceptibility to small-pox, and in its efficacy as a protective against both the mortality and the disfigurement occasioned by that disease. Small-pox occasionally occurs a second time in the same individual; it is not, therefore, surprising that small-pox does sometimes occur in those who have been vaccinated, more especially in those in whom the operation has been imperfectly performed. These facts were admitted by Dr. Jenner himself, the discoverer of vaccination. The mortality from small-pox occurring in the non-vaccinated amounts to 35 per cent. of those attacked, whereas the mortality in those who, having been properly vaccinated, subsequently take small-pox, amounts to less than 1 per cent. Disfigurement, more or less serious, is in the non-vaccinated the rule; in the properly vaccinated it is the exception. Experience has amply proved that revaccination of adults who have been vaccinated in childhood will, to a very large extent, protect against an attack of small-pox. Thus, to take one of many illustrations that might be adduced:—For more than thirty years all the nurses and servants at the Small-Pox Hospital, who had not previously had small-pox, have been revaccinated before entering on their duties; and not one case of small-pox occurred among these persons, although living in an atmosphere of concentrated infection. The College therefore deem it right—1. That all persons who have not been vaccinated, or who have not already had small-pox, should at once be properly vaccinated by competent vaccinators. 2. That all persons who have passed the age of puberty, and have not been revaccinated since infancy, should be revaccinated. 3. That all persons, of whatever age, who have not sufficient and characteristic marks, and are likely, as at the present time, to be exposed to the infection of small-pox, should be revaccinated. The advantages to be derived from revaccination may be best secured, both for the community and for the individual, by a systematic performance of the operation on every person upon passing the age of puberty. For the community, because a well-grounded confidence would replace the present recurring panics about small-pox; for the individual, because the operation performed as part of a system would be done in the manner most certain to be efficient, and not, as at present too often happens, under conditions little conducive to a protective result.

against any possible mischief that may attend unskilful vaccinations,—the vaccination of every child having been made compulsory within a certain time after birth. *Systematic inspections* of two kinds must be constantly and periodically made by competent persons—namely, one to ascertain as to the effectual performance of the operation, as evinced by the kind of cicatrix visible. This inspection may be most conveniently made in public and private schools; and the School Boards now formed throughout the country ought to keep this object in view. Another inspection should have for its object to ascertain the numbers vaccinated within a certain territory compared with the numbers born in the same place. The Royal Commissioners appointed to inquire into the working of the Factory and Workshops Acts collected most important medical evidence, which caused them to recommend that *successful vaccination* should be a condition to guide certifying surgeons of factories; and that it should form part of the certificate as to the fitness of juvenile applicants to work in factories. It is greatly to be regretted that this recommendation has been overlooked or not adopted. There are at the present time some thousands of young persons and children working in the factories of the United Kingdom who have either not been vaccinated at all or imperfectly, and who would come under medical supervision if the advice of the Royal Commissioners had been followed in the new Factory Act. Small-pox has, for the most part, its origin and existence in the overpopulated localities whence factory labour springs, and great facilities for its incubation exist among the young working in an overcrowded and contaminated atmosphere. Every attempt at *inoculation* from small-pox ought to be made a penal offence. Every case of small-pox ought to be treated in strict seclusion, and be as completely as possible isolated, following out all the directions given with reference to the management of epidemics at pp. 402–407, as are applicable to the case. A quarantine regulation to enforce segregation of the sick from small-pox is of far more importance in this country than it is for yellow fever, which does not find a *habitat* with us.

II. *How the Protective Influence of Vaccination has been Impaired.*

Since vaccination has been generally practised, it has now and then seemed apparent that “*the protective power of vaccination becomes gradually weaker, and at length dies out in the individual.*” Indeed, it seems now to be very evident that “*the protection against VARIOLA or HUMAN SMALL-POX afforded by VACCINATION in COW-POX is for a far shorter period than is generally believed*” (NIEMEYER). No definite result, however, has yet been arrived at as to the absolute duration of the protective influence;

The local effects produced by revaccination vary with a number of conditions. A revaccination with well-chosen lymph (not taken from a revaccinated person), producing some indisputable local result, may be regarded as affording evidence of efficient protection from small-pox. But on the other hand, where no local effect whatever is produced, the person can only be regarded as being in the same position as if the revaccination had not been performed. The practice of repeated or periodic revaccination does not appear to be generally necessary. But in instances where a person, after revaccination, has been subjected to serious constitutional or climatic changes, and is subsequently more than ordinarily exposed to the infection of small-pox, a further revaccination may properly be advised.”

and the practical conclusion is, to advise revaccination of everybody (but especially adults up to twenty or thirty years of age) at the outbreak of any epidemic, no matter what length of time has elapsed since their last vaccination. (Mr. SIMON, through Privy Council, Feb., 1871.) Vaccination must be repeated from time to time, if we would prevent a return of the liability to small-pox, seeing that the protective influence of vaccination is not always persistent through a lifetime. The works and reports which have been mentioned seem to demonstrate the truth of this statement; but in justice to Dr. Seaton (one of the greatest authorities on the subject) it must be stated that he does not subscribe to this belief. Indeed, in his last Report (*Appendix to Public Health Report* for 1861, p. 64), he states that where uniform care was taken in the selection of lymph, and in the performance of the operation, the results did not favour the hypothesis that there had been any necessary deterioration of the lymph,—that he has seen several cicatrices, the results of the vaccinations of Dr. Jenner and Dr. Walker, and that the work of the vaccinators to whom he refers, will bear comparison with the results obtained by these great authorities; and that the great object to aim at is *to vaccinate well in infancy and childhood*. In 1809, Mr. Brown, of Musselburgh, near Edinburgh, published his opinion that the protective virtue of cow-pox diminished as the time from vaccination increased. In 1818 and 1819, small-pox prevailed in Scotland as an epidemic, and many vaccinated persons passed through a mild form of variola. The terms “modified small-pox” and “varioid disease” about this time came into general use; and two classic monographs on the subject made their appearance, one by Dr. Monro, in 1818, and another by Dr. John Thomson, of Edinburgh, in 1820. Dr. Copland also writes that he saw and described, as early as 1823, small-pox as it affected members of the same family at different periods after vaccination, and in young persons who had been vaccinated only ten or eleven years. Contrasting such cases, he found that the severity and fully developed condition of small-pox was generally in proportion to the length of time which had elapsed from vaccination (*Dictionary of Practical Medicine*, Art. “Small-Pox,” p. 815). Again, from the evidence contained in the bills of mortality of 1825—from the experience of epidemics of small-pox in France and Italy in 1826, 1827, and 1829—from the experience of epidemics of small-pox in Ceylon in 1833 and 1834—and from the admissions into the London Small-Pox Hospital in 1838, it has been rendered obvious that the susceptibility to small-pox (which in vaccinated persons is destroyed for some years), returns with advancing age, and becomes greater as life advances. Some of the phenomena, also, which the practice of vaccination itself has made known to us, tend to establish the doctrine of a gradual impairment of vaccine protection, due to *lapse of time*, and as a result of *physiological changes in the healthy body*. This is especially indicated by the fact, that in proportion (undetermined) to the distance of time that has elapsed from the first implanting of the vaccine virus, so is the better development of the vaccine vesicle produced by revaccination. It has been shown, however, from a careful analysis of cases, that the lesser protectedness of certain vaccinated persons bears at least some proportion to the number of years which had elapsed since vaccination. Any *uniform*

rate of increased susceptibility to small-pox from year to year from the period of vaccination has not been demonstrated; but an increasing susceptibility to small-pox continues up to about thirty years of age at least, after which period of life it seems that the liability to contract small-pox continues to decline (HEIM, MOHL, RETZIUS, MARSON, SIMON). Surgeon-General Dr. Graham Balfour, adverting some time ago to these important facts, indicated the practical result to which they point—namely, *revaccination, as a most necessary supplemental measure to vaccination*. A large reduction in mortality, and in the occurrence of small-pox, can be shown to have taken place from the practice of *re-vaccination*, so as to leave no doubt of its practical efficacy. The records of the earliest experience of its usefulness date from Wirtemberg, 1829 to 1836. In 1833, between 40,000 and 50,000 adults were *revaccinated* in the Prussian army, and in about 33 per cent. of the entire number this *re-vaccination* “took” with perfect success. Amongst Russian soldiers at Kasan, the rate of perfect success was about 18 per cent. In the army of Denmark, from 1843 to 1847, nearly 20,000 *re-vaccinations* were practised, of which more than a half were attended with *perfect* success, and more than a quarter with *modified* success. Since 1843 *re-vaccination* has been compulsory in the Bavarian army. From that date till 1857 not even a single case of unmodified small-pox has occurred, nor a single death from small-pox. Similar good results have followed the institution of *revaccination* in the Danish army, the army of Sweden, of Baden, and in the British army also, according to Dr. Balfour’s interesting report for 1859. So great, indeed, is the practical importance of *re-vaccination*, that in the British army a departmental order was issued by circular, of date 21st September, 1858, and is still in force, which ordains that “every recruit, without exception, on joining the headquarters or dépôt of the corps or regiment to which he belongs, shall be vaccinated, even if he should be found to have marks of small-pox or of previous vaccination, and that a monthly return of the results (as to, (1.) a perfect vaccine pustule following the operation, or, (2.) a modified one, or, (3.) a failure) shall be forwarded by the Director-General” (*Statistical Report* for 1859, p. 21). On the other hand, it must be remembered, as Mr. Marson clearly shows, that “probably *revaccination* does not afford the same amount of protection that the first vaccination *well performed* does. *To vaccinate well in infancy* should be looked upon as the sheet anchor; and therefore a careless vaccination should be deprecated at all times, practised under the belief that, if it fails to take effect properly, it will be of no consequence, as the operation can be repeated. By such a proceeding the vaccination often takes effect *badly*, and will never afterwards take effect *properly*, and the individual may take small-pox *severely*.”

It has been alleged (but sufficient proof has not yet been adduced to show) that the vaccine virus becomes deteriorated by its passage through numerous human bodies. In other words, it has been supposed that its protective influence is weakened by length of time or of use, in consequence of the long succession of subjects through whom it has been transmitted since its direct inoculation from the cow. This doctrine is opposed to the obvious pathological fact, that the specific virus of cow-pox, small-pox, and other similar diseases, multiplies and reproduces itself in the system of

those who suffer in the natural course of these diseases. Considerable differences of theoretical opinion prevail upon the point. In the *Report of the National Vaccine Establishment* for 1854, it is stated "that the vaccine lymph does not lose any of its prophylactic power by a continued transit through successive subjects." Such an unqualified belief is not, however, by any means universal. It is certain that the vaccine lymph, when taken direct from the cow, shows an amount of infective power which is not usual in lymph of long descent; but how much of this effect is due to local irritation simply, and how much to specific action, does not seem certain. Lymph direct from the cow "takes" (as the phrase is) in persons with whom lymph of long descent has failed. This is more often obvious in revaccinations. It excites local changes of an intenser kind, so active, indeed, as to render caution necessary in its selection and use. The vesicle produced by it runs a full course, compared with which the progress of vaccine vesicles from lymph of long descent seems unduly rapid, and their termination premature. Also, the lymph direct from the cow renders more certain, and apparently more characteristic, that slight febrile disturbance which is proper to the action of cow-pox on the human system. Such febrile disturbance is undoubtedly an essential pathological phenomenon, which is beyond all proportion in comparison to the trifling extent and intensity of the local inflammation of the skin round the vaccinated part. This specific febrile state seems essential for the due protection of the vaccinated person and for the perfect development and local multiplication of laudable and efficient lymph at the spot where the specific vaccine virus was originally implanted. A bodily temperature of 104° Fahr. has been observed where the local inflammation seemed to be very slight (NIEMEYER). The more distinctly and typically the specific febrile action is expressed which follows the implanting of the vaccine virus, the more certainly is the person protected, and the more efficient is the local development of the lymph which has been multiplied at the sight of implantation. The development of any other febrile state, such as that from cold, or any other disease, is apt to hinder the development and progress of the vaccine vesicle altogether. Referring to the records of revaccination in the Prussian army, an extremely interesting fact is brought out by Mr. Simon, tending to confirm the doctrine that, by transmission through a succession of persons, the vaccine virus has degenerated—namely, "*that the revaccinations of 1836, as tested by eventual resusceptibility to cow-pox, were not half so stable as the vaccinations of 1813.*"

On the whole, therefore, there appears to be still room to believe that any diminution of protective influence from vaccination may be due to personal carelessness—first, in the *selection* of lymph for use, direct from the cow or of lymph of long descent; and secondly, in the choice of cases to continue the vaccinations from—cases, for example, being chosen where the lymph of the local vesicle at the site of vaccination has been developed in the *absence of the constitutional specific febrile phenomena*; and in which the lymph contents of the vesicle are not only impotent, but the anatomical development and structure of the vesicle in respect of its dissepiments are at the same time incomplete and imperfect; showing from its anatomy that its specific physiological qualities do not exist.

That the *protective power* of vaccination has been impaired by imperfect

vaccination, has been shown by Mr. Marson, to an almost incalculable extent—a fact which does not seem to be duly appreciated as yet, either by the Medical Profession or by the Public; and the results of spurious vaccination are demonstrated by large observations made by medical officers, alike of the United States and Confederate armies, during the American War. The following is a summary of these results:—

“(1.) There were three kinds of spurious vaccination prevalent in the American armies,—(a.) that which occurred in consequence of the loss of the specific property in the once good lymph or crust used; (b.) that which resulted from the impairment or destruction of the vesicle as soon as it began to form, in the marching and excessive exercise of the soldier, and that resulting from the use of the sero-purulent matter of such destroyed vesicle in vaccinating other persons; (c.) that resulting from the employment of matter from pustules or crusts that never had the genuine qualities of vaccine virus. (2.) Scurvy and all the asthenic dyscrasie of army life, not only prevented or greatly impaired the normal operation and effect of genuine vaccinia when soldiers with such conditions were subjected to vaccination, but they frequently became the causes of certain morbid phenomena, as obstinate ulcers, &c., which caused the greater part of the evils from vaccination during the war. (3.) In the armies it was never proved that the normal vaccine vesicle communicated any other than normal innocuous virus; but from carelessness in taking lymph at its perfection, and by neglecting to observe the rule never to use lymph or crusts not perfect in all respects, and free from blood or pus, frequent instances of inoculation with purulent matter or unhealthy blood happened. (4.) By the use of matter, fluid or concrete (purulent or morbid in either case), taken from sores of any specific and enthetic character, as (a.) erysipelatus and ethymatous; (b.) that of zymotic ulceration and destruction of tissues, and possessing the properties of a morbid poison; (c.) syphilis, primary or secondary, whether communicated by a lancet, or contaminated vaccine points or crusts; or, as occasionally happened, the manifestation of syphilitic phenomena in connection with, or supervening upon, genuine or spurious vaccination. (5.) The deterioration of genuine virus, by transmission through scorbutic and unhealthy persons, or where at the time of revaccination the protective power of a former vaccination was partially retained, or to the continued use of virus from adult soldiers, many of whom were suffering from unhealthy influences, instead of using lymph from the primary vesicles of healthy infants. (6.) The destruction or deterioration of originally good virus by heat and humidity. These results substantiate the observations of Jenner as to the necessity of guarding against *deterioration of the virus* of cow-pox, which, losing its specific property, ceases to be prophylactic. Army experience would go to prove that genuine vaccination is an absolute safeguard against small-pox” (Dr. ELISA HARRIS, United States Sanitary Commission, 1867).

The operation of vaccination ought to be performed in childhood, and it is ordained by law, 16 and 17 Victoria, chap. c., 1853, to be performed within *three*, or in case of orphanage, within *four months* of birth. The infant ought to be at least from *four to six weeks* old before a disease, sometimes attended with considerable febrile disturbance, is engrafted upon the constitution. Under *six weeks* of age infants should

never be vaccinated, unless in cases of urgent necessity, such as small-pox being in the vicinity. The age of *three months* is on the whole to be preferred. The child ought to be in good health; free from any eruptive cutaneous disease; free from disorders of teething; from disorders of the bowels; and other diseases peculiar to the age of childhood, otherwise the protective influence of the vaccination cannot be depended on. Weakly children, inclined to scrofula, ought not to be vaccinated during their first year of life, and not till the second or third year, when the teeth shall have been developed (Sir JAMES CLARK, COMBE, NIEMEYER).

Difference of opinion exists as to the number of vesicles it is proper to graft upon the arm, and the size of them. Some believe the person to be as thoroughly protected by a small vesicle, "the tenth of an inch in diameter, as if the arm were covered with inoculated points;" and many vaccinators regard the multiplication of vesicles only as a safeguard against failure, and attach value only to *one* successful insertion of the vaccine lymph (BUCHANAN, *Appendix to Fourth Report on Public Health*, for 1861, p. 111). On the other hand, the official instructions issued to vaccinators in England contain the following directions:—"In all ordinary vaccinations, vaccinate by *four or five separate punctures*, so as to produce *four or five separate good-sized vesicles*; or if you vaccinate otherwise than by separate punctures" (for some vaccinators prefer to make long scratches, side by side, or intersectingly, instead of punctures), "take special care to secure the production of *four or five separate good-sized vesicles*." This is considered necessary for securing to those who are vaccinated the full amount of protection which good vaccination confers. The superior value of several vesicles is especially insisted upon by our highest authorities, Marson, Seaton, and Simon. They have shown a constant relation to subsist between the *number* of the sufficient cicatrices and the *degree* of protection afforded.

The skin covering the insertion of the deltoid muscle is the place generally chosen for implanting the specific lymph of *variola vaccinae*. The methods of operating are as follow:—(1.) The part of the arm to be operated upon should be grasped with the left hand, and the thumb of that hand should draw the skin with sufficient tightness, so as to facilitate the introduction of the point of a lancet with the other hand. Three or four punctures should be made near each other, for each intended vesicle. These punctures should penetrate the cuticle to the extent of a few lines in an oblique direction, so as to make a minute valvular aperture, and so as to impinge upon or penetrate the *cutis vera*. The lancet used to make the punctures should be charged with the vaccine *virus* contained in lymph taken from a healthy child who had not been vaccinated before. Certain it is that lymph taken from the vesicles of revaccinated persons is much less certain to produce normal pocks than that taken from persons vaccinated for the first time (NIEMEYER). It should be taken on the seventh or eighth day after vaccination, from vesicles which are perfectly normal as to size, form, and areola. The lancet, loaded with the virus, should be allowed to remain in the punctures for *several seconds*, and, in the course of its removal, the site of puncture should be compressed for a moment or so, to prevent bleeding, and also to retain the virus from the lancet's point. About five punctures should be made, and sufficient space left between

each to ensure prevention of the pocks becoming confluent. (2.) Another mode of operation is often chosen—namely, to make an immense number of minute scratches over a very limited area of skin, and as close together as possible. In this way the number of groups of scratches will correspond to the number of vesicles intended to be engrafted. The scratches may be made with the point of a clean lancet, and may be either parallel to each other, or crossed in two or any number of directions. The number of these groups of scratches will vary according as three, four, five, or more vesicles are considered necessary, and the length of the individual scratches will determine the size of the resulting vesicle, and, to some degree, the soreness of the arm. It is necessary to remember these facts in dealing with young and delicate children, so as not to give rise to unnecessary suffering, torment, and danger. The scratches should be so slight as barely to result in the faintest possible exudation of blood, and that only after the lapse of a second or two. To the group of scratches from which blood first exudes, the “point” or lancet is to be applied, charged with the specific *virus*. The lymph containing this *virus* will be at once absorbed; and the blood with which the lymph may be mixed should be smeared over and pressed into the other scratches in succession several times. (3.) Simple abrasion of the cuticle is sometimes resorted to with very good success—namely, by scraping off the cuticle with the lancet, as an eraser is used to remove blots from paper (*Fourth Report on Public Health*, p. 107).

Arm-to-arm vaccination ought to be the rule; but otherwise, the lymph preserved in glass tubes, with the ends sealed, is the best. The part of the arm operated upon should be selected where it can be constantly covered, even when short sleeves are worn by children. If the operation is unsuccessful, it ought to be repeated after an interval of a few months. Compresses, wet with lead lotion, are the best application, if much inflammation extends round the points of puncture.

Signs of Successful Vaccination.—By the end of the second day small spots appear elevated over the sites of the punctures, or over the groups of scratches or abrasions. These, when examined by a simple lens, are seen to be vesicular, and surrounded by a slight redness. This stage continues for three to four days from the date of engrafting the virus. About the *third*, but rather towards the *fourth* day, the elevation is more perceptible and more red; and by the *fifth* or *sixth* day a distinct vesicle is obvious upon it, of a whitish colour, having a round or oval form, an elevated edge, and a depressed centre. Late on the *seventh*, or early on the *eighth* day, an inflamed ring or areola begins to form round the base of the vesicle, and continues to increase with the development of the vesicle during the two following days. This areola is of a circular form, and its diameter extends from one to three inches. *On the eighth day the vesicle appears distended with a clear lymph. This is the day of its greatest perfection; and it is then the proper period for obtaining the specific virus for continuing vaccination on others. The vesicle is now circular and pearl-coloured; its margin is turgid, firm, shining, and wheel-shaped.* Having reached its height on the *ninth* or *tenth* day, the development of the bright red areola is accompanied with considerable tumefaction of the skin, and with hardness and swelling of the subjacent areolar tissue. This ery-

thematous ring is often the seat of small vesicles. By the tenth day, also, the febrile symptoms of constitutional disturbance are well expressed, the lymphatics of the arm are engorged, and sometimes a roseolous rash supervenes over the body. On the tenth or eleventh day the areola begins to subside, leaving, as it fades, two or three concentric circles of redness. The vesicle now begins to dry in the centre, and acquires there a brownish colour. The lymph which remains becomes opaque and gradually concretes, desiccation commences, and tumefaction subsides, so that by the fourteenth or fifteenth day the vesicle is converted into a hard round scab of a reddish-brown colour. This scab contracts, dries, and blackens; and may fall off about the twenty-first to the twenty-fifth day from the date of vaccination. It leaves a cicatrix, which commonly is permanent in after-life. Indeed, the mark of a good cicatrix is indelible if it is not injured (GREGORY, MARSON, CEELY, CAZENAVE, SIMON). While these local changes are in active progress, febrile phenomena become established—first, so slightly from the fifth to the seventh day, that often the fact passes unobserved; and again, more considerably during those days when the areola is about its height. The patient is then restless and hot, bodily temperature 103° Fahr., or *more*, with more or less disturbance of stomach and bowels. About the same time, especially if the weather be hot, children of full habit not unfrequently show on the extremities, and less copiously on the trunk, a lichenous, roseolar, or vesicular eruption, which commonly continues for about a week. When vaccination is performed on such adults or adolescents as have not previously been vaccinated, and likewise when lymph is employed which has recently been derived from the cow, the resulting phenomena, as compared with the preceding description, are somewhat retarded in their course, and the areola is apt to be much more diffuse. There is also more feverishness, and eruption is less frequently seen (SIMON, *Health Report*, 1859).

Signs of Successful Revaccination.—When persons who have once been efficiently vaccinated are, some years afterwards, revaccinated with effective lymph, there sometimes result vesicles which, as regards their course and that of the attendant areolæ, cannot be distinguished from the perfect results of primary vaccination. But far more usually the results are more or less modified by the influence of such previous vaccination. Often no true vesicles form, but merely papular elevations surrounded by areolæ; and these having attained their maximum on or before the fifth day, afterwards quickly decline. Or, if vesicles form, their shape is apt to vary from that of the regular vesicle, and their course to be more rapid, so that their maturity is reached on or before the sixth day, their areolæ decline on or before the eighth day, and their scabbing begins correspondingly early. In either case the areolæ tend to diffuse themselves more widely and less regularly, and with more affection of the areolar membrane, than in primary vaccination; and the local changes are accompanied by much itching, often by some irritation of the axillary glands, and in some cases, on the fourth or fifth day, by considerable febrile disturbance (SIMON, l. c.)

Characters of the Cicatrix after Vaccination.—It seems now to have been agreed to arrange the characters of cicatrices after vaccination into

the following *three* classes:—(1.) “*Typical*,” “*excellent*,” “*perfect*,” “*good*,” or “*first-rate*” cicatrices are recognised by their circular form and pale or white appearance. They are somewhat depressed, and dotted, indented, or foveolated with minute pits or depressions over the base, supposed to indicate the number of compartments in the anatomical structure of the vesicle (referred to at p. 444). In some instances there are radiations from the centre. It has been considered that the normal diameter of a cicatrix produced by a single insertion is *one-third* of an inch; that scars of larger measurement are generally of double or multiple origin.

(2.) “*Fair*,” “*passable*,” or “*modified*” cicatrices possess the characters of the typical cicatrix, but they are less perfectly expressed, the contour being less regular, and the size just *within* the average above-mentioned. To irregularity of contour, however, it must be remembered that scars resulting from single insertions (as in the ordinary method of puncture) are notably uniform, so that irregularity of contour, when associated with a single puncture for vaccination, indicates that the progress of the vesicle has been irregular; but where the scar results from several contiguous insertions or scratches, no such inference can be made.

(3.) “*Bad*” cicatrices, which must be held as denoting “failures,” are such scars as cannot be recognised as the product of vaccination, by any circumstance beyond being found near the usual site of the operation. Scars also having a less diameter than a *quarter* of an inch ought to find a place amongst this class; and generally, all ill-defined, faint, scarcely discernible white patches, especially such as consist of large, flat, ill-defined shiny marks. Fruitless attempts at vaccination may be also recognised by the permanent traces left of the parallel or transverse scratches employed at the operation. It is, however, very difficult to describe the extent of differences between the results produced by different vaccinators. A large amount of *bad*, and a still larger amount of *second-rate* vaccination has been found to prevail in many districts; as the result of the inspections instituted by Mr. Simon in 1860 and 1861 abundantly testify. Medical men are found to vary exceedingly in their estimate of a satisfactory vaccine vesicle and cicatrix, or the reverse, for the standard is comparative rather than absolute (SEATON, SANDERSON, BUCHANAN). This is exactly what might have been expected, seeing that medical students have hitherto been left to pick up their knowledge of vaccination where they can. In fact, practical medical education at our schools of medicine has hitherto, or until very recently, been entirely *nil* in regard to this most important subject; and no test of knowledge has ever been applied. Many men, whose estimate of the quality of the resulting cicatrices is of a low standard, can scarcely appreciate the typical character of marks which are the ordinary results of good vaccination (SEATON). Excessively small cicatrices are apt to be the result of imperfect vaccination, and there are great varieties in the size of cicatrices of perfect character. It is therefore fairly presumed that cicatrices which thus vary cannot all have precisely the same value. The hand of different vaccinators can even be recognised by the kind of marks they leave behind them. The marks of some vaccinators are conspicuous for their excellence; the marks left by others are not so; and hence there are great differences between the vaccination of districts where different

vaccinators are employed. In the schools of large towns, for instance, Mr. Seaton informs us that, "where the work of many vaccinators was seen together, it was frequently possible to fit the work to the vaccinator by the kind of cicatrix."

With regard to the means of estimating the efficiency of vaccination, it seems established, that "*a distinct connection subsists between the NUMBER and the QUALITY of the cicatrices and the protection conferred by vaccination against small-pox; so that it may be confidently stated that that vaccination is the most efficient from which the most and the best cicatrices result.*" The evidence derived from the records of the Small-Pox Hospital, collected by Mr. Marson, regarding the superior value of several rather than few vesicles, appears to be conclusive on this point. Looking, therefore, to the characters or quality of the cicatrices, and to the number of the vesicles which have given rise to these cicatrices, FOUR DEGREES of protection conferred by vaccination may be specified, and the community inspected may be arranged into the following FOUR classes:—(1.) Best protected—having more than two TYPICAL marks. (2.) Sufficiently well protected—having two TYPICAL marks.* (3.) Moderately protected—having two or more PASSABLE, or one TYPICAL mark.† (4.) Badly protected—having BAD marks, or having only one PASSABLE mark.

Selection of Lymph for Vaccination.—The lymph used for vaccination ought to be taken from the vesicle on the EIGHTH day—the day-week after the operation—when the lymph is yet clear and the vesicle turgid, firm, shining, pearl-coloured, and translucent, and before the vascular zone has reached its full development. The lymph ought not to be taken from any but perfectly "*typical*" vesicles. Inferior, or merely passable vesicles, ought not to be used to propagate lymph. Small vesicles, exhausted vesicles, or vesicles far advanced (such as tenth or twelfth day) are to be avoided. Very early lymph appears, as a rule, to give the worst cicatrices. Thus the careful vaccinator does not indifferently vaccinate from the arms of all infants brought back on the eighth day, but exercises his judgment in selecting from among them. The fresh lymph from the vesicle ought to be engrafted *directly* upon the arm of the child about to be vaccinated—"arm-to-arm" vaccination, as it is called. Dry, or otherwise preserved lymph ought only to be used when fresh lymph cannot possibly be obtained. Properly dried lymph, however, seems capable of producing quite as good results as arm-to-arm vaccination; but it demands incomparably more care than it generally receives, first in its storage, and afterwards in its use. It may be dried and stored on "points" of ivory or bone, or upon small pieces of glass glued together by the dried lymph itself, or dried on lancets. These should be well charged—*i.e.*, coated twice or even thrice with the lymph, and rolled up in a covering of goldbeater's skin, and still further secured from atmospheric influences by an outer case of tinfoil hermetically sealed, or in a phial carefully corked, in which they may be packed with cotton, if they require to be transmitted to any

* Among cases in which the two cicatrices were *well marked* the death-rate was $2\frac{3}{4}$. Among cases in which they were *badly marked* it was $7\frac{1}{4}$.

† Among cases in which the one cicatrix was *well marked* or *typical*, the death-rate was $4\frac{1}{4}$. Among cases in which it was *badly marked* the death-rate was 12.

distance. Glycerine has been used with success to keep the lymph liquid. It may also be preserved in the capillary tubes devised by Dr. Husband. These are the best lymph carriers, and Dr. Ward Cousins, of Southsea, has adapted to them tube holders, thus removing old objections to these tubes, and rendering their application in practice easy and convenient. He has arranged the whole apparatus necessary for vaccination in a case containing two holders, for charged and uncharged tubes, of which there is a supply, consisting of a lancet with capillary tube cutters; and a supply of wax tapers for closing the tubes. By proper care, complete and perfect vaccination may be attained under every variety of method; but bad vaccination, as it prevails at present, is almost always directly dependent on the careless employment of improperly preserved *dry* lymph, and indirectly associated with irregularity of inspection, in consequence of which the vaccinator remains unaware of the number and extent of his *failures*, and loses all the advantages of experience. "The use of the capillary tubes of Dr. Husband affords considerable advantages to the public vaccinator, especially if his district be rural—*Firstly*, Because it furnishes him with an efficient means of maintaining his supply without having recourse to extraneous sources, and enables him to dispense altogether with the use of 'points,' 'glasses,' &c.; *Secondly*, Because in thinly populated neighbourhoods, in which experience shows that it is impossible to assemble all the children at any particular station, it enables him with equal advantage to vaccinate from house to house" (SANDERSON in *Public Health Report*, 1861). For a detailed account of Dr. Husband's method of preserving lymph, the reader is referred to the *Second Report of the Medical Officer of the Privy Council*, 1859. The protective influence of vaccination has been impaired by nothing so much as by imperfect vaccination; and so long as a public vaccinator is at once the operator and the judge of its success, imperfect vaccination may continue to impair the only remedial agent we have, which, if universal and efficient, would, humanly speaking, stamp out small-pox.

Not only at present is there no security that every child born shall be vaccinated, but there is no security that the true result of that vaccination has been seen and registered as successful or the reverse.

To remedy this defect, in carrying out the spirit of the Vaccination Acts, there ought to be a public referee or inspector for limited districts. On the seventh or eighth day after vaccination by the public vaccinator, the child or adult operated on, whose name has been entered in the vaccine station book, ought to be forthcoming (under a penalty) before the inspector of vaccination, who should judge as to its success or the reverse, and who should enter the result in a book. The records of the vaccinator and of the inspector ought to check each other by the names and dates of the children or persons vaccinated, and so give us reliable data as to efficient vaccination, which we do not at present possess.

CHICKEN-POX.

LATIN EQ., *Varicella* ; FRENCH EQ., *Varicelle* ; GERMAN EQ., *Windpocken* —Syn., *Wasserpocken*, *Varicellen* ; ITALIAN EQ., *Varicella*.

Definition.—*A disease the result of a specific contagium capable of inoculation, and having a period of incubation of eleven days (MURCHISON and SQUIRE) ; it consists of a specific eruption, in a series of new crops, usually appearing for several days in succession ; so that dried and fresh vesicles are often alongside of each other, on the breast, back, face, and extremities, preceded by fever. The disease may be protracted for a fortnight or longer, and may be epidemic. It affects the system only once.*

Pathology.—This disease derives an importance which it does not of itself possess, in consequence of its resemblance to small-pox, with the modified form of which it has been considered by some to be identical ; but the evidence is now conclusive that they are distinct diseases. It is for the most part peculiar to childhood and early adult age ; and its infectious origin may generally be traced. That it is communicable has been proved by inoculation, by Professor Steinar of Prague, who inoculated ten cases from the contents of the vesicle of varicella, in whom eight were successful—varicella and not variola being always produced, previous vaccination having no influence whatever on the result. In each of the eight cases the period of incubation was eight days (*Brit. Med. Journ.*, May 8, 1875, p. 610). Thus like small-pox and measles, the incubation period is shorter by inoculation than when it is contracted in the ordinary way. The theory of the disease, therefore, is, that a specific poison, after a given period of latency, gives rise to primary fever, which lasts from twenty-four to seventy-two hours, when the eruption appears, and runs a course of eight or ten days. The fever is much mitigated on the appearance of the eruption, and entirely subsides with it. That fever precedes the eruption is a phenomenon observed so generally that no exception is to be found in the accounts given of the disease by any writer excepting Heberden. It is of a mild character, and though for a few hours it may seem severe, yet it seldom passes into a stage so severe as to have the tongue of a brown and coated appearance. Nevertheless there is general discomfort, gastric derangement, loss of appetite, and headache. The eruption has three stages,—that of small papule, of vesicle, and of incrustation. After the fever has lasted from twenty-four to seventy-two hours, a number of red papulæ appear, which become vesicular, and perhaps in a few points pustular, on the first day. On the second day the vesicles are filled with a whitish or straw-coloured lymph. On the third and fourth days they attain their greatest magnitude, when they become *acuminated*, and shortly afterwards they burst and shrivel, except those which contain purulent matter, and have much inflammation around their base. Pus, indeed, is so rarely found, that when it does appear, it may be secondary to some local excitement of inflammation surrounding a vesicle. Individual vesicles develop in from six to twelve hours, their contents becoming cloudy on the second day, and commencing to dry up on the fourth. On the fifth day they begin to crust, and in four or five days

more the crust falls off, leaving for a time red spots on the skin, generally without, but sometimes with a "pit" or depression. The "pit" is permanent, the cicatrix generally whiter than the original tissue, and the patient consequently is marked or scarred. The eruption usually consists of a series of crops which succeed each other at intervals of twenty-four hours, and die away in the order of their occurrence. The first crop usually appears on the breast and back, where it is also generally most abundant, and afterwards on the face and extremities; but the face sometimes remains quite free, the brow or forehead being less frequently exempt. The number of crops may be limited to two or three, while in other cases a new succession will appear every twenty-four hours for ten, twelve, or fourteen days.

Symptoms.—Of varicella there were wont to be three forms recognised—the *varicella lenticularis*, the *varicella conoides*, and the *varicella globata*. The symptoms of these varieties are similar to each other, their only differences consisting in the size and form of the vesicle—that of the *varicella globata* being the largest. The fever which precedes the eruption is often as severe as that which precedes mild small-pox or measles; but it generally, though not constantly, remits on the appearance of the eruption, and does not return as the eruption approaches maturity. The urine is usually little affected in the early stages, when it is often as limpid as in hysteria; but when the fever runs high, it assumes the usual febrile characters (PARKES). The *globate chicken-pox* is known as the *swine-pox*, or, vulgarly, "*the hives*." The eruption consists of large vesicles not quite circular in form, but often a little larger than the pustules of small-pox, surrounded with a red margin, and containing a transparent fluid, which, on the second day of the eruption, resembles milk whey. On the third day they subside, shrivel, and present a yellow tint. Before the conclusion of the fourth day they are converted into thin blackish scabs, which dry and fall off in four or five days more.

Diagnosis.—Dr. John Thomson, who carefully studied this disease during the epidemics of 1815 to 1821, concluded that it was impossible to distinguish clinically chicken-pox from modified small-pox; but the most important pathognomonic characters derived from the respective phenomena of both diseases are as follow:—

1. Chicken-pox emits a peculiar odour, different from that of small-pox, and less decidedly partaking of the variolous fetor.
2. Chicken-pox appears indiscriminately, and almost equally all over the person, beginning first on the trunk in general, and then appearing on the face and scalp; while small-pox appears first on the face and neck, and the pimples are more numerous on the face than on any other part.
3. Chicken-pox eruption is generally completed in the space of twenty-four hours, or solitary vesicles come out irregularly afterwards in different points; but, in small-pox the eruption begins in the evening of the third, or morning of the fourth day, and proceeds regularly for the ensuing three days, until it is completely established.
4. While variolous pustules are on the first and second days of the eruption small, hard, globular, red, and painful, and communicate to the finger a sensation similar to that which would be excited by the presence of small round seeds under the cuticle; in chicken-pox, every vesicle almost has on the first day a hard red margin,

but communicates to the finger a sensation like that from a rounded seed flattened by pressure. 5. On the second or third day of the eruption of chicken-pox, the individual bodies are vesicles containing serous fluid, and giving them a whitish aspect. 6. These vesicles are surrounded by little or no inflammatory redness, and do not naturally, and independent of external violence, proceed to suppuration. 7. Chicken-pox may be confidently distinguished from small-pox on the third and fourth days by the state of the vesicles, some of which, being left entire, are shrivelled and wrinkled, while others, whose ruptured tops have been closed by incrustation of their fluid, are marked by radiating furrows. None present depressions on the *apices*; and as they do not suppurate, they incrust and disappear sooner than variolous pustules. 8. The marks left by chicken-pox, when they do leave marks, present a peculiar conformation, being round or elliptical, and less frequently irregular than those of small-pox, and in general smooth and shining. Lastly, it is said by Luders, that while small-pox is formed in the *cutis vera* or corion, the chicken-pox eruption is formed in the tissue situate between the corion and cuticle (CRAIGIE, Vol. I., p. 614).

Against the identity of chicken-pox and small-pox, there also stands the fact of the frequent occurrence of *varicella*, rather than small-pox, in children that have never been vaccinated, and that its occurrence does not prevent them taking small-pox, nor prevent successful vaccination. It is also known that children have been attacked by *varicella* who have been vaccinated, or had variola only a few weeks before,—all which are opposed to any pathological identity with small-pox. *Varicella* must therefore be regarded as a specific disease, *sui generis*, and distinct from small-pox. It usually occurs in more or less extensive epidemics, which occasionally accompany, precede, or follow small-pox epidemics, or may be co-existent with measles or scarlet fever.

Treatment consists simply in abstinence from animal food; having recourse to a milk diet; and careful attention to the bowels by saline luxations if necessary. The patient is to be kept at perfect rest and cool, by making him repose on a mattress rather than on a feather bed, with light coverings. It is necessary to be on the outlook against catarrh.

MEASLES.

LATIN EQ., *Morbilli*; FRENCH EQ., *Rougeole*; GERMAN EQ., *Masern*;
ITALIAN EQ., *Rosolia*.

Definition.—*A purely contagious and infectious disease, the virus or contagium passing off in the exhalations of a patient, causing the surrounding air to be contaminated. The disease is capable of inoculation by the blood and secretions. An eruption occurs in crops of a crimson rash, consisting of slightly elevated minute dots, about the size of millet-seeds, and having a small papular centre, scarcely perceptible to the touch, and without any sensation of hardness. Several of these may unite in irregular circular forms, or crescents, or they may be isolated. The eruption appears on the thirteenth or fourteenth day after exposure to infection (PANUM, Archiv. Gén. de Med., April, 1851, p. 451, and*

Ed. Med. Journ., June, 1858, p. 589, quoted by MURCHISON). It is preceded by catarrhal symptoms affecting the conjunctiva and air passages for about four days, accompanied with fever, which commences with repeated rigors—i.e., the real incubation-period is about ten days. It lasts six or seven days, and the whole duration of the disease is completed in from nine to twelve days. Measles affects the system only once; and sometimes prevails as an epidemic.

Pathology.—That an organic poison is absorbed in cases of measles, and infects the blood, is proven by its inoculation and results, inducing, after a period of incubation of ten or fourteen days (according to the mode of calculation*) a specific continued fever, which is followed at the end of three (more generally of four, and in some few instances of five) days, by a secondary or specific inflammation of the skin and of the mucous membranes of the eyes, nose, mouth, fauces, and bronchia. In a few cases the poison has more remote effects, inducing inflammation of the substance of the lungs, or of the pleura, which may aggravate and prolong the illness. The fever may vary in intensity, but it is uniformly present; and before the period of eruption the rise of temperature is very rapid, and often very great. The premonitory phenomena of cough, sneezing, and general *malaise*, are usually more prolonged than in other eruptive fevers. It does not always happen, however, that the functions of the mucous membrane are disordered, as well as the cutaneous surface. There are cases (few in number) in which no catarrhal symptoms occur; and such cases are described as "*morbilli sine catarrho*" during epidemics of the disease. On the other hand, there are cases where the eruption is limited, very indistinct, or altogether absent—"morbilli sine exanthemate"—which unmistakably result from infection with measles-poison. They run the course of a very severe catarrh, and often not without considerable danger to life, especially in those cases, afterwards to be noticed as asthenic septic measles, where patients die of increasing prostration before the appearance of eruption.

Measles was first noticed at the same time and in the same country with scarlet fever, and the two diseases have subsequently followed nearly the same course. They now prevail all over the world, are little influenced by season, are believed to be constantly in existence somewhere, and occasionally epidemic. Though incidental to every period of life, measles is most frequently contracted in childhood, when it is difficult to trace the effects of accidental circumstances; hence our knowledge of predisposing causes is most imperfect. Both sexes appear to be equally liable. With respect to the influence of season, it is generally supposed that measles is apt to begin in winter, increase till the vernal equinox, and

* The incubation-period of measles is quite as definite as that of small-pox. There is, it is true, some discrepancy in the statements of authors respecting it, but much of this discrepancy is due to a difference in the method of calculation. In different works we find the incubation-period set down as between ten and sixteen days, and as being most commonly fourteen days; but when we turn to the facts upon which these statements are founded, it appears that, in the case of measles, the incubation-period has, in the majority of instances, been calculated from the date of exposure to the poison, to that of the first appearance of the eruption; and that, therefore, the stage of *primary fever* (which lasts three or four days) has been included in the period of incubation. Making allowance for this source of fallacy, the incubation-period of measles is usually about ten or eleven days; and just as in the case of small-pox, when the poison is introduced by inoculation the incubation-period is shorter, or only seven days (MURCHISON, *Trans. Clin. Society*, Vol. XI).

then tends to subside towards the summer solstice. The greatest number and the largest epidemics occur in the winter and autumn, or in cold damp summers. The deaths, however, from this disease, registered in England and Wales, show that the influence of season is exceedingly trifling.

Propagation of the Disease by direct Communication and Infection.—It is now fully admitted by competent observers that a patient labouring under measles generates a material poison, which may be communicated directly, or which may contaminate the atmosphere with an impalpable poison. This poison seems to be most infectious when the eruption is out; and next, in the prodromal or catarrhal stage. Like scarlatina, measles is thus eminently communicable; so that no susceptible person can remain in the same room, or even in the same house, with an infected person, without hazard of taking the disease. In the year 1824 it was imported into Malta by some children belonging to the 95th Regiment, and spread extensively in that island, so that many natives died. This circumstance was the more noticeable as measles had not been in the island for many years. Such outbreaks furnish the best field for determining the incubation-period, and the best observations made on this point are those by Panum in the epidemic which occurred in the Faröe Isles in 1846. There were seventeen of those islands inhabited. Prior to April 1846 not a single case of measles had been observed in those islands since 1781; but, within six months more than 6,000 of the 7,782 inhabitants suffered from it. Panum was able to trace the importation of the disease from one island to another; and, in this way he had an opportunity of determining the period of incubation in a very large number of instances under the most favourable circumstances (MURCHISON, l. c.) The *infecting distance* of the poison, it will be plain from what has been stated, must be considerable; indeed, it is often very difficult to isolate the disease in public schools, or other large establishments, where it sometimes appears. The fact of measles being communicable has often been proved; but some difference of opinion has existed as to the possibility of communicating the disease by inoculation. Healthy children have been inoculated either by blood drawn from the arm of a patient suffering from measles, or with serum taken from the vesicles which are occasionally found intermixed with the eruption,—an experiment which appears to have been first made by Dr. Home, of Edinburgh, at the suggestion of Dr. Munro, in 1758, with a view of producing a mild disease; but no such result was obtained. The disease has also been successfully inoculated by Specanza of Milan in 1822; and by Michael of Katoma in 1842, who made 1,122 inoculations, only 7 per cent. of which failed. The induced disease was mild; not one of the inoculated died, although severe epidemic was prevalent at the time. The primary fever appeared on the seventh day, and the eruption on the ninth or tenth day after inoculations made with a mixture of blood and serum from the miliary vesicles or from the tears. More recently measles has been successfully inoculated by several German physicians, of whom Jörg and Wendt assert that the disease from inoculation is not less severe than that contracted in the ordinary way, (MURCHISON, *Trans. of the Clinical Society*, Vol. XI.)

Measles is extremely contagious in its primary or catarrhal stage, and is undoubtedly propagated by *fomites*. The strictest demonstration of this fact is, that the disease has been communicated by direct application of substances impregnated with the virus in attempts to inoculate the disease; it is also proved by the fact that children's clothes, sent home in boxes from schools where the measles has existed, communicate the disease; and by the same circumstance resulting when susceptible children have lain in the same beds, or in the same room, shortly after it has been occupied by patients suffering from the disease. Cold weather appears favourable to the development and propagation of measles. No age is exempt, from the foetus to the second childhood of old age; but it is much more frequent in children than in adults, and there are few who have not an attack of measles at some period of life. Children, however, *under* six months often escape during an epidemic of measles; and very old persons rarely suffer. It not unfrequently makes its appearance at the end of an acute disease (see page 389, *ante*).

The morbillous poison having once produced its specific effects leaves the patient exempt from all liability to a second attack. This law may be considered as proved both by Willan and Rosenstein—the former affirming that, after an attention of more than twenty years to eruptive complaints, he had not met with an individual who had twice had “febrile rubeola;” while the latter states that in a practice of forty-four years he had met with no instance of a second infection. There are, however, occasional exceptions to the rule. One variety of this disease—namely, the *rubeola sine catarrho*—is supposed to afford no protection against an attack of the *rubeola vulgaris*. There are many exceptions, however, to the non-susceptibility of persons who have passed through the *rubeola vulgaris*. Burserius, Robedieu, Home, Baillie, Rayer, and Holland, have all seen instances of a second attack of measles in the same individual.

It seems also ascertained that the specific poison of measles is generated as soon as the primary fever is established, and before the eruption appears—*i.e.*, in the primary or catarrhal stage, (BRISTOWE, MURCHISON, *l.c.*) The cutaneous eruption is one of the great characteristics of the disease. It is evanescent after death. It first appears as a circular spot or blotch, similar to a flea-bite, slightly prominent, and scarcely sensible to the touch. It is “quite free from the feeling of grittiness, so characteristic of small-pox,” although it is a collection of inflammatory exudation at a circumscribed spot. Its colour is of a pinkish red, or deep raspberry hue, and in rare instances (as in the *morbilli nigri*) is livid or black, from extravasation of blood in the cutis. In severe cases, especially if the patient be of tender age, the eruption assumes a papular form; and, when at its height, occasionally a vesicular form, the latter being most common on the arms, the neck, or the breast. The colour of the eruption is evanescent on pressure, but returns on the finger being removed. In most of the spots, however, which have existed for a considerable time, a very slight escape of blood into the cutis complicates the hyperæmia. Such spots lose their colour slowly and incompletely under pressure of the finger; and after the eruption has quite gone, dirty brown stains, or bright yellow discolorations, may be left on the skin. The patches of

eruption are numerous, so that little of the healthy skin intervenes between them; and they not unfrequently become confluent, forming large maculæ, sometimes of a semilunar form. Its seats are the face, back, and loins; the parts least affected being the pudendal and popliteal regions. The inflammation of the cutaneous texture extends in some degree to the subjacent areolar tissue; the face is œdematous, tumid, and swollen, but not so much as to close the eyelids; otherwise the skin may retain its normal colour between the spots. The eruption does not at once cover the whole body, but occurs in three crops, each of which follows the other at an interval of about twenty-four hours, the duration of each crop being from three to four days. In its most simple uncomplicated form, on the third or fourth day of the primary fever (which is continuous) the first crop of the eruption appears on the face, about the mouth and eyes, spreading to the neck, the breast, and upper extremities. On the following day the second crop covers the trunk; and on the third day the third crop appears on the lower extremities, so that the whole body is then covered with the eruption, which is at its height. At this time the perspiration of the patient has a peculiar odour, strongly suggestive "of a freshly picked goose" (NIEMEYER). On the fourth day of the eruption it begins to decline from the face, neck, and upper extremities; and on the fifth day it fades from the trunk. On the sixth or seventh day it is evanescent over the whole body, and terminates by resolution, followed by a furfuraceous or bran-like desquamation of the cuticle generally. During the eruptive state the constitutional disturbance and catarrhal symptoms increase; and the maximum of the fever or fastigium is reached, when the height of the eruption has come to its full extent, about the fifth day. The period of fastigium may last from twelve to twenty-four hours, to be immediately followed by a rapid and almost complete defervescence, the temperature sinking in one night two or more degrees, (Fahr.) It continues to decrease throughout the following morning and day; and on the second day from the beginning of the defervescence the normal temperature is arrived at. It is only in very severe cases that this steady decrease is prolonged beyond twenty-four or forty-eight hours more. In severe cases the decrease of temperature may be slower and more protracted; but if the defervescence be more prolonged, it is a fair ground for suspecting some untoward complication.

A similar course of temperature, as regards defervescence, has been observed to obtain in cases of erysipelas of the face, but the fastigium lasts longer, and the epoch for the commencement of the defervescence vacillates between the fourth and eighth days. It is the amount of fever present—*i.e.*, the increased bodily heat, as measured by the thermometer—which greatly aids in deciding diagnosis and prognosis. From this point of view slight, severe, and complicated cases are to be distinguished. During the period of incubation there is generally no symptoms of disease, but slight feverishness, depression, and catarrh may be present in adults. The commencement may be marked by violent shivering, or merely by chilliness, characteristic of catarrhal fever. The inflammation of the mucous membrane, of the eyes and nasal fossæ, indicated by more or less constant sneezing, and sometimes epistaxis, generally commences either with or before the primary fever, and consequently precedes the

THE FOLLOWING DIAGRAM REPRESENTS THE RANGE OF TEMPERATURE IN A CASE OF MEASLES, IN WHICH THE FEVER IS SEVERE. THE RECORDS INDICATE MORNING (A.M.) AND EVENING (P.M.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE FOURTH DAY OF THE DISEASE.

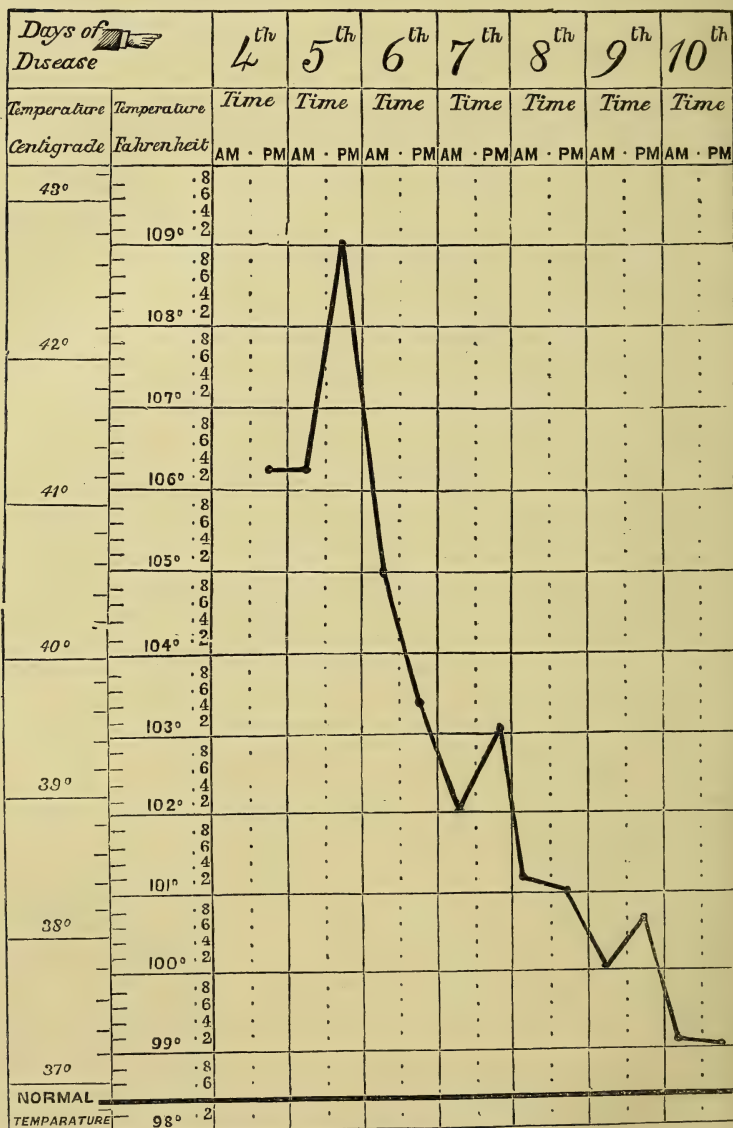


Fig. 82.

eruption by some days. There is increased frequency of pulse, and the local symptoms are very decided. The eyes burn and shun the light,

which causes them to fill rapidly with tears. There is pain over the frontal sinus, and the nasal passages are stuffed up from the swollen mucous membrane in apposition, and the nose discharges a limpid salty secretion, which stiffens linen. This inflammation, for a few hours, may be confined to fixed spots, and is marked by itching at the mucous orifices; then it becomes diffuse, and quickly changes to mucinous discharge. A profuse watery discharge from the eyes and nostrils shortly follows, technically termed "*coryza*," which usually continues till the decline of the eruption, and in some cases to a later period. Children, as a rule, are not anxious to seek their beds; even with a temperature of 104° Fahr. they are still able to remain out of bed; but in cases of pneumonia, with the same temperature, they desire to lie down at once. The temperature rises rapidly towards the breaking out of the eruption, but is rarely severe enough to threaten life, as it is in scarlet fever. Its rise is steady to the *fastigium*, but if remissions are marked they occur in the morning. If the fever is high for many days before the eruption, it indicates a severe case, and is apt to be attended with such nervous derangements as are indicated by somnolency, jactitation, or delirium. The *fastigium*, or maximum of temperature, generally coincides with the period of the eruption. It usually does not exceed 103° Fahr., and simultaneously with this increase of the fever the nervous symptoms are apt to predominate. In the majority of uncomplicated cases the temperature falls within the first *twenty-four* hours after the appearance of the red spots, and the *fastigium* in such cases does not extend beyond *twelve*, and in a few cases beyond *twenty-four* hours. Protracted *fastigia* are always connected with severe cases. In some *defervescence* is completed within *twenty-four* hours—an example of complete crisis. But pauses sometimes occur to interrupt this rapid defervescence—pauses which may extend from *twelve* to *thirty-six* hours. Such are examples of protracted crisis, and are always anxious cases.

The Symptoms of measles result from the fever and the consecutive local lesions. The varieties of the disease, however, are few, so that intensity of the disease merely has led physicians to consider the phenomena under two grades—namely, "*morbilli mitiores*" and "*morbilli graviore*s." The essential characters of the former are such as have been already described—namely, a primary fever, preceding a specific inflammation of the skin and mucous membranes, the defervescence of the fever taking place while the eruption fades. The symptoms may be divided into three stages:—the first embraces the primary fever, or the period before the eruption, and may last from three to five days; while the second stage embraces the period of the eruption, and lasts from seven to six days. These two stages very commonly comprise the whole disease, whose usual course is then from nine to twelve days. The third stage includes any inflammatory lesions which may be caused by the tertiary action of the poison, and only occasionally exists. The early symptoms of the primary fever are seldom severe, and greatly resemble those of an ordinary acute catarrh; so much so that, in the absence of any epidemic of measles, they could not be otherwise recognised. They are, shivering, alternated with heat, frequent pulse, headache, derangement of the bowels, sometimes accompanied by nausea and vomiting; and these affections are so con-

siderable that the patient usually takes to bed. At the end of a few hours the fever becomes continued, and the specific action of the poison commences by the mucous membrane of the eyes and nose inflaming, so that the light is painful; the senses of smell and taste are lost, followed by a copious discharge of mucinous fluid from the nose and eyes, attended with more or less constant sneezing, and sometimes epistaxis. The buccal and bronchial membranes may become affected at the same time, and the patient is then troubled with a frequent cough, which has this peculiarity, that it occurs in paroxysms. The cough does not remit till about the seventh day, and is often accompanied by hoarseness, by a sense of constriction across the chest, so that the cough is short and barking in its character, by diarrhœa, and sometimes by ischuria. At night this kind of breathing and cough may simulate an attack of croup. The duration of this first stage may be three, four, five, or even six days. The second stage commences with the appearance of the eruption, whose course and character have been described. On the appearance of the eruption the fever is often aggravated, but the distressing nausea and vomiting seldom last beyond the fourth day. The fever, therefore, together with the coryza, sneezing, coughing, hoarseness, and diarrhœa, continues with unabated severity till the eruption has reached its height, and is fully out over the whole body, which is on the third or fourth day after its first appearance. From this period, in favourable cases, all the symptoms begin to decline; and on the eruption disappearing the cuticle desquamates, and the disease terminates on the ninth, tenth, or eleventh day from its commencement. In a few cases, however, on the subsiding of the eruption, or about the ninth, tenth, or eleventh day of the disease, and in some instances earlier, the pectoral symptoms do not subside as they ought to do; but inflammation of the substance or of the lungs of the pleura takes place, prolonging the duration of the disorder, and endangering the life of the patient. The inflammation of the bronchial membrane is denoted by the expectoration either of a thick viscid mucus or of pus, and which may or may not be streaked with blood, while the mucous or sonorous rattle will point out the peculiar seat and extent of the mischief. If the substance of the lungs be inflamed, the breathing is more difficult, the cough more troublesome, and the countenance livid; but the loud mucous rattle which accompanies it seldom allows us to hear crepitation, or to determine the absence of respiration in any given portion of the lung. If the pleura be inflamed, we have, in addition to the cough, severe pain in the side, and an impossibility of filling the chest with air, except in a very limited degree. This condition is often accompanied by dulness on percussion, by bronchophony or ægophony, assuring us that fluid is effused into the cavity of the chest. The main characteristic of the more severe form of measles,—*morbilli graves*—is the eruption becoming suddenly black, or of a dark purple with a mixture of yellow. The early writers on measles describe this form of the disease as being much more common in their time than we find it to be in the present day. It is the *asthenic typhous* or *septic* form of measles. Sydenham considers this appearance as extremely formidable, and that persons so seized are irrecoverably lost. Willan writes that he has seen this discoloration, but thinks more lightly of it. The eruption is sometimes greatly delayed from causes not quite manifest. Excessive

purging is thought to have this effect, or anything which greatly debilitates the system, hereditary or acquired unhealthiness of constitution, or the peculiarly malignant nature of the disease. The occurrence of the eruption is therefore to be looked for with anxious care, as the appearance of it, even though late, is in itself a favourable indication. If the eruption suddenly disappears, or "goes in," it is no less an unfavourable omen, and is apt to be attended by other dangerous results—diarrhœa, dyspnœa, coma, convulsions—all which unfavourable signs may again disappear on the re-appearance of the eruption. In these severe cases the eruption is irregular in its spread and appearance, and of various dark shades, as a result of hæmorrhage into the cutis; and petechiæ may appear on the skin interspersed amongst the eruption. In such forms of asthenic septic measles, the pulse, from a strong one, becomes small, weak, and so frequent that, when very small, it cannot be counted. There is great depression, with coldness of the hands and feet, while the body burns. The mind becomes confused, the tongue dry and crusted, and death ensues from prostration, sometimes in convulsions or stupor, even before the appearance of eruption. Great increase of bodily temperature from blood infection, or the blood-poisoning alone, are each, or both, set down as the source of this brain paralysis in this asthenic septic form of measles; but the evidence is more in favour of high temperature being the cause of the paralysis; and that remedies directed to lowering temperature have a favourable effect on the symptoms (NIEMEYER).

Diagnosis.—The diseases with which measles may be confounded are scarlet fever and some forms of syphilitic eruptions. The diagnostic symptoms between measles and scarlet fever are numerous; for there are many differences, both in the general course of the fever, the ranges of temperature, and particular symptoms of these diseases, by which they may readily be distinguished from each other. Thus, the periods of the latency of the poisons are different—that of scarlet fever being from two to ten days, while that of measles is from ten to fourteen days. The eruption in scarlet fever seldom appears later than the second day of the primary fever; in measles it is delayed till the fourth day. In scarlatina the exanthematous patches are large, and the surface they cover ample; but in measles they are not larger than flea-bites, and when most confluent the clusters are small, sometimes forming crescentic patches. The colour is also different, being of a bright red in scarlet fever, while in measles it partakes more of a pinkish red or raspberry hue. The affections of the mucous membranes are also different in the two diseases. In scarlatina the tonsils are almost always greatly enlarged and ulcerated, while in measles they are little or not at all affected. In scarlatina the eyes are free from coryza, while in measles this is the most prominent symptom. The tertiary actions of the poison are also different, being, in scarlatina, inflammatory affections of the joints and dropsy; while in measles they are inflammations of the lungs or pleura; and, lastly, in measles the fever usually subsides on the disappearance of the eruption; but in scarlatina the fever often continues many days or weeks after the eruption has run its course, or till the sore throat has healed.

Prognosis.—The mortality from measles greatly varies in different years. During each of the four years previous to 1858 the proportion of deaths

from measles in every 1,000 deaths from other causes has been, in 1851, 24·107; 1852, 14·599; 1853, 11·818; 1854, 21·463. Percival says, that out of 3,807 cases of measles, 91 died, or 1 in 40. Watson says, that in one year, at the London Foundling Hospital, 1 in 10 died; and in another, 1 in 3. In the same establishment, in 1794, out of 28 cases none died; in 1793, out of 69 cases 6 died; in 1800, out of 66 4 died; and the aggregate of these data will give us an average of 1 death in 15; so that the prognosis in every case of measles is favourable in the first instance. The prognosis, however, is more favourable in the country than in large metropolitan towns; for it appears by the Registrar-General's reports that the proportion per cent. of the population that died of measles in London has been much greater than in England and Wales. It is found that high fever, having small daily fluctuations, especially with the co-existence of nervous derangements, delirium, and the like, are very unfavourable symptoms. Prolific eruptions are more favourable than scanty exanthema. Short duration of the *fastigium*, rapid *defervescence*, and speedy disappearance of the eruption, are the most favourable events. On the other hand, exacerbations are always unfavourable.

Of the complications of measles, the most important is catarrhal pneumonia; and a fatal issue, from such a complication, is apt to ensue in cases of young children. In them the pulse and respirations are enormously accelerated, the face is flushed, and the movements of the body are lively. The contractions of the heart are apt to abate in frequency, and the breathing to become very inefficient; while, simultaneously with diminishing expectoration, the breathing surface of the lungs becomes less and less, owing to the bronchiæ being rendered more and more impervious, as *capillary bronchitis* extends to the alveoli of the lungs. Portions of lungs then collapse, and others become emphysematous. "If measles be accompanied by severe disease of the respiratory organs, and the consequent difficulty of breathing or fever induce collapse, the eruption becomes pale, and may entirely disappear in a short time. This symptom is often wrongly interpreted, when the disappearance of the eruption is regarded as the cause, not the result, of the distress, the collapse, and bad symptoms in the organs of respirations" (NIEMEYER). The mucous membrane of the mouth and fauces in most of the severe cases inflames, but the inflammation differs from that of the eyes and nose in not being accompanied by any discharge. In other respects it is exactly similar to the cutaneous eruption, for a number of exanthematous patches, more or less confluent, are seen upon the palate, uvula, tonsils, and *velum pendulum palati*, and they equally terminate by resolution. They appear also at the same time with the eruption on the face, neck, and upper extremities, but do not decline till the eruption fades from the body generally. The cough and expectoration, which indicate the attack of catarrhal pneumonia, and accompany it, are constant, and the latter shows that it partakes of the same mucinous catarrhal character as that of the nasal and ocular membrane. When the substance of the lungs is thus affected, a serous exudation pervades that tissue, and the quantity of fluid effused is frequently so considerable as to stream from the lung after death as soon as its tissue is divided. In severe forms of the disease, either the red or

grey hepatisation of the lung may supervene; but these results are rare. The pleura does not at all times escape the morbid action; and the diffuse, the serous, the adhesive, and even the purulent inflammation, may invade that tissue, and either destroy the patient or retard his convalescence. Few analyses of the urine in cases of measles have been made. Albumen is extremely common in some epidemics, and appears simultaneously with the eruption; it may then disappear, and reappear during the fading of the rash. Blood in small quantities is also common. In the Leith epidemic of 1854 the recoveries were most speedy when the albuminuria was the greatest (PARKES *On the Urine*, p. 262).

The chief danger arises from bronchial and pulmonary inflammation, and the danger of this is greater after the disease has begun to decline than during its progress. An epidemic of measles occurred at Kiel in 1860. In the fatal cases the chief cause of death was a peculiar state of the lungs, which in part were collapsed, with *foci* of purulent infiltration in various parts, or a condition of carnification. Intense bronchial catarrh was present, extending to the minuter ramifications of the air-tubes, but not of a croupal character (VIRCHOW'S *Arch.*, Vol. XXI., p. 65; *New Syden. Society Year-Book*, 1861, p. 132). In strumous patients measles may end in the development of miliary tubercles in the lungs; increasing cough, emaciation, and a harsh, dry skin being the symptoms of such an untoward result. The catarrhal affection during the desquamative stage forms generally the connecting link with the sequelæ of measles; and the cough often remains for weeks or months after desquamation is over, and grows worse from the most trifling causes. It may depend on simple bronchial catarrh, or on severe disease of the lungs. The nature of that disease, however, is not always tubercle, but more often a caseous transformation and disintegration of the products of lobular pneumonia, with caseous degeneration of the bronchial glands—one of the most common complications of measles. Croup sometimes supervenes, and cuts off young patients. It tends to be of the asthenic type, and is not unfrequently preceded by diphtheritic inflammation of the fauces, which gradually passes down to the larynx. Diarrhœa is another danger to be encountered. During convalescence there is a tendency to looseness of the bowels, but which, if moderate, ought not to be counteracted, as it is commonly rather advantageous; but if suffered to continue, the consequences may be fatal. Catarrhal ophthalmia, otorrhœa, swelling of lymphatic glands, if the constitution be strumous, must also be watched for, and, if possible, prevented. Measles, in any of the malignant forms described, is highly dangerous; and the danger is greater in the old than in the young—in cold than in warm weather.

Treatment.—The constitution during measles is little impaired by the short continuance of the fever. The rule, therefore, is to interfere as little as possible as long as the disease is pursuing its normal course, and merely to moderate and subdue symptoms when they threaten danger, knowing that the great majority of cases end in recovery without any medical treatment. The *morbilli sine catarrho* is usually of such a mild form as to require no other treatment than a milk diet, the customary attention to the bowels, and the prevention of exposure to cold and wet. Measles will not bear exposure of the surface of the body to cold so well as either

scarlatina or small-pox, on account of the great tendency to bronchial and pulmonary inflammation. Children must therefore be watched night and day to prevent them lying uncovered, and special care must be taken to avoid exposure to cold during convalescence. An even and moderately moist temperature must be maintained in the room, steam from a boiling kettle of water being admitted. The temperature ought to be regulated by the thermometer, and kept at 60° to 65° Fahr. The room must also be regularly subjected to a current of fresh air daily, the patient being sufficiently protected at the time.

In the *morbilli mitiores* the cough, the frequent vomiting, and the heavy catarrhal symptoms which so generally attend the primary fever, render medical attendance necessary from the first moment of the attack. The treatment of these symptoms, however, and also of the eruptive stage, as long as the patient continues free from any serious inflammatory affection of the lungs, need not necessarily be active, it being sufficient to alleviate the cough, allay the vomiting, and check the catarrh by some of the large class of saline laxatives, linseed tea, or mucilaginous mixtures, to which *antimonial* or *ipecacuanha wine* may be added, if necessary, as a diaphoretic, and to subdue high vascular action. In making a selection from these, the physician must be principally guided by the state of the bowels and the condition of the stomach of the patient. If the bowels be constipated, the milder salines, as the *sulphate of magnesia*, are to be preferred. Often, however, a diet of stewed prunes or fruit will be sufficient. On the contrary, if the patient be purged, and the vomiting distressing, a neutral mixture or effervescing draught will be found most beneficial. There are many persons in whom the cough and catarrh are the most urgent symptoms; and in such cases, if the stomach be quiet, the *liquor ammoniæ acetatis*, combined with *camphor mixture*, from its more powerful action on the skin, is an excellent addition. Another remedy, equally or perhaps still more useful, is *ipecacuanha wine*. Willan was of opinion that an emetic, given on the second or third evening, *somewhat* alleviated the violence of the catarrhal symptoms, and contributed to prevent the diarrhœa which usually succeeds measles. An emetic is especially useful if the disease be threatened with croup as a complication. During the eruption he adds, "I have not observed any considerable effect from antimonials or other diaphoretics." The use of *aconite* has been found to moisten the skin; and certainly helps the emergence and development of the eruption when due, though it seldom reduces the temperature before the eruption comes out, but it reduces the pulse (PHILLIPS). Bathing the feet every evening seems a beneficial application. Emulsions and mucilages afford but a feeble palliation of the cough and difficulty of breathing; but if there is much oppression, with a sense of tightness, linseed poultice jackets must be applied to the chest. With respect to opiates, they are not advisable should bronchitis be extensive, and in the early stages especially; opium produces an increase of heat and restlessness, without conciliating sleep. The catarrhal symptoms are frequently accompanied, even in the very earliest days of the disease, with much bronchial inflammation, and sometimes with pneumonia; or these affections may occur at any later period, after the decline of the eruption, from the tenth to the twelfth day of the attack. For these a stimulant treatment

is required. If pleurisy alone supervenes, *mercury* is best used in the form of friction with *blue ointment* over the chest, a little *croton oil* being added to promote its absorption (ANDREW ANDERSON). In such cases stimulants are called for—e.g., *subacetate of ammonia*, *chloric ether*, *wine*, or *brandy*.

During the whole course of measles it is necessary to enjoin an abstinence from animal food, and to limit the patient to a low diet and to slops. Liquid food should be given in small quantities at frequent intervals. The chamber should not be subject to any sudden change from heat to cold, and the strictest cleanliness should be observed. With a view to protect the eyes, the room should be kept dark, so that the patient may be prevented reading or using his eyes. In large establishments separation is necessary, to prevent spreading of the disease, if possible. Should the eruption disappear (“*striking in of the eruption*”), or be retarded, and untoward symptoms appear, it is just as unscientific as it is dangerous to attempt its “*restoration*.” It is dangerous, because the rules usually laid down for this emergency may have an injurious effect on the course of the disease. The disappearance of the eruption is not to be regarded as the cause, but as the result of a bad form of the disease, and due to the general collapse of the patient, in which the skin participates. But this bad form of the disease usually depends on the appearance of some complication, especially pneumonia; and, if this fact be not remembered, and the patient be rubbed with irritating applications, or be placed in a hot bath, with or without mustard, or wrapped up in wet blankets with decoction of mustard, “*to bring the eruption out again*,” we shall often do much harm, even if we succeed in our object, because these procedures do not generally act advantageously on the pneumonia and other complications, while they increase the fever” (NIEMEYER, *Text-book of Practical Medicine*). When convulsions occur in children, it is in all cases necessary, in the first instance, to determine the most probable source of the irritation giving rise to the convulsions—i.e., whether they depend upon the specific poison of the disease, upon dentition, or upon intestinal irritation or cerebral disorder. Hot foot-baths sometimes give relief, as well as sinapisms to the limbs; after which, if the convulsions do not subside, it may be necessary that blood be taken by leeches from the temples. Diarrhoea should not be checked suddenly, but kept under control. The patient ought to remain in bed so long as fever is present, while desquamation is going on, and the cough is severe; and he ought to keep his room so long as catarrh is present. A low state of the general health may finally result, requiring *quinine*, *iron*, or *cod-liver oil*; change of air—especially to the sea-side—and sea-bathing, or cold bathing in salt water.

SCARLET FEVER—*Syn.*, SCARLATINA.*

LATIN EQ., *Febris Rubra*; FRENCH EQ., *Scarlatine*; GERMAN EQ., *Scharlachfieber*; ITALIAN EQ., *Febbre Scarlattina*.

Definition.—*A specific febrile disease, the product of a specific contagium, which is reproduced during the progress of the affection. The duration of the incubation stage may be only a few hours; in a large proportion of cases it probably does not exceed forty-eight hours, and very rarely exceeds seven days (MURCHISON). On the second day of the illness, or sometimes later, a scarlet efflorescence appears on the fauces and pharynx, and on the neck and face, which spreads over the whole body, and commonly terminates in desquamation from the fifth to the seventh day. The fever is accompanied with an affection of the kidneys, with albuminuria, often with severe disease of the throat, or lymphatic glands of the neck, and is sometimes followed by dropsy and other lesions. The disease runs a definite course, and, as a rule, occurs only once during life.*

Pathology.—The origin and nature of the scarlet fever poison are unknown; but its ordinary sources, the way in which it is propagated and the means by which it may be destroyed, are known with certainty. It is unquestionably contained in everything that proceeds from the body of a patient labouring under the disease. It is specially contained in the skin, which peels off at the close of the attack, and which is scattered to the winds in the form of tiny scales, or as absolute powder. Such portions of dried skin may settle down in undusted corners, or may become entangled in clothing or bedding, in either case preserving their virulence for an unknown period of time, always liable to be disturbed from their resting-places, and thus going forth to reproduce the fever out of which they sprung. The earliest source of the poison is distinctly traceable to Arabia; and the disease has now spread over the whole world. It prevails at all seasons of the year, is always in existence somewhere, and often epidemic. It has been found to spread more extensively, and with greater fatality, among the poorer than among the wealthier classes of society; and both sexes are attacked in nearly equal proportions. All ages are liable to the disease, but it is most common in childhood, most frequently in the third and fourth years of life; and as children grow older, the less

* A considerable number of synonyms has been given by the College of Physicians in their Nomenclature, not with the view of sanctioning their employment, but that, in the registration of disease, whenever they occur, they are always to be rendered into the corresponding term sanctioned by the College. The Committee had a twofold object in mentioning these synonyms: *First*, There are several diseases which are commonly designated by an objectionable name, and when a preferable term could be found, the other has been given as a synonym to indicate that exactly the same disease was meant by the new name. A good instance of such a change is found in the substitution of "enteric" for "typhoid fever." *Secondly*, The enumeration of synonyms in common use seemed likely, in many cases, to give precision to the term adopted by the Committee, and in many instances served to identify it, and obviated the necessity of giving a definition—a matter always of considerable difficulty, and sometimes almost impossible in the compass of a few words, such as seemed suitable for a nomenclature of disease. As an instance of such an employment of synonyms may be cited the subdivisions of Bright's disease, where a definition of the generic name having been given to avoid misapprehension, the simple addition of the names applied by different observers to each form of the disease seemed quite sufficient for the purposes of identification.—*Med. Chir. Review*, Oct., 1869, p. 364.

liable are they to be attacked. The chances of attack decline rapidly after the fifth year (Dr. B. W. RICHARDSON). The seasons also seem to influence its prevalence and intensity. The months of October, November, and December, furnish, in England, the maximum amount of the disease—the months of April, May, and June, the minimum.

The specific fever being established, the patient generates a poison, which may be communicated also directly, as shown by cases of successful inoculation—*e.g.*, Miquel's (*Lancet*, Aug. 13, 1864). The disease is otherwise eminently communicable, so that no susceptible person can remain in the same room, and hardly in the same house, without contracting it, unless special care be taken to isolate the sick. That scarlatina is capable of being directly communicated is shown by the fact that children have been inoculated with the serum found in the vesicles which sometimes accompany the rash, and have taken the disease; but the inoculated disease not having proved milder than the natural, the practice was properly abandoned. Another proof of the directly communicable nature of scarlatina is, that it has often been propagated by *fomites*, as by the clothes and boxes of boys returning from school. Susceptible persons also sleeping in a room lately occupied by patients labouring under scarlatina, and before the furniture has been washed and the bedding and walls well ventilated, have thus become infected. The virus is destroyed by heat at the boiling point, or it may be disinfected artificially, as by the fumes of *nitrous acid*. A succession of cases of scarlet fever occurred in a college connected with the University of St. Andrews several years ago. Servants were the first sufferers, then students. Some of the latter went away, carrying contagion with them, and occasioning fresh outbreaks; but the succession of cases in the college, where isolation and disinfection had been practised, was at first very perplexing. Scattered cases occurred in the town also, in families having no communication either with the college or with one another. At last it was found that the infection had been brought from the farm that supplied the several habitations with milk. This farm was a small one. The farmer's wife, with one child, visited a distant place, where they were exposed to scarlet fever, and brought it home with them. The wife appears to have suffered very slightly. She nursed the child and milked the cows. A second child took the disease, and continued the infection. The boy who carried out the milk was next infected; and in him the fever was so slight that it never prevented him from accomplishing his daily round. On making out a list of the houses in which scarlet fever showed itself in St. Andrews, and on asking the farmer for a list of those to which he sent milk, the two were found to be identical. With one exception, the inmates of every house supplied with the milk had been attacked by the disease, and twenty-six cases and two deaths were directly traceable to this centre of contagion. In the same communication, Professor Bell mentions having seen a child with scarlet fever in a small crowded room. Upon inquiry he found the following facts:—"The father had charge of an extensive society's bread shop; the mother was a washerwoman, taking in clothes to her house to wash; the eldest girl attended, throughout the day, the children of a lady's family, and came home to sleep at night; the other children attended, some an infant school, some a large mixed school, where hundreds of other children met.

The youngest played with young children in a house on the opposite side of the passage." Among the most common sources of the spread of scarlet fever may now be reckoned the movements of the dwellers in infected houses, the dispersion of schools in which the disease has appeared, and the unchecked going to and fro of the friends of the sick. There are facts which render it probable that the material of contagion is carried by persons not themselves infected by the disease, and also through the medium of animals. It is of extraordinary tenacity.

Out of 2,000 cases that Dr. Willan attended, he witnessed no instance of a *second attack*. Still there are some exceptions to the statement that an attack of scarlatina gives an immunity from a second attack. Dr. Binns has seen instances of scarlet fever occurring twice in the same person; while Sir Gilbert Blane met with an instance of its occurring thrice in a young lady, without the least suspicion of ambiguity or possibility of mistake in diagnosis. Dr. B. W. Richardson shows that it may recur once or even twice in the same person. But these events are rare; and death from a second attack is unknown as a fact. Scarlet fever has often co-existed with the vaccine disease, with erysipelas, and with diphtheria. The poison of scarlet fever is absorbed by the mucous membranes, and (as is evident from the fact of inoculation) through the skin. Children have been born with the disease.

The period of latency varies from a few hours to seven days. In the *Lancet* for August 13, 1864, the late Dr. Murchison published thirteen cases illustrating the incubation-period of scarlet fever; and came to the conclusion that while in rare instances there was no appreciable incubation-period, it varied in most cases from one to five days; and very rarely exceeded six days. He has since then collected all the cases illustrating this point, seventy-five in number, with some very interesting general results. When a large number of persons became infected from a common source (at a dinner party, and on shipboard) the incubation-period was in every instance short. In all the seventy-five cases it was possible to fix the *maximum limit* of the latent period, but in three only of the cases could the moment of infection be fixed with precision—they gave thirty-six hours, three days, and four and a half days respectively. His investigations into this matter lends no support to the opinion expressed by some writers to the effect that the latent period of scarlet fever is longer in adults than in children; nor that scarlet fever differs from all the other acute exanthemata in the great variation of its incubative stage; nor does it give any countenance to the common argument that the disease has not been transmitted by a nurse or the medical attendant, because of the short interval that has elapsed between their first communication with the patient and the appearance of the disease: such an argument is shown to be untenable. His observations have led him to the following practical conclusions:—(1.) The duration of the incubative stage may be only a few hours; (2.) In a large proportion of cases, it probably does not exceed forty-eight hours; (3.) It very rarely exceeds seven days; (4.) Consequently a person who has been exposed to scarlet fever, and does not sicken after a week's quarantine, may be pronounced safe,—provided their clothing has been properly disinfected. Many facts show that scarlet fever has an infecting power from the earliest stages

of the malady; although the comparative facility of preventing its spread in a school when the first case is properly isolated, points to this power being much less during the first two or three days than in the case of measles. On the other hand, scarlatina has the power of infecting over a period of many weeks; and the ordinary practice of pronouncing a patient ineffective as an agent of propagation at the end of a month, or after the apparent cessation of desquamation, is not always safe; in proof of which he quotes a case where a patient, convalescent, imparted the disease after the fortieth day, and after desquamation had to all appearance quite terminated. It is therefore a good rule to regard no case as safe from being a centre of infection until after the expiration of the eighth week (MURCHISON, *Trans. Clin. Soc.*, Vol. XI, 1878). In one case *inoculated* by Rostan the eruption appeared on the seventh day after inoculation, the incubation-period being therefore probably five or six days. In Miquel's experiments the incubation-period was only thirty hours; and the specific poison is probably capable of communication from the patient to others as soon as the primary fever has formed; for there are no facts on record which prove that any acute specific disease can be transmitted during the incubation stage, either by mere contact or through the atmosphere.

After a limited period of latency or of incubation, the peculiar poison of scarlet fever induces a disorder of the blood, which is, in the first instance, made manifest by a specific *febrile state*, and a *disturbed condition of the great nervous centres*. This primary fever having lasted for one, two, or three days, does not entirely subside, but persists while a peculiar eruption develops itself preceded, followed, or accompanied by a sore throat, and also by intense hyperæmia and catarrh of the uriniferous tubules. The eruption runs a course of from six to eight days, but the duration of the affection of the throat is more indefinite, and varies from eight to twenty or more days. The fever continues during the eruption, and as long as the sore throat exists; after which it subsides, and convalescence ought then to advance. In a few instances, however, remote results succeed, as dropsy or inflammation of the joints, pleura, pericardium, ear, or cornea, lesions quite as formidable as any which had preceded them, and each or all of them localised expressions of the influence of the scarlet fever poison on the body. Scarlet fever thus essentially consists of a specific fever and certain local inflammations; but among its more striking phenomena is the sudden and remarkable depression of the nervous and physical powers of the body which its poison produces—a depression so great as sometimes to cause death in a few hours, without any reaction, or any very sensible local lesion of the throat or other part being discoverable after death. Death has been known to take place in six hours from the commencement of the disease—the child, in fact, dying poisoned (Dr. ANDREW ANDERSON, *On Fever*, p. 77). In such cases the patients seem to die of paralysis of the heart, preceded by symptoms of excessive adynamia (NIEMEYER). The question is therefore suggested, as in measles and diseases of this specific kind:—Has the poison through the blood a directly paralyzing effect on the nervous system, and especially on the nerves of the heart? or, Is the injurious effect due to the excessive increase of the heat of the blood alone—sufficient of itself to kill, or to induce the

paralysis? In suddenness of danger scarlet fever thus approaches yellow fever and cholera. The eruption presents several forms, all evanescent after death. In the smooth eruption the surface of the inflamed skin presents no inequality either to the sight or touch. The *scarlatina papulosa* has an eruption in which the papillæ of the skin are enlarged by inflammatory exudations at circumscribed spots, and the appearance is that of roughness (*goose-skinned*). The third form is when the eruption is accompanied by numbers of vesicles of various size, filled with serum, or with limpid or cloudy yellow liquid, which ultimately shrivel up and desquamate. From this form inoculation may be effected. Whatever the ultimate form of the eruption may be, its first appearance is by innumerable small bright-red puncta, dots, or maculæ, separated by interstices of healthy skin. These puncta or maculæ are at first very minute points all over the affected parts, which are usually more or less rough to the touch; but they quickly become confluent in patches of greater or less extent, and in a few hours the redness becomes general over the parts attacked. The anatomical change in the skin is that of extensive erythematous inflammation, consisting of intense hyperæmia with an inflammatory œdema of the superficial layers of the cuticle. The colour, in ordinary cases, is in the first instance a bright red, like that of a boiled lobster, but on the decline of the disease it becomes deeper, and more resembles that of beet-root, while in severe cases it is of a pink blush rather than a scarlet efflorescence, or it may be livid, and intermixed with petechiæ. But whatever tint the eruption may assume, it has this peculiarity, that it disappears on pressure, and again returns from the periphery to the centre on that pressure being removed. The colour is also always brighter and more vivid in the flexure of the joints, and about the hips and loins, than over the rest of the body. A sign of scarlatina, in connection with the eruption, has been described by Bouchut, as pathognomonic. It consists in an enduring white stripe, produced by pressure with any hard substance on the skin occupied by the eruption. This phenomenon is ascribed to an increase of the contractile power of the capillaries, which is proportionate to the intensity of the disease, the regularity of the eruption, and the amount of vital power (*Syden. Society Year-Book*, 1861, p. 130). As in the malignant cases of the previously described exanthematic fevers, so in scarlet fever, the hyperæmia of the skin is occasionally accompanied by more or less extensive hæmorrhages, in the form of petechiæ and extensive ecchymoses, a most unfavourable sign. The termination of this specific inflammation of the skin is generally by desquamation of the cuticle, which begins with the decline of the eruption, and is usually completed by the end of the second week, unless it is longer delayed by successive crops of eruption, and consequent succession of exfoliations. There is no fever in which this phenomenon of desquamation is more conspicuous. A few days after its commencement albumen may be detected in the urine in small quantity, which continues to be given off for several days, along with a considerable amount of epithelium, resulting from the hyperæmia and catarrh of the uriniferous tubules (Dr. J. W. BEGBIE). There are also cases in which albuminuria is associated with *anasarca*; and three stages have been recognised in which this complication occurs:—(1.) A febrile stage, characterised by

fever of an intermittent character, and by rapid serous extravasation and infiltration. (2.) A chronic stage, in which the affection follows a slowly progressive course. (3.) A period of resolution (HAMBURGER). Such cases sometimes terminate by uræmic symptoms and convulsions. An unusual case of this nature has been recently recorded by Biermer. It happened with a boy five years and a half old, and ended fatally on the thirty-fifth day. No urine was passed for 108 hours between the twenty-first and the twenty-sixth days of the disease, and extremely little for five days more. Yet during these ten days there were no uræmic symptoms, nor any notable dropsy. The uræmic symptoms first set in after the urine began to be secreted freely, when it was but slightly albuminous (*Syden. Society Year-Book*, 1861, p. 218). Occasionally the *squamæ* of the cuticle are so large as to preserve entire the whole epidermis of the palms of the hands and of the soles of the feet. Frank has seen the cuticular *squamæ* come away with the hair, nails, and even with *veruæ* attached. Frequently, however, the material of desquamation is furfuraceous or scaly. In a few instances the termination is by ulceration and sloughing of large portions of the integument.

Symptoms.—Although several varieties of scarlatina are described by authors, they are not equally distinctly defined in nature. Yet it not unfrequently happens that the characters of each variety are tolerably well marked in typical cases. The following varieties have been distinguished, namely:—(a.) “*Simple Scarlet Fever.*” *Definition*: “A scarlet rash, with redness of the throat, but without ulceration.” (b.) “*Anginose Scarlet Fever.*” *Definition*: “A more severe form of the disease, with redness and ulceration of the throat, and a tendency to the formation of abscess in the neck.” (c.) “*Malignant Scarlet Fever.*” *Definition*: “The throat tends to slough; the scarlet rash is scarcely, if at all, visible; petechiæ are often seen on the surface, and the fever is of a low form.” To this definition a note is added by the College of Physicians, to the effect that “scarlet fever occurs occasionally without any rash or sore throat being observed.” If such cases could be always recognised, they ought to furnish a fourth variety—namely, *Latent Scarlet Fever*.

The symptoms of a typical case of scarlet fever under ordinary circumstances may be divided into three stages. The first stage occupies the period from the commencement of the disease (including a latent period of incubation) till the appearance of the eruption. Sometimes during this stage fever begins with its usual symptoms, which is technically termed the “*primary fever*”; the second stage, or period of eruption, that from the appearance of the eruption till its entire subsidence; while the third, or that of desquamation, is reckoned from the disappearance of the eruption, when the symptoms begin to subside, till the termination of the disease. The duration of the first stage is one, two, or three days; that of the second, from six to eight days; while the third stage may vary from a few hours to two or three weeks,—making the whole duration of the fever from eight to thirty or more days.

Simple Scarlet Fever is a name which has been given to those cases where the fever maintains an inflammatory type, and where the perceptible localisations of the disease are expressed by the scarlet rash,

severe catarrhal sore-throat, with more or less redness of the throat without ulceration; and with catarrh of the kidneys; and simple though this variety may appear to be, it is a most serious affection. It has been also named *Scarlatina mitis* and *Scarlatina sine angina*; but these names ought not to be longer in use. The primary fever is usually sudden and distinct in its attack; or the patient may complain for some days of weariness and depression, or a feeling of sickness or of slight indisposition during the period of incubation. The early symptoms, whatever be the variety, are headache, pains in the back and loins, loss of appetite, sickness, and white tongue. The disease is, indeed, usually ushered in by vomiting—sometimes very obstinate and troublesome, with much thirst and total loss of appetite. In slight cases vomiting may be the only noticeable symptom. Still there are symptoms which distinguish it from other continued fevers; for the pulse, instead of being full and strong, is small, and weak, and rapid—120 to 130 beats a minute; the heat of the skin *rapidly* becomes more ardent—104° to 105° Fahr., or higher; and with such ranges of temperature, these phenomena continue through the whole course of the disease. The fever, however, varies greatly in intensity, in height of temperature, and in duration,—the characteristics being a rapid ascent, a very high maximum, and an interrupted defervescence. The symptoms, on the other hand, may be extremely mild, so that the patient is frequently not confined to bed. The primary fever (except that the pulse is rapid) is little more than a mere febricula, and is not aggravated on the appearance of the eruption. With the disappearance of the rash the fever of this simple variety ceases, and the disease terminates; but it often leaves the patient in a state of considerable debility, and may be followed by albuminuria.

Whatever may be the colour or description of the eruption, it does not attack all parts of the body simultaneously, but appears partially, or in a succession of crops, the order of which may be stated as follows:—On the first or second day of the primary fever it appears and spreads universally over the face, neck, and upper extremities; on the following day over the trunk, but is less general on the back than on the abdomen; and, lastly, on the third day, it has extended itself over the lower extremities. The duration of each crop is about three days, when it disappears in the order of attack, fading from the head and upper extremities on the fourth day, from the trunk on the fifth day, and from the lower extremities from the sixth to the eighth day. It has generally disappeared before the ninth or tenth day, when desquamation begins. The order of attack, however, which has been mentioned is not constant. In some few instances the eruption appears first on the trunk and lower extremities; and only on the second day very faintly on the face and upper extremities. The patches of eruption are generally very distinct over the flexures of the joints. The colour of the rash is more marked in the centre of each spot. It disappears completely on pressure, but soon returns again on the removal of pressure; and is always more florid during the night than in the day. The period of eruption usually commences with an increase of the fever heat, while at the same time the general symptoms increase, such as headache, weakness, restlessness; and convulsions are apt to occur in children. These early and

severe symptoms are almost pathognomonic, as expressed by the fever and the pulse, such as are not often observed at the commencement of other diseases. If sore throat commences, there can be no longer any doubt. The fauces become red and dry, the neck stiff, and tenderness is felt about the jaws. The disease attains its height, and the fever maintains its course, usually from the fifth to the ninth day, when, in favourable cases, a continuous but more often an interrupted defervescence sets in, and all the symptoms begin to decline. The fever does not subside on the appearance of the rash, as is the case with small-pox, but continues, with various degrees of violence and ranges of temperature, until the eruption is fully developed; the maximum of temperature usually coinciding with the maximum of eruption. The pulse is often 120 to 130 or 160 in a minute, and sometimes beats with considerable force. The skin frequently indicates a temperature of 105° , 106° , or even 112° Fahr. without a fatal issue; and it is dry, with a sensation of burning heat, till about the third day, when the maximum of temperature is attained. From the third to the ninth day the range is maintained, with slight morning remissions, between 103.8° and 102.9° Fahr., and begins to subside about the tenth or twelfth day, after which defervescence ought to be continuous. The pulse comes down as the temperature lowers. The difference in these respects between scarlatina and measles may be appreciated at once by a glance at the account and the diagram given of measles, and comparing it with the following diagram, which shows the typical range of temperature in *Scarlet Fever*. The temperature may be higher in this fever than in any other without a fatal issue; and is sometimes extremely high where the eruption is deficient or absent.

There is no remarkable increase of fever heat preceding complete defervescence; and after the *exanthema* has reached its maximum, the decrease of temperature proceeds by no means rapidly. The commencement of the decrease may be marked by a few decided and rapid falls; but its further fall is decidedly lingering, and is even sometimes interrupted by small increases of temperature (like enteric fever, but with less exacerbation towards evening), so that the whole process of defervescence occupies, as a rule, from five to eight days. It is only in very mild or anomalous cases that the temperature rarely exceeds 101.8° Fahr. These cases sometimes show a rapid defervescence, completed in a single night. It is essentially a short fever, the ranges of temperature forming cycles composed of a variable number of days, generally of five; a fall of temperature taking place on the fifth, tenth, or fifteenth day of the disease (RINGER, *Med.-Chir. Society Trans.*, 28th January, 1862).

The poison of scarlatina frequently expresses itself on the mucous membranes of the eyes and nasal fossæ, and excites a similar eruption over those parts as on the skin, at first consisting of a distinct punctated or dotted appearance, which changes in a few hours to one of diffuse redness. The inflammation of the ocular membrane, however, does not distress the sight, for the eye bears light without inconvenience. The lingual and buccal mucous membranes are also often the seat of a similar exanthem. The papillæ of the tongue are singularly elongated and enlarged. They stand up salient and erect, of a deep scarlet colour, above the thick, white, creamy mucous fur which coats its surface; hence the term

TYPICAL RANGE OF TEMPERATURE IN A CASE OF SCARLET FEVER. THE RECORDS INDICATE MORNING (A.M.) AND EVENING (P.M.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE SECOND DAY (Wunderlich).

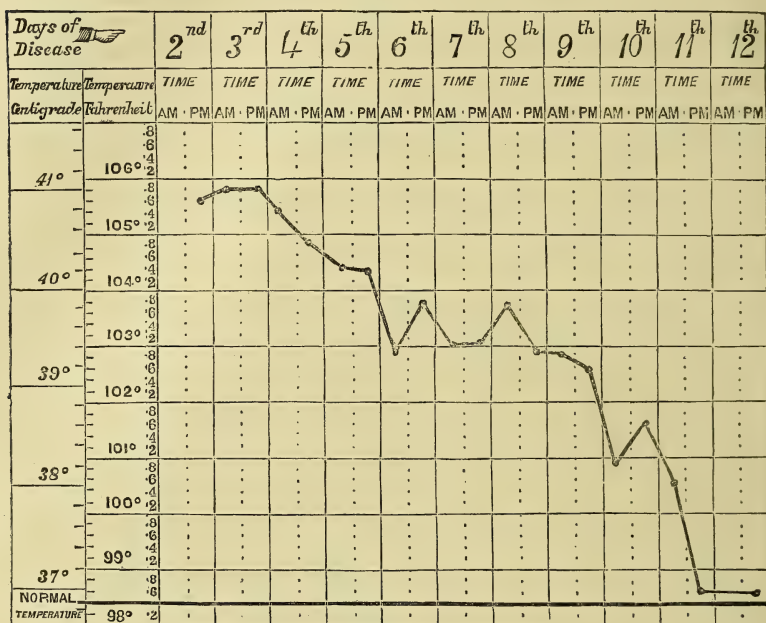


Fig. 83.

"strawberry tongue," from its supposed resemblance to the exterior of a strawberry, or "cat's tongue," from its resemblance to the rough surface of the tongue of the feline tribe. Its tip is of a vivid red, also through development of the papillæ. By and by the fur falls off, and the whole dorsum of the tongue is then left clean, red, and raw-looking. The sore throat is often a grave disease. It may either precede the other symptoms, or may occur at any period of the fever. The inflammation is at first punctated, then a diffused redness of the fauces with cedema and swelling. The mucous surface is dry or covered with viscid mucous, and a thick opaque secretion is often seen on the tonsils. Ulceration may supervene, or the tonsils may suppurate; and the character of the ulcer is so completely in unison with the state of the constitution as to enable us, according as it is slight or severe, to divide scarlatina into two great varieties—namely, into the "*anginose*" and the "*malignant*." The first, or sthenic form, is marked by a greatly enlarged or swollen state of the tonsils, which are of a vivid or bright-red colour; and, when ulceration takes place, the ulcers are seldom deep, or the sloughs slow to come away, but usually they separate about the fifth or sixth day. In mild cases the sore throat is healed about the eighth or tenth day, and in more severe ones about the fifteenth or twentieth. In malignant cases, the tonsils are much less tumefied and

enlarged, but much more loaded with blood, and of a deeper, and sometimes of a livid colour. The ulcers, also, are deep and formidable, and the sloughs are thrown off later in the disease. They are slow to heal,—not till the end of three weeks, and in severe cases not till four or even six weeks have elapsed, during which period the fever continues, and the patient remains in considerable danger. Subjective symptoms are therefore usually well marked—namely, soreness with pain and difficulty of swallowing. The inflammation is not limited to the tonsils, but extends over the fauces generally and uniformly, or symmetrically on either side, as is common with blood diseases. The glands about the angles of the jaws become enlarged and painful, while the subcutaneous tissue is œdematous and puffy. It may extend to all the neighbouring parts, and an abscess may form in the pharynx, or pus may even issue from the ears. The tympanum, in some cases, has been eroded, and in a few instances the inflammation has extended to the larynx, and the patient has died from œdema glottidis or membranous formation in the larynx of a diphtheritic nature. Sometimes the glands in the neck do not become affected till after the sore throat has healed; and sometimes they become affected when there has been no previous sore throat, as if these parts were the seat of a specific action of the poison. Glandular swellings on both sides of the neck are not unfrequent, and have been described as “the scarlatinal bubo” (TROUSSEAU). They may be the best and most accurate index of danger in the later period of scarlet fever, in so far as the danger depends on the sore throat, and on the putrid infection of the blood (septicæmia) which accompanies it (W. T. GAIRDNER, *Clinical Medicine*, p. 193). There is then great difficulty in opening the mouth and examining the throat, which is painful; and the breath is extremely offensive and disagreeable. Much distress is experienced from fluids entering the posterior nares during the act of swallowing. Some œdema of the submucous or subcutaneous aerolar tissue takes place as soon as the rash appears, causing the hands to swell, so that the patient is unable to bend his fingers, and his face becomes tumefied and painful. In mild cases, however, the serum effused is absorbed, and the disease terminates without any unpleasant consequences; in severe cases it has a tendency to terminate in ulceration or in mortification. In children the toes of one foot have been known to slough off; and in some the integuments of the leg have mortified from the knee to the foot; while in others mortification, commencing in the upper lip, has been known to spread till one-half the cheek has been eaten away. Some have been known to die of mortification of the rectum, and others of a similar affection of the pudenda.

Such are the primary and secondary affections of scarlet fever; but this poison has also more remote actions, giving rise to dropsy, as well as affecting the synovial membranes of the joints, the serous membranes of the pleura and pericardium. The dropsy which sometimes occurs after scarlet fever usually commences between the fifteenth and twenty-third days of the disease, and, almost uniformly, not till after all the other symptoms have subsided. The patient is especially liable to it during desquamation, and for a considerable time afterwards during convalescence. It is indicated by pallor, and begins with œdema of the face, and afterwards of the hands and feet. In some instances there is general anasarca, the whole

areolar tissue of the body filling with fluid so rapidly as sometimes to destroy the patient in a few hours, the cavities of the chest and abdomen frequently filling at the same time. According to extensive observation it has occurred more frequently after mild than severe cases. Its forms are *anasarca*, *ascites*, *hydrothorax*, *hydro-pericardium*, and even *hydrocephalus*, which is commonly preceded by the usual hydrocephalic headache, or by convulsions, and sometimes by blindness. During the progress of the fluid effusion, oedema of the glottis must be watched for and relieved. The dropsy is generally accompanied with scanty and albuminous urine; and although the presence of albumen, without diminished secretion, is almost a regular phenomenon in the course of the disease, independent of dropsy, yet, if the urine becomes highly albuminous and diminished in quantity, the dropsical complications may be apprehended (*Edin. Med. Journal*, Jan., 1849, and Oct., 1852). More or less congestion of the kidneys occurs in every case of scarlet fever, although, like the sore throat, it may often be so slight as not to give rise to any prominent symptom (BEGGIE, ANDERSON). The scarlatinal dropsy, however, is very generally considered as most intimately connected with the kidney disease; and when the nephritis is well marked, the characters of the urine exactly resemble those in acute Bright's disease (PARKES). The symptoms are indeed similar to those of acute Bright's disease, the poison of scarlet fever being one of its chief causes. On the other hand, there is also evidence in favour of the opinion that albuminuria may be wanting in scarlatinal dropsy (see PARKES, *On the Urine*, p. 264). *The condition of the urine* in scarlet fever ought to be ascertained daily in every case, especially during the period of convalescence. "It is of more importance," writes Dr. Andrew Anderson, "that you should examine the urine, than that you should feel the pulse of a convalescent from scarlet fever." The urine has the ordinary febrile characters. During the first six days the amount is small; the urea and uric acid are increased in amount, and sediments of urates occur. On the other hand, "urea is certainly not increased in the pyrexial period of scarlatina; but is diminished after the first few days of the disease, bearing no relation at all to the temperature" (Dr. EDWARD LONG FOX). The chlorine is sometimes greatly lessened, and augments during convalescence. On the sixth to the eighth day, if the case goes on well, the urine becomes abundant, pale, and the reaction neutral or feebly acid. There is bile-pigment present during the first six days; and in a large proportion of cases, though not in all, the urine becomes albuminous. Albumen is present at some period in almost every case (Dr. WARBURTON BEGGIE). It is usually associated with a large amount of renal, pelvic, and bladder epithelium, but not with renal cylinders unless there be dropsy. The albuminuria occurring during desquamation is usually transient; but it may continue till an attack of dropsy occurs—disappearing and reappearing when dropsy comes on a fortnight or three weeks later. A rise of temperature, even to the maximum point of the original fever, marks the progress of *acute tubal nephritis*. The urine is diminished or suppressed; highly concentrated, with a smoky colour, and may contain more or less blood-corpuscles, renal epithelium and casts (epithelial, bloody, or granular), and highly albuminous (*croupous nephritis*). In malignant scarlatina, as in malignant

variola, there may be considerable hæmaturia or passage of dissolved hæmaturin (PARKES, *On the Urine*, p. 263).

Intercurrent inflammations of the synovial membranes have been described by Withering, Sennertus, Heberden, and others, in the wrist, ankle, or knee-joints, which usually terminate by effusion of serum. In some cases the cavities of the joints contain pus. Such inflammations seldom occur till after the eruption has subsided, and are more often set up after mild than after severe cases. The attack resembles one of acute rheumatism. Thus the fever is maintained, and the duration of the illness is prolonged many days beyond the usual period.

Such are the morbid phenomena which have been observed in typical cases of scarlet fever, with sufficient constancy to mark the disease as due to a specific poison. In severe and rapid cases the patient may die, not from any organic lesion, but from the intensity of the shock, in the first instance, on the nervous system. Bretonneau, Tweedie, and Sims, all speak of having examined the bodies of persons who have died early in the disease, in which there were scarcely any appreciable lesion—coma, or other violent cerebral affection, carrying off the patient.

Anginose Scarlet Fever.—In this form of the disease the specific action of the poison is mainly limited to one region. It is “a more severe form of the disease than the *simple* variety, with redness and ulceration of the throat, and a tendency to the formation of abscesses in the neck.” The eruption on the skin may be altogether wanting, or appearing at a later period than usual, generally by one day; as a general rule, it is less copious and less diffuse than in the other forms, and is disposed to fade and return again. This form is only to be distinguished from simple sore throat from cold by its occurrence during an epidemic, by the high fever, quick pulse, and great constitutional disturbance at an early period of the stage of incubation. The symptoms of the *anginose variety* are more violent than in *Scarlatina simplex*. Nausea or vomiting, great restlessness, headache, and some delirium, frequently occur as early as the second day. The heat of the skin is more considerable, and often raises the thermometer as high as 105°, while the pulse is quick, feeble, and fluttering, and shows the extreme debility the poison has occasioned. The eruption may run the course which has been described in simple scarlet fever, but its colour is more intense, its duration more variable, and its attack more partial. The *angina*, so marked a symptom in this affection, may precede the primary fever, may commence with the eruption, or may occur at some later day in the disease. It has many grades of severity. Thus, in slight cases the throat has merely the sensation of heat, dryness, and roughness, with some pain, which is increased by deglutition; and then the mucous membrane of the tonsils and soft palate is seen to be dark-red and swollen. In other cases, the tonsils are enlarged and ulcerated; while in cases of still greater severity they are swollen to a degree almost to occlude the fauces. In this latter case the act of deglutition is not merely painful, but in many instances impossible, and is impeded by a thick viscid mucus, which frequently requires the effort of vomiting to remove. The irritation of the fauces is sometimes propagated to the larynx, and the patient's voice is hoarse or inaudible, and perhaps he may ultimately die from this new affection. The parotid and submaxillary glands often enlarge, sometimes previously

to the sore throat, more commonly about the fifth day, and again after the sore throat has healed. When the constitutional disturbance is very great, the sore throat may be of a diphtheritic character, attended with an irregularly developed eruption, pale or livid, remaining expressed only for a short time, and accompanied by petechiæ, which continue after the eruption has disappeared. The fever continues high, the pulse weaker, and prostration greater. Such cases are generally attended with so-called "typhoid symptoms," the tongue, gums, and teeth covered with *sordes*. The degree of fever is usually proportioned to the severity of the angina, and is accompanied by headache, and sometimes by delirium. The temperature does not abate on the appearance of the eruption, but continues high after the rash has gone till the throat is healed. If the sloughs come away early, or on the fourth or fifth day, the throat heals, and the fever perhaps subsides within a day or two after the eruption. It sometimes happens, however, that the sloughs do not separate till the fourteenth or fifteenth day; and in this case the fever runs on with equal violence after the disappearance of the eruption, and the whole disease is sometimes prolonged for three weeks or a month. In this case the tongue may become brown or dry. The general symptoms are prone to be of a low type; and when adynamia is marked from the first, the variety is sometimes named:—

Malignant Scarlet Fever.—In the more severe forms of the *scarlatina anginosa* (which have been described by some authors as the "*scarlatina maligna vel gravior*, or *malignant scarlet fever*") the specific actions of the poison are the same as in the milder forms; but the symptoms, both local and general, are more severe, and the tertiary affections more frequent, and consequently, the disease is more grave and the danger more formidable. There is extreme prostration from the first, with intense nervous depression; and in this severer form the tonsils and uvula, though less swollen, are more gorged with blood, more livid in colour, while the ulcers are foul, deep, and burrowing: the secretions of the mouth are more copious, and generally impregnated with the offensive *sordes* of the sloughs; while deglutition, if less difficult, is perhaps infinitely more painful, and the mouth often so tender that the slightest touch excoriates it. The ulcers are slow to granulate, and only heal after a tedious treatment. In the worst cases they spread in every direction, the parts tending to vesicate, and even to spreading gangrene previous to the death of the patient, which is almost certain to happen. This form is that which is known as the "malignant sore throat," or "putrid sore throat" of some authors; and is the name now generally applied to certain cases of extreme severity, into which some of the forms already described may pass, as if by insensible gradations. The throat tends to slough; the scarlet rash is scarcely, if at all, visible; petechiæ are often seen on the surface, and the fever is of a low form." The violence of the attack is in some cases so sudden that the patient is at once struck down by the force or virulence of the poison, the type being at once septic, adynamic, typhoid, and malignant. The extreme severity of the constitutional symptoms is marked by the smallness, feebleness, and irregularity of the pulse; the respirations are oppressed, short, and quick; the child becomes faint, sick, pale, and cold, almost to complete collapse;

raving, stupor, and sometimes coma, appear early, alternating with fretfulness, violence, dulness, and suffusion of the eyes, flushing of the cheeks, and dark-brown furred tongue. The circulation being impeded, there is duskiess of the face, and capillary congestion in the dependent parts. The rash appears late, and is of uncertain duration, and soon assumes a dark or livid colour, or disappears in a few hours, reappearing again after several days, if life is so far prolonged. Aphthous elevations in the throat, surrounded by a livid base, become dark; and, bursting, they expose a surface of an excoriated, grey, or dark and gangrenous appearance. The passages of the fauces are always clogged up with much viscid mucus or phlegm, which produces a rattling noise in breathing, and increases the pain and difficulty of swallowing. The discharges, often sanious, are remarkably acrid and offensive, which issue from the nostrils and posterior nasal passages, causing soreness, excoriations, and even blisters on the surfaces and orifices over which, or through which, they flow. To this source the diarrhœa may be ascribed, which is sometimes severe at this period, and generally adds greatly to the sufferings of the patient. Such malignant sore throat not unfrequently occurs during what seems at first a simple or benign case of scarlet fever, which may not at first seem dangerous to life. But, insidiously, a diphtheritic inflammation may be seen on the throat, showing itself by dirty-white and firmly-adherent spots or points on the dark-red mucous surface. It soon spreads to the nasal cavities, whence there flows a discharge, at first of an apparently bland fluid, but which soon becomes fœtid. The dirty-white patches begin to separate as partially diffuent sloughs, which, being imperfectly detached, leave a grey unhealthy ulcer. The secretion flowing from nose and mouth then assumes a yellowish tinge, and is very fœtid, and seems to corrode the skin of the cheek over which it flows. The lymphatic glands of the neck now become "scarlatinal buboes," forming hard lumps on each side of the neck. In such cases the pulse tends to become small and rapid, 140 to 160 per minute, and with a high temperature, 105° to 106° Fahr. *The eruption* offers some peculiarities, being often later by some hours in coming out, its colour darker and more livid, its duration more uncertain, and its distribution more irregular and capricious than in the milder forms. The primary fever, likewise, is usually longer, the delirium earlier, and the depression more complete than in the milder forms; and towards the close of the disease the tongue becomes brown, and the symptoms closely resemble those of the last stage of typhus fever.

Such are the more marked characters of the severer form of scarlatina; but it often happens that the progress of this disease (unless the range of temperature is regularly and continuously recorded) is silent, slow, insidious, scarcely marked by any prominent symptom, till the degree in which the constitution is subdued by the poison is shown by the inflamed nasal membrane discharging its fœtid ichor, causing mortification of the alæ of the nose, or mortification of the lip or cheek; or it seizes on some remote part, as the toe, the leg, or the whole of a lower extremity, from which I have seen nearly the whole integument separate as a slough. Such cases are often fatal. The severity of the symptoms may produce death on the second, third,

or fourth day of the disease, as from gangrene occurring in the course of the œsophagus or alimentary canal. When the disease is continued beyond the first week, death is foretold by the rapid, small, and weak pulse; by the rapid, languid and oppressed respiration; frequent fluid acrid discharges issue from the bowels, and blood may be discharged from the nostrils, mouth, and throat, or even from the kidneys; petechial or purpuric spots appear on the skin; and the patient is at last destroyed with local manifestations of the morbid state in several different parts and organs.

Latent Scarlet Fever.—The marked prevalence of anasarca in children has led to the discovery that such children have had previous attacks of scarlet fever in such a mild form that it has escaped detection. In such cases the constitutional affection of scarlatina has been produced, but without any rash or sore throat being observed. On the kidneys alone the poison makes itself felt, and the dropsy which ensues is more severe, complicated, and fatal than that which follows the regular forms of the disease.

Special Lesions after Scarlet Fever.—Under this head it is proposed to notice more remote effects of the poison of scarlet fever,—often called by the people the “*dregs*” of the fever. The principal source of some formidable symptoms is found to be the primary obstruction to which the functions of the kidneys are liable. *Croupous nephritis* is a frequent and important lesion, not necessarily due to cold or exposure during the eruption, but which is really a localisation of the blood-poison of the specific disease. It is a more frequent lesion in some epidemics than in others. Effusions into the pleuræ may occur during its course. These effusions are of two kinds:—(*a.*) Those depending on a mechanical cause—the obstruction to the circulation through the kidneys and systemic capillaries; and, (*b.*) those resulting from inflammation of the chest; the former being much more amenable to treatment than the latter—the fluid of simple hydrothorax, or dropsy of the pleura, being more easily reabsorbed than effusion resulting from *pleuritis*. Another most important lesion is an extension of the original affection of the throat towards the internal ear, by the Eustachian tubes. When this takes place it not unfrequently happens that the small bones of the ear are completely destroyed, the tympanic cavity becomes inflamed, ulceration of the membrane takes place, perforation follows, and deafness for life is the result. This morbid state is most difficult to remedy. A chronic discharge from the middle ear is established, of the most offensive kind, which may continue till the whole of the internal ear is involved in the destructive inflammatory process; till the delicate and soft tissues in the cochlea and semicircular canals are destroyed, and the petrous portion of the bone itself dies; till the mastoid process, with its capacious osseous areolæ, becomes the seat of an obstinate carious disease; or even till the brain itself, or the membranes, are involved in an unhealthy and often fatal inflammation. Such a combination of effects occasions great and protracted sufferings, so that death is really a wished-for end (BRUCE, ANDERSON). A similar inflammation may destroy the tissues in the back part of the pharynx, extending towards the base of the cranium and upper cervical vertebræ.

Diagnosis.—The only diseases with which scarlet fever can be confounded are the acute forms of *roseola* and *measles*; and also *erysipelas*. *Roseola*, though usually accompanied by fever and sore throat, is distinguished from scarlatina by the eruption being confined generally to the chest. The diagnosis between *measles* and *scarlatina* will be better understood after the disease noticed in a note to this article has been described—namely, the hybrid form sometimes assumed by the concurrence of the two diseases; and by a careful study and observation of the ranges of temperature in each.

Prognosis.—The mortality from scarlet fever varies greatly according to the season, and also according to the fatality of the epidemic, varying from 1 in 6 to 1 in 30. There is perhaps no disease which is so capricious; the epidemic type complication and possible sequelæ must all be taken into account in giving a guarded prognosis. The mortality is greatest in the period of infancy and childhood—from one to five years. In relation to mortality, it seems second in this country as to severity, typhus fever standing first (RICHARDSON). It is twice as fatal in towns as in the country; and there is one condition in which the disease is almost invariably fatal—that is, the puerperal state. No precaution ought, then, to be neglected, no precaution ought to be thought excessive, which tends to prevent a woman from receiving the poison of scarlatina while pregnant or recently delivered. Fever during pregnancy most certainly ends in abortion and death; and if the woman be recently delivered, the disease will be of the most malignant type, and almost always fatal.

“The continued prevalence and the great fatality of scarlet fever in England and Wales, and especially in the London registration district, are such as to call for the earnest consideration of all classes of the public. To show the magnitude of the evil, it will be sufficient to say that the disease in the two years 1863 and 1864 destroyed in England alone more than 60,000 persons. In 1869 the number of victims in London amounted to 5,803. In the quarter ending on the 30th of June, in 1870, the deaths from scarlet fever in London were 1,076, and in England and Wales, 5,973; and the number registered in London alone has lately been about 200 weekly. The disease varies so much in character and severity, is sometimes so slight and sometimes so malignant, that it would be difficult to state any definite proportion between mortality and prevalence. If we assume this proportion to be one in twelve—an estimate that would probably not be very far from the truth—it follows that there have existed in London during a single quarter nearly 13,000 cases, each one of which, whether trivial or fatal, would be a new source of infection to the district in which it occurred. Putting the matter in another form, and allowing each patient a fortnight of infectious activity, we may say that London maintains a permanent stock of 2,000 centres of contagion for the propagation of this disease.” A mortality below 10 per cent. is reckoned comparatively a benign epidemic; 30 to 60 is a malignant epidemic.

Treatment.—Scarlet fever being accompanied by apparently inflammatory symptoms, the practice of bleeding has prevailed on the first breaking out of the disease in all countries, and *with the most disastrous results* (WILLAN); so that Morton speaks of witnessing 300 deaths from

scarlatina in a week. This practice prevailed down to the time of Huxham, who abandoned it, and introduced a treatment by bark. Thus an entirely opposite system of treatment has been gradually introduced, and the records of medicine enable us to state the results of these opposite modes:—"Of cases treated at the Foundling Hospital by bleeding in 1786, and of cases treated at the London Fever Hospital in 1829, in the same manner, one in six died after bleeding, while only one in twenty-two died after a directly opposite mode of treatment. The conclusion was inevitable, that the chances of recovery are diminished by the practice of bleeding, nearly in the ratio of four to one, as compared with the chances of recovery supposing the patient not to have been bled.

It should be laid down as a maxim, that in cases of scarlet fever medical advice ought always to be had recourse to as early as possible; for the worst cases we meet with are those in which the disease has, from its apparently mild character, been left to itself. In the mildest form of the disease it is sufficient to confine the patient to the house; to enjoin strictly a milk diet; to regulate the bowels; and, above all things, to avoid the *nimia diligentia medicorum*. Any active interference with the normal course of the disease is to be avoided. The sick room ought to be kept at a uniform temperature, and not exceed 60° to 65° Fahr. The patient must not be overlaid with bed-clothes; and the skin ought to be sponged over twice daily with tepid water, different parts being exposed in succession, and carefully dried with soft cloths. The water should be used without soap, but with an abundance of *Condyl's fluid* in it. As much fresh air as possible should be admitted to the room. The best drink is water, pure and cold; while soups of meat, Liebig's extract of meat, stewed fruit, and milk, ought to form the staple diet. Constipation is to be overcome by enemata of tepid water and salt, avoiding purgation by medicine. In the simpler cases of scarlet fever such management is sufficient.

In the cases commencing with much sickness and general disturbance, a gentle emetic at the outset is believed to modify the future course of the disease. *Ipecacuanha*, with or without *tartar emetic*, is the best form for administration; and half an ounce of *castor-oil* ought to be given after the action of the emetic has ceased. Looking to the morbid condition of the blood, and to the tendency which exists to the deposition of fibrine in the right cavities of the heart, small doses of *carbonate of ammonia* (three to seven grains), administered every hour, or every three hours, as soon as the symptoms are decided, have been recommended (PEART, WITT, RICHARDSON). Or, the *liquor ammoniac acetatis* may be used with an excess of *ammonia*, to the amount of from three to five drops of *liquor ammonia* added to two fluid drachms of the former in a liberal quantity of distilled water (RICHARDSON). It is important to administer these medicines in small and frequently repeated doses; and, if possible, let the remedy be taken as a drink; and as such *chlorate of potash* may be used freely.

The treatment of the milder forms of the fever, when the tonsils are considerably enlarged, is, first, to tranquillise the stomach and allay its perverted action when vomiting exists, either by small doses of the *sulphate of magnesia*, or by effervescing salines—medicines which, according to the state of the bowels, may be given every four or every six hours. The gum

resin of *guaiacum* is of great service in subduing the *cynanche tonsillaris*, and may be prescribed in the following formula:—

R. Magnes. Sulp., ℥vi.; solve in Aqua, ℥viii.; adde Pulv. Guaiaci, ℥iss.; Pulv. g. Tragacanth. co., ʒij.; *misce bene*. One-sixth part of this mixture may be given every four hours, till the bowels are freely moved.

As soon as this object is effected, and it is ascertained that the tonsils are still greatly enlarged and swollen, it may be necessary to relieve them, if no adynamic symptoms exist, by the application of six to twelve leeches about the angles of the jaw; and the bleeding may be further encouraged by the application of a poultice. The trifling loss of blood thus sustained does not impair the general strength of the patient, if it is done sufficiently early, while it greatly reduces the swelling of the tonsils, and may prevent them becoming permanently enlarged. Another advantage is gained by the application of leeches to the throat—namely, that they relieve the affection of the head symptoms. If the tonsils are thus relieved, the fever ought to be permitted to run its course uninfluenced by medicine, beyond the occasional administration of the saline draught and ice, or the inhalation of steam. Such remedies should be persevered in till the disappearance of the eruption, and till the healthy granulations of the throat, and the decline of the fever, give evidence of a state of convalescence. At this point some mild tonic medicine is desirable. With children under ten years of age, however, it is better to trust to the soothing effects of warm poultices round the throat, and inhalation of steam, than weaken the child by loss of blood.

The severe forms of the disease, characterised by the less swollen state of the tonsils, and by their being more livid and gorged with blood; by ulceration being deep and spreading; by the slough being fouler than in the milder varieties; and by the greater tendency of the inflamed parts to run into mortification—the necessity of adopting a more stimulating plan of treatment, and one more calculated to support the powers of the constitution, is manifest, and experience has shown its good results. Nourishment in the form of soups, meat extracts, or milk, must be given in small quantities frequently repeated, in proportion to the prostration of the patient. The administration of wine, and of the “*extractum carnis Liebigii*,” should therefore be the basis of the treatment of such cases. The quantity of wine for an adult may be from four to six ounces in twenty-four hours, and for a child about half that quantity or less. The wine should be taken in small quantities, mixed with two-thirds water; or it may be given with sago, arrow-root, jellies, or other demulcent food. The earlier the wine is given in the disease the better, whether delirium does or does not exist; regardless, also, as to whether the tongue is moist and white, or brown and dry; and it should be continued till the patient is convalescent. Liebig’s extract of flesh may be given like beef-tea, as a drink. While pursuing this plan, it is necessary that the patient’s bowels should be attended to. In cases more severe, *brandy* may be required, or *carbonate of ammonia* in *liquor cinchonæ*; and *iron* in some form should be given in frequently repeated small doses, either with the nourishment or with the medicines already mentioned; or it may be combined with *quinine* and *aromatic sulphuric acid*.

If an emetic is first given it may be proper, before adopting any special continuous mode of treatment, to follow its action by a dose of calomel as a purgative, and this especially with robust children, to be followed in six or seven hours by *castor-oil* or *magnesia*; and the bowels are ever afterwards to be kept open by remedies suited to the state of the patient and the nature of the disease. The following are the principal indications which must guide the treatment:—If there is much excitement of the system, in an otherwise strong, well-grown subject, depleting cathartics are to be given; if nausea and vomiting prevail, a *seidlitz powder* is of service; if the discharges from the rectum are acrid and acid, with acidity of the stomach, *magnesia* is preferable; if there is abdominal pain, *castor-oil* with *opium* (WOOD).

Again, when the synovial membranes inflame, and the joints become enlarged and swollen, all stimuli should be withdrawn, and a moderate action of the bowels should be kept up by means of the *sulphate of magnesia*, with *camphor mixture*, or with *carbonate of ammonia*, or *quinine* in infusions of *chiretta* or of *gentian*; and if pain be severe, some sedative should be added, as the *tincture of hyoscyamus* in a dose of *fifteen minims*.

The more formidable affection in scarlet fever is dropsy, sometimes with effusion into the pleuræ. Bleeding by leeches over the region of the kidneys is then of service, in cases which will tolerate this remedy, especially if oedema appears in the face, and is accompanied by headache, —from two to four ounces in the child, and from four to eight ounces in the adult may be taken. The good results of cupping (even of dry cupping) are also very remarkable; and of continuously hot poultices over the lumbar region, with or without the addition of *digitalis* (see p. 308, *ante*). By these means renal congestion is relieved, and the urine becomes more copious and less albuminous. Hot air or vapour baths, to excite cutaneous action, may be also of service, combined with diluent drinks. Diaphoretic doses of *antimony*, and moderate but not severe purging, may be had recourse to, if anasarca makes progress. The *compound powder of jalap*, or the *bitartrate of potash* alone in drachm doses, three times a day, are then most useful, especially as an electuary, in which the *cream of tartar* is mixed with nearly an equal quantity of *honey*, *treacle*, or *marmalade*, or *syrup of ginger*, and flavoured, if necessary, with a few drops of peppermint oil. *Squills* and *digitalis* are also of special service as diuretics till the urine resumes its natural appearance. In young children (of five years of age and under) *digitalis* in scarlatinal dropsy is almost always beneficial, given in combination with *perchloride of iron*. It is followed by a steady increase in the quantity of urine, with a corresponding subsidence of effusion. Five minims of the *tincture* of the *perchloride of iron*, with a drachm and a half of the infusion of *digitalis* in *anis* or *caraway* water, may be given three times a day, and continued without intermission for fourteen days. Children bear such doses, and larger ones, of *digitalis* remarkably well; and the *infusion* in preference to the *tincture*, with *iron* and a nutritious diet (CHEADLE, *Brit. Med. Journ.*, November 23, 1872). The *muriated tincture* has the best reputation. The *iodide of potassium* in small doses is also useful; and with this latter remedy the *syrup of the iodide of iron* may be combined, if it is desirable to continue the *chalybeate*;

or syrup of the phosphate of iron, combined with glycerine, in drachm doses of the combination.

Gargles are unnecessary for children, for they cannot gargle; but they are of the greatest service, especially the deodorising gargles or washes, when the patient can be taught to use them. A weak solution of *chloride of lime*, or of *chlorine water*, or of *Condy's fluid*, or *permanganate of potash*, is well adapted for such a purpose. The following is recommended as a most effectual gargle:—

Solution of peroxide of hydrogen (containing ten volumes of oxygen), six ounces; tincture of myrrh, an ounce; rose water, five ounces (RICHARDSON).

This gargle may be used at pleasure: it is refreshing to the patient, and removes the offensive secretions. With young children, who are unable to use a gargle, the throat may be washed out, by holding the little patient with the face downwards, and then pumping the solution over the surface of the fauces through a bit of gum catheter from a double-acting india-rubber bag (RICHARDSON, *Clinical Essays*, p. 110). As an invariable routine practice, Dr. W. T. Gairdner strongly recommends that "*the steam of hot water should be inhaled from the beginning to the end of the fever, as long at least as the throat is sore.*" In slight affections it is sufficient to employ *infusion of linseed* in water, acidulated with *nitro-muriatic acid*, weak solutions of *alum*, *nitre*, or *common salt*. When membranous diphtheritic patches are observed on the fauces, and the colour of the mucous membrane is of a dark red, *capsicum infusion*, or *powdered red pepper*, is an excellent application (WOOD); and in children who cannot gargle, it may be applied with a hair pencil. Solutions of *zinc*, or *nitrate of silver*, are also of service. Ulcerations may be touched daily with a solution of *nitrate of silver* (one drachm to two ounces of water), applied by means of a hair pencil or a sponge probang; while a weaker solution (grains v. to x. to two ounces of water) is at the same time injected through the nostrils when there is discharge, and it gives great relief. *Turpentine* and *glycerine* in equal parts brushed over the ulcerated throat is also a valuable application, and the *sulphurous acid spray* may be of service. In swelling of parotid and sub-maxillary glands, ice in bags of oil silk fastened round the neck and angle of the jaw, with lumps of ice in the mouth, diminish engorgement, and tend to avert suffocation. These details are given because the physician must decide, upon the merits of the individual case, the nature of the treatment he will adopt. It must be remembered that cases of scarlet fever, if left to themselves, with rest and careful nursing, will generally get well unless the epidemic constitution is a malignant type. The mere intensity of the fever is no ground for active interference by way of treatment, if the pulse is full and of good strength. Much is to be trusted to the shortness of the fever, remembering that there is no disease in which the patient is more apt to be delirious, and in which the temperature may reach a high degree, with less danger, than in scarlet fever, (W. T. GAIRDNER, *Clinical Medicine*, l. c.) On the other hand, it must also be remembered that any continuous and excessive increase of the temperature of the body may be followed by the occurrence of adynamia and threatened paralysis of the

heart from excessive prostration, and that such cases suddenly and urgently demand such therapeutic interference as may tend to reduce temperature and maintain the strength. Treatment by the *cold water douche or affusion*, as originally advocated by Drs. Jackson and Currie, is now again recommended in the treatment of malignant scarlet fever, where the cerebral symptoms are early and severe. Niemeyer and others bear testimony to its having a most markedly beneficial effect. The patient may be placed in an empty tub, and have the cold water poured over him, or his naked body may be wrapped in wet sheets, to be renewed and reapplied at intervals of ten to fifteen minutes, the patient being put to bed in the interval (p. 305, *ante*). If either of these measures fail, then such powerful stimulants as *carbonate of ammonia*, *camphor*, or *alcohol*, may be had recourse to, administered by enemata if need be, combined with *milk* or *beef-tea*, or *gruel*.

Preventive Treatment.—Mere fumigation will not, it should be remembered, destroy the miasmata in the sick-room; and, consequently, the doctrines of separation, of ventilation, and of cleanliness, are as imperative in this disease as in small-pox. The isolation of healthy persons from those affected with the disease, and from those who have intercourse with such patients, is essential, and is the only rule that promises any good results. The following rules (drawn up from the experience of Drs. Ballard and Budd) ought to be carried out in every house where conveniences can be obtained; such conveniences ought to exist in every hospital, and in the houses of the poor they ought to be carried out as far as practicable:—(1.) Remove from the sick apartment all superfluous woollen or textile matters, such as carpets, curtains, and anything of that nature, which are known to be retentive of disease-germs. (2.) Measures of disinfection should be used as early and as thoroughly as possible. Carbolic acid in solution, or as carbolate of lime, is especially useful to sprinkle on the floor, and with which all parts of the room may be washed prior to cleansing and lime-whiting; and all articles to be washed ought to be soaked first in a solution of carbolic acid. (3.) A basin charged with chloride or carbolate of lime, or some other convenient disinfectant, is to be kept constantly on the bed for the patient to spit into, which must be emptied and replaced at regular intervals. (4.) A large vessel (a tub) containing water impregnated with Condyl's fluid, or carbolic acid solution, should always stand in the room (or near by) for the immediate reception of all bed and body linen on its removal from the person or contact of the patient. (5.) In place of using pocket handkerchiefs, use small pieces of rag for wiping the mouth and nose, so that each piece, after being used, may be at once burned. (6.) Two basins, one containing Condyl's fluid or carbolic acid solution, and another containing plain soft water with carbolic soap, and a good supply of towels, must always be ready and convenient, so that the hands of nurses may be at once washed, first with soap and water, and then in Condyl's fluid water *without* soap after they may have been soiled by specific excreta. The dresses of nurses and attendants should be of linen, or smooth washable material. (7.) Glasses, cups, and other vessels used by or about the patient, are to be scrupulously cleaned before being used by others. (8.) The discharges from the bowels and kidneys are to be received, *on their very issue from the body*, into vessels charged

with disinfectants. (9.) To prevent the minute particles of desquamation from flying off as impalpable powder, their power for evil must be destroyed *in situ*, by anointing the surface of the body (the scalp included) twice a day with olive oil. It may be slightly impregnated with camphor, which Dr. Budd considers sufficient, or carbolic acid. The process relieves the itching of the skin, and is very soothing to the patient. So soon as efflorescence is observed on the skin of the neck and arms (as early sometimes as the fourth day), which marks the first liberation of the germ-carriers of the specific disease-poison, the employment of the oil is to begin, and ought to be continued until the patient is well enough to take a warm bath, in which the whole person (scalp included) is well scrubbed, carbolic acid soap (CALVERT'S or MACDOUGALL'S) being abundantly used during the process. These baths are to be repeated every second day, until four have been taken, when, as far as the skin is concerned, the disinfection may be regarded as complete, although a further quarantine of a week may be advisable. (10.) The chamber in which the sick person has been confined must now be thoroughly washed out, using freely carbolic acid and soft or black soap (which may now be got combined for the purpose) (*Brit. Med. Journal*, 1869, Jan. 9, p. 23).

If a patient be from the outset thus isolated from all who are susceptible of contagion,—if everything proceeding from him is drenched with chemical disinfectants before it leaves the sick-room,—if the exhalations from the skin, and the peeling skin itself, are mechanically imprisoned by inunction with oil until they can be removed by a warm bath,—and if all clothing, bedding, and furniture are disinfected at the close of the illness, a case of scarlet fever ought to remain barren of results. But to carry out such a process thoroughly requires the command of space, of money, and of attendance; and requires also wisdom and self-denial on the part of relatives and friends. If it were possible to begin to-morrow, and to carry it out in every case, scarlet fever would have ceased to exist in the country at the end of two months, and it is possible, or even probable, that it would never return. “When, however, we look abroad at the actual condition of the people among whom the disease works its ravages, we see at once that, with regard to very many of them, and especially with regard to the poor in towns, isolation and disinfection are no more than idle words. In the class above the very poor, among small tradespeople, small employers, and the like, where the adoption of proper measures, although difficult, would not be impossible, it is rare to find the intelligence and the active conscientiousness that would induce them to bear restraints, to take trouble, and to incur loss, for the sake of preventing injury to their neighbours. Even among the wealthy and educated, disinfection and isolation are but imperfectly carried out. Such people like to do as they please, and resent dictation from medical men. If the cases are severe, mental anxiety and distress break down the barriers of precaution. If they are trivial, strict precaution would too often be thought fussy and unnecessary. Medical men cannot in such cases enforce what is right. They can only recommend it; and they recommend it subject to the remembrance that their business is to heal the sick, not to quarrel with the healthy. It is a curious weakness of human nature, that many otherwise rational creatures are angry at the suggestion that *their* loved ones

can be sources of danger to all around them, or that *their* house is properly shunned by the neighbours; and beyond this there is the belief, not altogether unfounded, that it is useless to isolate and disinfect in a single case, where there is no reason to believe that a similar course will be generally adopted. In the case of schools and public institutions, it might be well to prevent dispersion by positive enactment. But in other instances, if the principle were once asserted and acted upon, that an infectious disease must be registered, as a matter of public concern, public opinion would do the rest. Such provisions as we have suggested are, perhaps, as much as could be at present carried into effect; and their influence, both sanitary and educational, would be of incalculable value."

There are also beginning to arise some sceptical misgivings regarding our present hygienic views and practice with regard to scarlet fever. "One failure starts a doubt in a believer's mind; a second confirms it; a series of failures makes one look out for a better creed. Are we doing any good with our present preventive means? If they could be universally enforced, as they have been in isolated families with disciplined minds, they would succeed; but when this is attempted with large thickly packed communities, can they be, or have they ever been, successfully enforced? By the imperfect execution of them through the length and breadth of the land, do we increase or lessen the mortality from disease?"

These questions were put by Dr. Davies, of Bristol, who, writing of an epidemic of scarlet fever there in 1875, continues as follows:—"I feel certain that we increase the anxiety of the domestic and social troubles of the public by our preventive measures; and I feel doubtful of the answer to the former question. Take, for example, an imaginary but typical case in private practice. There is a family named X., in easy circumstances. They have five children—three boys and two girls. There is an epidemic, of a very mild type, of scarlet fever in the locality. Hundreds have had the disease; but there has been no death. The season is July; the weather rainy, the atmosphere moist. The youngest boy, aged 4, contracts the disease in the prevalent mild type. The other four children, by the advice of their medical attendant, are sent to a distance, and escape the infection. The sick child recovers. Two years elapse. The eldest son goes to Eton, Rugby, Marlborough, or some other public school. An epidemic of a severe type has just broken out where he is: he contracts the disease. The parents are telegraphed for to see their son, supposed to be dying. They travel to and fro three hundred miles. The patient ultimately recovers, and escapes only by the skin of his teeth. The school is temporarily broken up. The convalescent is sent to a fashionable watering-place before he is properly disinfected, and there he spreads the disease. The second son has escaped, through being kept at a distance from his brother. He has, in the course of time, taken his degree at college; has been admitted as a barrister; has, after a long struggle, made his position; has been married. He passes through Paris on his honeymoon. The weather is cold, the wind easterly. In Paris an epidemic of scarlet fever prevails. He contracts the disease and dies in a strange land; and leaves his young bride a widow among strangers. In time, the eldest girl is married to a rich man. She is in due time confined of her first-born. The bells are ringing, and all goes on merrily as a 'marriage bell.' A fortnight afterwards, that house is

desolate. In that house are now only a bereaved husband and a motherless infant; the subtle seeds of this mysterious disease have been inadvertently introduced to the puerperal patient in the nurse's shawl, or on the sleeve of the doctor's coat. I will not follow this case further; but ask, in all seriousness, would not life and suffering have been saved if all this family had been allowed to pass through the disease when it first appeared among them in a mild type, when they were all children? I wish it to be thoroughly understood that, as a health-officer, I have never had the moral courage to discard my former profession of faith, and to undertake the responsibility of giving practical effect to my doubts. I have never used disinfectants so extensively as during the present epidemic, and yet our failure is complete. The doubts I have expressed do not in any way extend to typhus and enteric fevers, small-pox, and Asiatic cholera. It is a fact beyond contradiction that occasionally this tiger of pathology seems to stalk abroad, having left both claws and teeth at home. Could we not play with him *then*? Inoculation from mild cases of variola previous to the discovery of vaccination proved of immense benefit. We can import scarlet fever. We can choose our type and season of the year. *Is there a milder exanthem of an allied species that can prevent or modify this disease?* Röthlen is nearly allied, is very infectious, but harmless. Does it modify scarlet fever, or is it modified by it? I have for some time looked upon it with affection, but am afraid to theorise on the subject. Can any one throw light on this unexplored field? Observation convinces me that there is some unknown condition which gives immunity to some persons from scarlet fever: what is this condition? An analogous condition was known in the Vale of Berkeley regarding small-pox before the days of Jenner, and might have remained unknown to the outer world had not that great man unearthed it. Who will be the Jenner of scarlet fever?" A somewhat similar argument is used by Thomas in his article on scarlatina in Ziemssen's *Cyclopædia*, Vol. II., p. 300.

Different prophylactic medicines have been recommended; amongst which belladonna has had the greatest number of advocates, but its value has diminished greatly, as the weight of testimony is against its possessing any prophylactic virtues (WOOD). Even when continued for a week, it affords no protection against scarlet fever (NIEMEYER).

RUBEOLA OR RÖTHELN.

LATIN EQ., *Rubeola** (*Scarlatinosa vel Morbillosa*); FRENCH EQ., —; GERMAN EQ., *Rötheln*—Syn., *Feüermasern*; *Ritteln*, *Falschen Masern*.

Definition.—*A specific eruptive disease, having an incubation-period of ten to fourteen days (SQUIRE), or from two to three weeks (THOMAS), followed by fever, watery discharges from the eyes and nose, sneezing, and sore throat. The eruption*

* Although this form of disease has not found a name and a place in the nomenclature of the Royal College of Physicians, yet, as the evidence of its occasional occurrence is so clear, I think it proper to continue the description I have hitherto given of it in previous editions of this Text-book. I find also that Niemeyer describes, under the name of "Rose rash" (*Roseola febrilis*), an eruption with red spots which, on the authority of Constatt, one is in doubt whether it should be classed

appears on the third or fourth day, and consists of crimson stigmata, rapidly running together into patches of an irregular shape, with obtuse angles, and of sizes varying from a threepenny to a crown piece, according to the severity of the case. The eruption continues from six to ten days, and terminates in desquamation by furfuraceous scales.

Pathology.—Those diseases now fully considered in the previous pages—namely, *small-pox*, *measles*, *scarlet fever*—have been termed exanthematous diseases, in consequence of a very marked eruption being one of their most conspicuous phenomena. The Arabians first described them, and considered them as varieties of one and the same disorder. Many essential differences, however, were soon seen to characterise and render small-pox distinctive; but the points of resemblance between measles and scarlet fever were so many, that it was not until fatal accidents had occurred, from the error of confounding them, that their differential characters were remarked, and their separate identity established. Now it is a generally received doctrine that measles and scarlatina, in their essence and in their symptoms, present two well-defined diseases. This is, indeed, one of the most indisputable facts in Pathology. By Schönlein *measles* has been classified as a peculiar exanthematic form of catarrh, and scarlet fever is placed amongst the group of erysipelatous diseases; while, according to the experience of Dr. Küttner, of Dresden, there are “androgynous” cases calculated to embarrass the most experienced “diagnostiker.” *Measles* and *scarlet fever* were especially confounded under the common name of *morbilli*; and even as late as the middle of the eighteenth century writers of the highest repute supported the identity of measles and scarlet fever (the *morbilli confluentes* of Sir William Watson). All authors before Sauvages (1768) had used the term *morbilli* (the term now in use) to designate measles; but he adopted a new name, and called measles by the designation of “*rubeola*”—an innovation which has caused much confusion, having been adopted by some (such as by Willan and Bateman) and rejected by others. Hildebrand, following the old nomenclature, calls measles *morbilli*, and scarlet fever *scarlatina*; and terms the disease—now about to be described—“*rubeola*,” as has been done by Dr. Copland. The German authors call it “*rötheln*,” and by this name it was first described by a distinguished and learned Scotch physician, Dr. Robert Paterson, of Leith, in 1840. He is the only physician in this country who has given an original description of the disease in the English language, his description being drawn from numerous cases of it which occurred in his practice, some of which he showed me when I was his pupil in the fever wards, sheds, and tents, of the Hospital in Edinburgh, in 1846 and 1847. A difference with scarlatina or measles, from the general symptoms and affection of the mucous membranes. Moreover, the epidemic form of the disease he considers to arise from infection, and to consist in modification or combination of scarlet fever and measles. By *rubeola scarlatinosa* is to be understood a scarlet fever where the exanthem resembles measles, while the high fever, the throat affection, and the drowsy which often follows, resemble the course of scarlet fever. By *rubeola morbillosa* is meant a form of measles where the exanthem is confluent, and resembles that of scarlet fever, while the affection of the respiratory mucous membrane, and the escape of the pharyngeal mucous membrane, leave no doubt of the morbillous nature of the disease. This short description of Niemeyer is sufficient to identify this hybrid disease with *Rubeola* or *Rötheln*. It is also described by Thomas as an independent and specific disease (ZIEMSEN'S *Cyclopædia*, Vol. II., p. 133).

of opinion prevails amongst authors as to whether or not this disease is of a distinct and specific form. Those who have most recently described it (Hildebrand, Paterson, and Copland) consider it to be a disease possessing characters common to both measles and scarlet fever as well as characters peculiarly its own. Thomas considers measles and rubeola to be two specifically distinct infectious diseases—rendered evident by the fact that the two diseases afford no mutual protection. Nor does rubeola protect from scarlet fever or *vice versa*. Each disease may occur in the same person within a few weeks or months; and infection always produces the same disease.

Symptoms.—The febrile stage of the disease varies, like all the diseases already noticed, not only in the severity of the symptoms, but also in the length of the attack, when compared with scarlet fever. It usually commences with rigors, not severe, but continuous. More or less cough soon makes its appearance—of the same clanging nature which is observed in the febrile stage of true measles—and is very shortly accompanied with itchiness, redness, and weakness of the eyes, lachrymation, frequent sneezing, and watery discharge from the nose. In persons more advanced in life, severe frontal headache is complained of, together with rheumatic pains, more especially in the muscles of the back and chest, nausea, and sometimes vomiting, together with constant drowsiness. The skin is hot and dry, with the pulse above the natural standard. Sore throat is a most constant symptom—one of the most characteristic features of the disease, occurring in the slightest and most gentle cases (ROBERT PATERSON). In some cases it is extremely slight, amounting only to a roughness of speech and trifling difficulty in swallowing; in others it goes on to severe inflammation of the *tonsils*, *velum pendulum palati*, and surrounding parts—more severe during the eruptive stage. The odour given forth by patients under this disease is described by Dr. Heim, of Berlin, as similar to but stronger than that which scarlet fever patients emit, and has been likened to the smell of a place where fish is kept—in short, fishy. The appearance of an eruption is sudden and general on the third or fourth day. It breaks out all at once over the whole body, consisting of bright and thickly set stigmata, which appear on the trunk, but are more sparingly dispersed over the face and extremities. It assumes different aspects and degrees of confluence, according to the severity of the case. Its first appearance resembles measles, but the stigmata rapidly run together, and soon assume an irregular shape, with obtuse blunt angles. These irregular patches are of an intense red colour towards the centre, being gradually shaded off towards the margins, which approach in colour that of the surrounding skin. The size of the patches in ordinary cases seldom exceeds a sixpenny piece; but in the severe forms of the disease they run still further together, and are to be seen of the size of a crown piece. In such cases, which are usually of a malignant nature, the whole body may be covered over with patches, varying from the size of a sixpenny piece to a crown piece, thickly set together, and of an intensely dark colour towards their centres. The eruptive patches are felt to be distinctly elevated above the skin—some more than others, and always greatest in the centre of the patch.

During the continuance of the eruption, the general symptoms already

described are usually aggravated, and not unfrequently new symptoms are superadded. The sore throat becomes much worse. The hoarseness becomes so great as frequently to cause entire loss of voice, and generally more or less external tumefaction of the throat takes place. In severe cases this is great, and is accompanied with much redness and swelling of the throat internally. There is a total inability to swallow even the slightest portion of fluid, which generally regurgitates by the nose. A large secretion of mucus of a vitiated nature takes place, the cough is constant, and is rendered doubly severe by the state of the throat. The pulse is frequent; the skin hot and dry; and there is great restlessness, children tossing the head frequently from side to side, accompanied with frequent starting; and they are sometimes seized with convulsions. It is in this stage, in the worst forms of the disease, that death generally occurs, by coma. It may, however, take place either by suffocation from the large quantity of vitiated mucus, or by convulsions, and subsequent coma. Vomiting is an occasional symptom, and, like convulsions, is sometimes seen in mild cases of the disease. The eruption in mild cases continues distinct for from four to five days, during which time the other symptoms are going on favourably, becoming gradually milder as the period of the decline of the eruption draws near. In severe cases, however, the rash keeps its bright colour and distinct form for a much longer period—*e.g.*, six, eight, or ten days. The termination of the eruptive stage is, in some instances, marked by what is termed a distinct crisis,—such as the occurrence of copious sweating, deposits from the urine, diarrhoea, and epistaxis. Most commonly, however, there is no such crisis, but the eruption gradually fades, and the disease subsides. As this happens, the desquamation by furfuraceous scales gradually ensues—an event indicated by the appearance of scales towards the centre of the patches of eruption, to the margins of which they gradually extend, and soon spread over the whole body. The scales are small, and not unlike those of measles. On the hands and feet they are larger, but never reach the size of those of scarlet fever (ROBERT PATERSON).

Lesions seen in Fatal Cases.—The accounts of these are few in number. They vary according to the period of the disease at which death occurs, which most frequently happens during the eruptive stage, from coma, or from the affection of the throat and lungs. No morbid appearances of a uniform nature can be observed connected with the mode of death by coma; but when death happens from pulmonary oppression, the lungs are found much congested, the mucous membrane of the bronchia injected, with a copious mucous secretion. The throat presents very similar appearances to those which are seen in scarlet fever,—great tumefaction, and dark coloration of the membrane lining the throat, dark aphthous spots, and large quantities of vitiated viscid mucus.

Diagnosis.—The accompanying febrile symptoms at once distinguish the disease from *roseola*, as also do the peculiar characters of the eruption. The only other affections with which it may be confounded are *measles* and *scarlet fever*. The following table points out the diagnostic marks more clearly by contrast than can otherwise be done, and shows that "*rubeola*," or "*rotheln*," the mixed disease, has every right to be considered as a distinct affection:—

TABLE SHOWING THE MOST PROMINENT DISTINGUISHING CHARACTERS
OF SCARLET FEVER, RUBEOLA, AND MEASLES (Paterson).

SCARLET FEVER.

RUBEOLA OR RÖTHELN.

MEASLES.

Symptoms of First Stage, or Premonitory Fever.

Rigors; nausea; sometimes vomiting, thirst, and heat of skin; with sore throat, hoarseness, and delirium, in the anginose variety of scarlatina.

Shiverings; nausea; rarely vomiting; itching; redness and pain of the eyes, with increased flow of tears; sneezing, and watery discharge from nose; cough, sore throat, and hoarseness.

Rigors; nausea, and sometimes vomiting; frequent starting during sleep; itching; redness; pain of eyes; watery discharge from eyes and nose; sneezing; harsh cough.

Duration of Premonitory Fever.

Premonitory fever is of short duration; the eruption most generally making its appearance on the second day.

The eruption generally breaks out on the third or fourth day, so that the premonitory fever is prolonged over that time.

Eruption makes its appearance towards the close of the third, or beginning of the fourth day.

Appearance of Exanthematous Eruption.

It first appears in innumerable red dots or points, being at first of a pale red colour, soon acquiring a deeper tint, and at last giving the affected portion of skin a uniform red appearance.

The rash appears in minute dots, and rapidly assumes the appearance of irregular-shaped patches, with obtuse angles, varying in size from that of a three-penny to much larger than a crown piece. The red rash is gradually shaded off with the surrounding skin.

The rash appears in minute red points, like flea-bites; several of them soon coalesce, and form rounded masses, irregular-shaped crescents, or semi-circular patches.

Roughness or Elevation of the Affected Skin.

There is a perceptible roughness in the skin affected with scarlatina. It is in general most evident on the breast and extremities, and seems to consist of the enlarged papillæ of the skin.

In this disease, more especially in the severer forms of it, the patches of eruption are distinctly and considerably elevated, and more especially towards the centre of the patch.

The elevation of the patches of eruption in measles is slight; though in general distinct in the worst cases, they are certainly not at all elevated as a rule.

Part of the Body First Affected.

The efflorescence is first perceptible on the face, neck, and chest, gradually passing downwards, and becoming diffused over the whole body.

The efflorescence first appears on the trunk of the body, the whole of which it at once occupies. It is always more sparingly seen on the extremities, but seems to break out there at the same time as it does on the trunk.

The efflorescence first appears on the forehead and among the roots of the hair; and spreads slowly and successively over the neck, chest, trunk, and extremities.

Duration of the Eruption.

The eruption remains present three days; begins to disappear on the fourth day; and is almost entirely gone by the termination of the fifth day.

In the röteln the duration of the eruption seems to depend upon the severity of the disease; in mild cases remaining out four or five days, and in bad cases six or ten days.

In this disease it remains out three days.

Symptoms accompanying the Eruption.

The symptoms which accompany the eruption in each of the three diseases are quite the same as those of the premonitory fever. It is proper here, however, to remark, that it is only in the anginose and malignant varieties of scarlatina that we have sore throat, there being little or none in the simple scarlatina, while in the mildest kind of rütheln this is always a prominent and troublesome symptom.

Desquamation.

The cuticle in this disease is thrown off in patches of considerable size, the largest being from the hands and feet.

The desquamation of rütheln consists of minute portions of cuticle, like scales of fine bran. The desquamation always begins towards the centre of the eruptive patch, and gradually extends to the circumference.

The desquamation of measles consists of minute portions of cuticle, like scales of fine bran.

Sequelæ.

Anasarca is the most common sequela of scarlet fever. It is extremely common, and most frequently occurs after the mildest cases; swelling and suppuration of the cervical glands are also common.

“I have noticed one case of dropsy after a mild, though well-marked attack of this disease; swelling and suppuration of the cervical glands also frequently take place” (Dr. PATERSON).

Affections of the lungs and pleura; tedious distressing cough; chronic bronchitis; pneumonia tubercles; gangrenous inflammation of cheeks, gums, lips, genital organs, &c.; dropsy occasionally occurs, but very rarely; diarrhœa is very common after some epidemics.

Prognosis.—In general it is a mild disease; but the prognosis requires to be as guarded as in scarlatina; for, like scarlatina, rubeola is often an extremely and rapidly fatal disorder. The greater or less acuteness of the premonitory fever generally affords us a means of judging as to the probable severity of the eruptive stage. To have a copious secretion of mucus in the back of the throat is always a bad symptom, or regurgitation of fluids by the nose. The chest ought to be examined from day to day, as sudden inflammatory action is apt to be established, and it may rapidly prove fatal. The condition of the urine requires also to be daily investigated.

Treatment.—The treatment is similar to that of scarlet fever. The functions of the skin are if possible to be stimulated; and Dr. Paterson found that the *aqua acetatis ammoniacæ*, in the proportion of two ounces to half an ounce of *antimonial wine*, and four ounces of *water*, was a most useful remedy. The use of *colchicum* was also had recourse to with decided benefit.

DENGUE.

LATIN EQ., *Denguis*; FRENCH EQ., *Dengue*; GERMAN EQ., *Dengue*; ITALIAN EQ., *Dengue*.

Definition.—*A specific continued fever or febricula, eminently contagious, and having an incubation-period of a few hours to one to seven days (CHARLES),*

characterised by frontal headache, by severe pains in the limbs and trunk, and sometimes by an eruption, resembling that of measles, over the body. The disease occurs chiefly in the West Indies.

Pathology.—This is a peculiar febrile affection having two eruptions—an *initial* rash and a *terminal* rash—attended with severe pains in the joints which swell, succeeded by general heat of skin, intense pain in the head and eyeballs, the appearance of the cutaneous terminal eruption or efflorescence on the third day, spreading over the whole body within forty-eight hours, and rarely continuing visible beyond twenty-four hours. Remissions and relapses are numerous; and the disease may persist about two months, associated with great prostration and cachexia. The relapses are marked by rheumatic or neuralgic phenomena, and a return of all the excruciating pains of joints which began the disease.

The disease seems to combine an exanthematous eruption, ushered in by fever, with a rheumatic or neuralgic state; and the course of the malady is so divided by intervals and remissions as to give one the idea that relapses are of frequent occurrence in its course. It has been chiefly prevalent in Rangoon, Calcutta, Berhampore, Patna, Benares, and Chunarghur, in the East Indies; the island of St. Thomas in the West Indies; the Southern States of America; the ports on the Gulf of Mexico; the towns of New Orleans, Savannah, Charleston, Philadelphia, and New York. It was epidemic in America in 1824-28; and nothing appears to have been heard of it again till 1847 and 1850, when it again visited the Southern States. An epidemic of dengue has been recently described by Lemmon as having occurred in Virginia (*Amer. Med. Times*, Feb. 16, 1861). It prevailed as an epidemic on the East Coast of Africa, in Zanzibar, from July, 1870, till January, 1871, an excellent account of which is given by Dr. James Christie, Physician to the Sultan of Zanzibar. It has been described by Nicholson, Mellis, Kennedy, Wood, Cavell, Twining, Mouat, and Goodeve. It broke out in Spain in 1764-1768, and again in 1865-1867; so that, like relapsing fever, it occurs at long intervals as an epidemic. In Spain it was called *la piadosa*—the piteous disease, also *la pantomima*. In 1870 it again appeared as an epidemic in India, having been absent twenty years; so that it remains an unknown disease to a generation of medical men. The epidemic of 1870 evidently came to India from Eastern Africa, Zanzibar, and Aden; and obtained a very wide distribution in Hindostan, especially in Burmah, Bengal, Madras, and Bombay Presidencies; and in some stations hardly an individual escaped. An excellent account of this epidemic has been published by Dr. T. E. Charles, Professor of Midwifery in the Calcutta Medical College. It is not known as an epidemic disease in Great Britain.

Dr. Richardson, in his admirable *Clinical Essays*, notices the circumstance that we not unfrequently meet with "scarlet fever connected with acute rheumatic fever;" and he ascribes the first notice of this connection in this country to Dr. Golding Bird. Subsequently the circumstance was noticed by Dr. Kelso, of Lisburn, and by Dr. Ross. Dr. Andrew Anderson, in his *Lectures Introductory to the Study of Fever*, notices that rheumatic pains of the arms and legs, often very severe, connected, as he supposes, with the poisoned state of the blood, are not unfrequently met with. Lastly, Dr. Richardson himself records the cases of four children,

in which the rheumatic state, combined with scarlatina, was distinctly expressed (*Clinical Essays*, p. 85). Dr. Wilkes has noticed similar cases; but Dr. Charles and others agree that dengue has no pathological kinship relating it to scarlatina more closely than to measles, or any other eruptive disorder.

Symptoms.—The invasion is very sudden, and the development rapid. The patient goes to bed quite well, and wakes up about two or three in the morning with a hot skin—so hot that in a few hours the temperature in axilla ranges from 99° to 103° Fahr., and a pulse varying from 78 to 108, and a countenance indicative of utter helplessness and prostration. In the greater number of cases the first symptoms have been headache, with intolerance of light, restlessness, and more or less chilliness, debility, pains in the back, the limbs, and joints. The small joints swell, and there is soreness, with stiffness of the muscles. The skin soon becomes hot and dry, the pulse frequent, the face flushed, and the eyes red and watery. The tongue, though red, is usually clean. An initial rash or erysipelatous-like discoloration appears, accompanied with a puffy swelling of the face. Painful swellings in the lymphatic glands of the neck, axilla, and groins are common in the third period of the disease. The testicles also swell, and continue so till the subsidence of the other symptoms. The febrile state lasts from twelve hours to three or four days, after which it subsides, leaving the patient very feeble. This remission lasts for two, three, or four days, when a return of the fever and pains, with a thickly-coated tongue, nausea, and epigastric tenderness, mark another phase of the disease. In the Indian epidemic of 1870 the tongue presented one or more of the following characters, namely:—(1.) A thick coating of yellow pus on the dorsum; (2.) a slight whitish pus on the dorsum with red tips and edges; (3.) the whole tongue glazed and red. Vomiting was present in 12 per cent. of the cases (MORGAN, *Madras Journal*, August, 1872, p. 89). On the third, fifth, sixth, or seventh day an eruption (terminal) appears in the form of a scarlet efflorescence at the root of the neck and upper part of the chest, whence it spreads rapidly downwards over the body, and gives relief to the symptoms of febrile irritation. The eruption is extremely variable in character, sometimes smooth, red, and continuous, as in scarlet fever; sometimes in patches, rough, and of a dark hue, as in measles; and occasionally either papular, vesicular, pustular, or furunculous; often with a mixture of two or more of these forms. The complaint gradually subsides, and leaves the patient with some rheumatic stiffness or soreness for a longer or shorter period; and with feelings of weakness and mental depression. The duration of the affection varies with the length of the remission; but on the average is about eight days. The victims of dengue are to be commiserated for the horrible and agonising pains peculiar to the disease; and are reassured by the fact that it is of a non-fatal nature. In a few instances young children may die from convulsions during the severity of the attack. The recurrence of the excruciating pains in the limbs at a period long after the subsidence of the fever, ought to be held in remembrance in any prognosis as to the probable duration of the illness.

Diagnosis.—The peculiar initial rash and subsequent terminal eruption

with the characteristic phenomena of rheumatic-like pains, and intercurrent remissions of febrile phenomena, distinguish the disease from rheumatism on the one hand, and malarious fever on the other. The great debility and prostration of the vital powers are suggestive of influenza; but the implication of the mucous tract is far less than in influenza.

Treatment.—Time is an essential element in the treatment. Emetics and purgation may be given if indicated. *Calumba* and *rhubarb* with *soda* form a useful alterative medicine. Emetics of tartrate of *antimony* and *ipecacuanha* bring away large quantities of bile, relieving the pains of the head and limbs almost immediately; and the eliminative action ought afterwards to be maintained by *sulphate of magnesia* and *tartar emetic*. It may be necessary to repeat the emetic till bile is discharged from the stomach. Dr. Charles' experience leads him to say that most patients get on better without any emetic, which ought only to be given where the tongue is much loaded and where there is gastric derangement and nausea. *Colchicum*, *sweet spirits of nitre*, *nitrate of potash*, and *antimony*, in proportions sufficient for diaphoresis, given every two hours, with an additional effervescing draught, exercise a beneficial influence on the pains. After the acute symptoms subside, forty to sixty drops of the *wine of colchicum*, with twenty-five drops of *laudanum*, to which thirty drops of *antimonial wine* may be added, always ensure a good night's rest. Dr. Christie treated his cases by purgation during the first day, and in other respects as he would a case of malarious fever, by five-grain doses of *quinine*. When the febrile symptoms subsided *iodide of potassium*, in four-grain doses, was given, and continued during the remission, and for a few days after desquamation of the cuticle had taken place. Dr. Charles considers purgation ought not to be used in dengue, as it adds to the tortures of the disease and is too lowering. He considers *belladonna* a sovereign remedy in the pains of dengue, and prescribes it thus,—To one drachm of the tincture of *belladonna* he adds five drachms of *orange syrup*, and directs a *sixth part* to be taken every hour by an adult till the third dose is given, which may give so marked relief that no more need be taken. Less than three such doses seldom is sufficient, and in very severe pains two or three doses may be given in one. *One-third to half grain* doses of the extract, repeated twice or thrice in the twenty-four hours, will also generally suffice. *Atropia* may be used hypodermically in solution of *one grain* of the *sulphate* to *an ounce of water*; of which 8 *minims* are sufficient for most cases, while 10 *minims* (containing $\frac{1}{48}$ of a grain) is a full dose for a powerful, well-developed adult. Such a dose must not be exceeded in any case; and *atropia* is only to be used thus to afford rapid relief when pain is very intense, and has lasted for some time. *Five minims* of the *solution* is enough for a child of twelve years (containing $\frac{1}{96}$ of a grain). *Cold water* in the form of cold sponging has been found of great service in the hyperpyrexia of dengue; and the cold bath is worthy of consideration.

TYPHUS FEVER.

LATIN EQ., *Febris Typhus* ; FRENCH EQ., *Typhus* ; GERMAN EQ., *Exanthematischer Typhus*—Syn., *Fleckfieber* ; ITALIAN EQ., *Tifo*.

Definition.—*A continued specific fever, having a duration of from ten to twenty-one days, usually fourteen, characterised by an eruption on the skin, of a general dusky mottled rubeoloid rash, appearing between the fourth and seventh days, at first slightly elevated ; disappearing on pressure, but, after the second day, persistent, and remaining so for eleven or twelve days ; and often becoming converted into true petechiæ. Languor and weariness are prominent from the first, gradually passing into sluggishness of intellect with confusion of thought, followed at the end of the first week by delirium, stupidity, oblivion, and complete prostration. In still more severe cases, somnolence, stupor, and sometimes coma, with tremors, subsultus, and contracted pupils, supervene, when prostration becomes profound. The disease may terminate favourably from the thirteenth to the seventeenth day. If it proves fatal, it is generally between the twelfth and the twentieth day, leaving no specific lesion in any part of the body, beyond hyperæmia and blood-changes, softening and disintegration of the heart and voluntary muscles, hypostatic congestion of the lungs, atrophy of the brain, and œdema of the sub-arachnoid and pia mater. Typhus fever is eminently contagious, and occurs in strongly-marked epidemics (MURCHISON).*

Pathology.—(a.) *Historical Notice.*—The first authentic accounts of typhus fever in this country are to be found in the early British chronicles. The disease is described as having spread in our courts of justice, giving rise to what was termed “the black assizes,” the last of which happened at the Sessions of the Old Bailey in 1756, when the Lord Mayor of London, two of the judges, and several eminent persons died, infected by the prisoners. Hence this fever has had many popular appellations; such as, the *jail fever*, *hospital fever*, *ship fever*, *putrid fever*, *brain fever*, *bilious fever*, *spotted fever*, *petechial fever*, *camp fever* ; and we are indebted to Pringle and to Fordyce for having shown that these supposed different fevers are identically the same, having no such essential differences as to constitute them distinct species. Typhus fever is as old as the disputes of nations; and it has been the grand scourge of armies in temperate climates, just as cholera and yellow fever have been destructive agents in tropical wars; and in all the English wars there has been plenty of typhus poison waiting for favourable conditions to assume activity. Its prevalence in the army arose from the peculiar system of recruiting. Commissions or commands of regiments were wont to be given to those who collected a certain number of men. Every low purlieu, every infamous haunt, every jail even, used to be ransacked for recruits. Wherever these men went they carried typhus, at that time the constant scourge of our towns and jails; and complaints of the introduction of typhus fever from this source are frequently found in the writings of army surgeons of the last century. In connection with this point, Dr. Donald Munro, in 1764, gives the following caution:—“That particular regard be paid to those soldiers picked up in the streets, or who have been taken out of the Savoy or other jails. All dirty rags

from such people should be thrown away or burnt," (Dr. PARKES "On the Causes* of Sickness in English Wars," *Journal of Royal United Service Institution*, Vol. VI.) The poison may be said, like that of small-pox, to be constantly in existence. Wherever men are closely crowded together in ill-ventilated, unwholesome dwellings, typhus is sure to appear. Hence the belief in its *de novo* origin; but, on the other hand, it has often passed from the army to the civil population, and has thus dispeopled towns, and even great districts of country. Its ravages, however, in the English army have never been comparable to those which have occurred in foreign forces (MURCHISON and PARKES). "In the year 1489 no fewer than 17,000 of the troops of Ferdinand, then besieging Granada, were destroyed by a spotted fever, to which the Spaniards applied the same name that they afterwards gave to typhus. In 1552 a petechial fever devastated the army of the Emperor Charles V. during the siege of Metz. In 1556 the notorious '*Morbus Hungaricus*' appeared in Hungary in the army of Maximilian II., and thence spread over the whole of Europe" (MURCHISON, l. c., p. 21). "In 1620 the Bavarian army, in a few months, lost in Bohemia not less than 20,000 men from *spotted typhus*, and the disease, being carried into other parts of Germany, obtained the name of 'the Bohemian disease.' In 1628 and 1632 the Swedish army under Gustavus Adolphus carried typhus into Northern Germany, and the population was so destroyed that, fifty or sixty years later, villages were left without inhabitants," (PARKES, l. c.) In the spring of 1643, while the Earl of Essex was besieging the town of Reading, this disease broke out in the army of the Parliamentary General, and in the garrison commanded by Charles I. It was communicated to the inhabitants of the surrounding country, and proved very fatal to them (MURCHISON, l. c.) The wars of Louis XIV. were always followed by this disease, and the losses of the French army from typhus were enormous (PARKES). In 1799-1800 an epidemic of typhus occurred at Genoa, when the garrison was besieged by the French and half-famished; and the French army, during their retreat from Italy, communicated typhus fever to the inhabitants of fifteen towns and villages where they halted on the route (FODERE). It was during the first fifteen years of the present century that the greatest ravages of typhus have been recorded, especially in the armies of Napoleon, and among the population of the countries which were the seat of war. It always became developed under circumstances of misery and privation; and it was particularly preva-

* These causes are as follow:—(1.) A defective commissariat, especially as to food and fresh vegetables, causing diseases, but mainly predisposing to many more—*e.g.*, malignant malarious fevers, scurvy, and bloody flux. Carthage, 1741; Burmah, 1824; China, 1840. (2.) Undertaking military operations in an unhealthy site, and with an unhealthy season impending. Carthage, 1741; San Domingo, 1796; Walcheren, 1747; Java, 1811; American War, 1814; Bulgaria, 1853-54. (3.) Exposure to cold, with insufficient clothing and food, giving rise to catarrhs, slight dysentery, rheumatism, and inflammations. Wars of 1742-1760; Crimea, 1854. (4.) Propagation of typhus poison, favoured by bad ventilation, overcrowding, and filth. Examples as above detailed. (5.) Similar propagation of putrid dysentery. Indian campaigns. (6.) Propagation of typhoid fever poison and cholera, through the bad sanitary condition of camps, and the occupation of old camping grounds. Egypt, 1801; Bulgaria, 1853; India. (7.) The enlistment of boys as soldiers whose bones are not yet matured, in place of full-grown men at least twenty-one years of age. Crimea, 1854. [See a short publication *On the Growth of the Recruit and Young Soldier*, by William Aitken, M.D.] (8.) Want of cleanliness, excessive use of spirits, and debauchery (PARKES, l. c.)

lent and fatal among the inhabitants of besieged cities; as, for example, Saragossa and Torgau, Dantzic and Wilna, in 1803. It also told with awful severity upon the famished French troops during the retreat from Moscow in 1812 and 1813 (MURCHISON). When Sir John Moore's army landed from Corunna, typhus became epidemic in the military hospitals in the south of England (CHEYNE, *Dub. Hosp. Report*, Vol. II., p. 3). In May, 1812, the Bavarian army serving among the French numbered 28,000 men; in February, 1813, there were only 2,250 men under arms. The great destroyer of these men was *typhus fever*. In August, 1813, the first Prussian army consisted of 37,728 fighting men, having lost 16,000 men by the sword, and 10,000 men by disease, almost entirely typhus. In Mayence alone, of 60,000 French troops composing the garrison in 1813-14, there died of typhus in six months 25,000 men (MURCHISON, p. 224). The last great ravages of typhus in armies which attracted public attention were those in the French and Russian troops in the Crimea during and after the capture of Sebastopol. Typhus had prevailed in the winter of 1854-55 amongst both the English and French troops; but in the following winter it was mainly confined to the French and Russian armies. In the spring of 1856 it was computed that more than 17,000 men of the French forces perished in less than three months; and the highest authority stated that the safety of the whole French army was endangered by the outbreak (PARKES and MURCHISON). One important conclusion results from the history of typhus fever, among others noted by Dr. Murchison,—namely, that although typhus fever varies in its variety and duration at different times and under different circumstances, there is no evidence of any change in its type or essential character. The typhus of modern times is the same as that described by Frascatorius and Cardanas. The period during which epidemic fever was said to present an inflammatory type, was that in which *relapsing fever* was most prevalent; and the times in which the type has been described as adynamic, have been those in which *relapsing fever* has been scarce or absent (MURCHISON, 2nd Edition, p. 56).

(b.) *Origin and Propagation*.—It is yet a question, nevertheless, with those who believe in the *de novo* generation of specific diseases, whether great overcrowding and vitiation of air by organic impurities emanating from the respiratory, cutaneous, and other functions may not absolutely generate typhus fever *de novo*. There is, however, ample proof, on the other hand, that typhus fever may be communicated by *fomites* adhering to apartments, articles of clothing, and the like; and, provided fresh air be excluded, it is known that such articles will retain the poison for a long time. Herein lies a fallacy which pervades the argument from cases to prove the generation of the disease *de novo*. The late Dr. Murchison quotes some striking instances of the propagation of typhus fever by *fomites*. For example, the instance related by Foderé, in which the soldiers of the French army, during their retreat from Italy, in 1799, communicated fever to the inhabitants of towns and villages where they halted on their route, although the army was not attacked by fever, and soldiers travelling *singly* did not communicate the disease. But as he omits to connect this with the fact that typhus prevailed to a great extent in the towns they besieged (and in some instances obtained posses-

sion of), the source of the fomites is not made apparent; and, therefore, in a previous edition (fourth) I was led to misrepresent this instance given by him, and to put it forth as an example of generation *de novo*. (See p. 88, Second Edition, of his work *On Continued Fevers*.) He quotes it to show that the disease may be propagated, under certain conditions, by persons not suffering from it. He quotes also the comparatively recent instance of the Egyptian vessel, the "Scheah Gehald," at Liverpool, the crew of which disseminated the poison of typhus by their clothes and persons, although (as he says) they had not the disease themselves. But the careful investigation made by the late Dr. Parkes into the history of this Egyptian ship clearly shows that the crew did suffer from typhus fever (*Statistical, Sanitary, and Medical Reports of the Army Medical Department* for 1860, p. 359; see also *Sixth Edition of this work for a detailed history of the events with reference to the disease on board this ship*). The case of this Egyptian vessel afforded almost the best opportunity seen in this generation for the investigation of the important question of the *spontaneous generation of typhus fever*. The opportunity was, however, lost. That all the circumstances which have been supposed to be capable of calling into existence the specific poison of typhus were present in this foul and filthy ship is clear; but every one who reads the published statements will at once perceive that one link of the chain of evidence is wanting,—that it has "*not been proved that some of the crew were not ill with typhus when they embarked at Alexandria, or became ill within the incubative period.*" On the contrary, the interpreter informed Mr. Pemberton that some of the men were sick when they came on board. It can never now be ascertained whether there were such cases of illness or not; and the history of the outbreak at Liverpool affords another instance of the loss of a great opportunity for definitely setting at rest a most important question (PARKES). The case of the "Scheah Gehald" now assumes exactly the same aspect as many instances historically quoted as examples of generation *de novo*—namely, that however plausible may seem the probability, there is *no proof* that typhus fever arose *de novo*. Seeing that such is the state of the question as to the origin of typhus; that it is exactly in the same state as our knowledge regarding the origin of small-pox or of typhoid fever; that it has been in existence from the earliest periods of the world's history; that it is undoubtedly propagated from pre-existing *foci*, and by continuous succession,—the immediate direction of investigation ought to bear especially on the following points, namely:—How long can the *typhus poison* exist or be maintained in a condition fit to assume activity under favourable circumstances? What is the distance at which it is potent? Has atmospheric temperature any influence upon it? What are the conditions or combination of circumstances more or less essential to the development and propagation of the *typhus fever*?

The fact that typhus fever is contagious is based on evidence which shows,—(1.) That when typhus commences in a house or district, it often spreads with great rapidity; (2.) That the prevalence of typhus in single houses, or in circumscribed districts, is in direct proportion to the degree of intercourse between the healthy and the sick; (3.) That persons in comfortable circumstances, and living in localities where the disease is unknown, are attacked on visiting infected persons at a distance; (4.) That

typhus is often imported by infected persons into localities previously free from it; Lastly, That its contagious nature is indicated from the success attending the measures taken to prevent its propagation, more especially the early removal of the sick (see MURCHISON). The specific poison seems capable of transmission in various ways; but many circumstances point to the cutaneous and pulmonary exhalations of the sick as the media which convey it from the diseased to the healthy; "and it is obvious that its propagation must be favoured by the concentration of those emanations." It is thus conveyed through the air or by fomites. Such material particles are inhaled or swallowed, and so find admission into the bodies of the healthy, to exercise their morbid influence on the blood. A peculiar pungent odour emanates from the *typhus fever* patient. It is especially obvious from the breath, and from the skin on turning down the bed-clothes; but there is no evidence to show the extent of space through which the typhus poison can be transmitted through the air. From some observations, however, it would seem that the contagious influence of typhus is confined to a narrower sphere than that of small-pox. Dr. Murchison concludes that, "if a patient be placed in a large well-ventilated apartment, the attendants incur little risk; and the other residents in the same house—none whatever. There are likewise no grounds for the popular belief that typhus may be propagated through the atmosphere from a fever hospital to the houses in its neighbourhood. On the other hand, medical attendants who auscultate typhus patients, or who inhale their concentrated exhalations from under the bed-clothes, run no small danger, and the danger is always increased or diminished in proportion to the supply of fresh air" (MURCHISON, l. c., 2nd Edition, p. 87). "*Typhus fever* was several times carried to the West Indies; and even there prevailed apparently to some extent" (PARKES, *On the Causes of Sickness in English Wars*). The poison may also adhere as *fomites* to the walls of dwellings, to beams of wood, and to articles of furniture. Dr. Murchison, besides that of Foederé (*ante*), quotes an account by Pringle of twenty-three persons being employed in refitting old tents in which typhus patients had lain, when seventeen of these persons died of the infection. He also refers to an observation of Lind, who mentions several instances in which infected ships continued to impart the disease long after the sick had been removed. Similar cases are recorded by Jacquot respecting the Crimean typhus of 1854. Nurses and other attendants in fever hospitals are well aware of the danger of contracting typhus from infected clothes, and from cleaning the soiled bedding of the sick. In some instances they are in the habit of "measuring the amount of danger by the badness of the smell." Thus they are liable to contract *typhus fever* without having had any direct communication with the sick. With regard to the kind of clothing most apt to retain and convey the specific poison, woollen textures are found to be the most dangerous. Haller of Vienna has made experiments on this point. He observes that DARK-coloured materials are more apt to absorb the contagion of typhus, and to convey it to other individuals, than those which are LIGHT-coloured. He found that, among troops wearing dark-coloured uniforms, it more frequently happened that new cases of *typhus* entered the hospital after a convalescent patient joined his corps

than those wearing light or white uniforms. The fact has been often observed, that in dissecting rooms dark clothes acquired the cadaveric odour sooner, and were deprived of it less readily than light ones; and Haller ascertained by experiments that the absorption of odours is regulated by the laws which govern the absorption of light. He also found that the specific poison of typhus fever is lighter than atmospheric air. When the under storeys of an hospital were filled with typhus patients, those in the upper storeys were always observed to become infected when there was a communication between the air of the two storeys. On the other hand, when only the upper storeys contained cases of typhus, the patients in the under part of the house enjoyed perfect immunity (*Edin. Med. and Surg. Journal*, 1853). If the poison be very concentrated, the length of the period of exposure sufficient to contract the disease is very brief—not more than a few minutes; and the latent period during which it remains in the body, without betraying its presence in any way, has been variously estimated. There is, in fact, no fixed period of incubation. It varies in different cases; in a large proportion, it is about twelve days; in exceptional cases it is longer; but it rarely, if ever, exceeds three weeks; and in many cases (one-third or more) it is less than twelve days, and occasionally there is scarcely any latent period, the symptoms commencing almost at the instant of exposure to the poison (MURCHISON, l. c.) These results have been confirmed by Virchow and also by Obermeier in the Berlin epidemic of 1867–1868 and 1873. In the extremely short incubation-period (such as is related by Dr. T. J. MacLagan of Dundee) extremely susceptible persons are generally conscious of the peculiar and offensive pungent odour emanating from the beds or bodies of the sick; and say they are sure they have caught the disease, which is generally true; for they are then immediately seized with prostration, nausea, rigors, and headache, followed by the regular development of the disease. Such persons are almost conscious of the moment at which the poison entered their system. On the other hand, the length of time between exposure and attack may be greatly prolonged. In my own case, I was three months in close attendance, making examinations and recording cases, in the fever wards of the Dundee Infirmary, for many hours daily, in cases of typhus fever, before I took the disease. Opinions vary as to the stage of *typhus* at which the poison is most powerful. Some consider it most powerful during the period of eruption; others, during the period of convalescence. Dr. Perry was of this latter opinion; and Dr. Murchison's observations lead him to confirm the opinion of Perry; but he is inclined to think that the disease is really most apt to propagate itself from the end of the first week up to convalescence, when the peculiar *typhous smell* from the skin and lungs is the strongest; and that the body ceases to give off the poison as soon as the fever subsides and the appetite and digestion are restored. During the first week also of typhus there is little danger; and when the patient is removed within this time the disease rarely spreads (MURCHISON, l. c., 2nd Edition, p. 93). Dr. James Russell of Glasgow, thus writes regarding the time when a typhus fever convalescent may be considered to have lost the power of infecting others from the exhalations of his own body; that, *theoretically*, such would be the case

when his various bodily functions were restored to healthy action, and when he has regained some measure of physical strength—not necessarily his full vigour, but sufficient to enable him to walk a few miles with comfort. *Practically* the question is difficult of determination. Dr. Russell's observations are so much to the point that I conclude this account of the pathology of *typhus* with an extract from his Report for 1870 of the City of Glasgow Fever Hospital:—

“Infectious diseases differ one from another in infecting power, as in other characteristics. The virus of small-pox and of scarlet fever is much more tenacious of life than that of *typhus* or of *enteric fever*—a property which belongs more to the infecting medium (whatever it may be) as conveyed during the disease to clothing and other material objects, than to their continued activity in the body. It is hardly possible that *after* an attack of any of these diseases, a thoroughly healthy person can carry about, in his tissues, the germs of disease. They may be in his clothing, but scarcely in the substance of his body in a communicable state. The practical question, therefore, becomes one of disinfecting the ‘belongings’ of the individual. This being done, and the individual being restored to health, so far as he is concerned the disease is ‘stamped out.’ With regard to the *latent period of typhus*, it must be remembered that from the date a person shivers and becomes ill, you must go back for about a week or ten days for the date of infection of *typhus*. The poison has been lying dormant in the system during that period. Cases but rarely occur in which (just as if we had given a dose of poison and marked the hour, and then watched for the appearance of the symptoms of poisoning) we can date such an event as sleeping one night with a fever convalescent, and then note the first fever-symptoms, and so prove a latent period. From various stray observations, however, nine days is about the average latent period of *typhus*; and Dr. Murchison comes to the same conclusion. Again, *typhus* fever patients are with great regularity in the eighth day of their disease when admitted to hospital. All save a fraction of the cases have gone as far as from the sixth to the eighth day. It is quite certain, therefore, that for the date of infection we must go back from the date of appearance of any case of fever in the books of the hospital, or of the Sanitary Office, at least a fortnight; and if we go back from the date of ‘invasion,’ or active outbreak of the disease, we will find the date of infection at least a week previous. On these grounds, no case of fever, arising even where convalescents have returned home within these periods of a fortnight from the date of admission, or a week from the date of invasion, can be ascribed to those convalescents. Of course there is also a reasonable limit in the other direction. *Individual susceptibility* varies, but not to a marked extent. The staff of a fever hospital unfortunately provides abundant material for the accurate determination of these questions. It is remarkable to find the close agreement in the length of time different systems, under similar circumstances, can resist the disease. . . . The nurses usually are attacked in between twenty and thirty days; but if they tide over that period they become acclimatised, so to speak, and may remain secure for two or three months. The facts regarding the nurses are most valuable, as their duties, diet, &c., are all so much alike. They fall into two very distinct classes. One class, numbering 21, withstood the *typhus* poison for periods ranging from ten days to thirty-seven days, giving an average of twenty-five days. The other class, numbering 5, withstood the *typhus* fever poison,

in exactly the same circumstances as the others, for periods ranging from 47 to 118 days, and giving an average of eighty-four days. The scrubbers furnish data scarcely so pure, as, although their duties do not lead them into close contact with the patients, still they are employed occasionally as substitutes, when they are as much exposed as the nurses, yet they very rarely live for sixteen hours out of the twenty-four in a fever atmosphere, as nurses do. Their average period of resistance is consequently much longer—forty-nine days; and looking at the individual periods with the knowledge of the extent to which each scrubber had been employed as a nurse's substitute, there is an evident relative approach to the nurses' shorter period. The only scrubber who was never, or almost never, employed in any other way did not fall ill for ninety-five days. The gatekeeper and assistant medical officer (who much resemble the nurses in the degree of exposure) resembled them also in their period of resistance—viz., twenty-eight days and twenty-seven days respectively. It is curious to note that the vanman carried typhus patients (about 1500 of them) in his arms out of their houses to his van, and from thence to the ward, for two years before taking ill. The domestic servant went more or less about the wards for two years before seizure, and was then caught from lending friendly aid to an old nurse who required help with her patients. The matron, who spends some time every day in and about the wards, but without contact with the patients, has not been infected after five years' exposure, though she has never had *typhus*. The storekeeper has been at his post for five years, in daily contact with the nurses when getting their provisions, &c., and still retains his health. All these facts concur in proving, (1.) that where attention is paid to personal and general cleanliness *typhus* does not carry far, so to speak, through the atmosphere, and is not portable; (2.) close approach to, and contact with, the infected individual and his dirty belongings lead with great certainty, even in the best sanitary circumstances, and in healthy and well-fed people, to an attack at the end of about four weeks in the majority of cases, but not in a few until the lapse even of some months; (3.) that individual insusceptibility does not exist, except that which is conferred by a previous attack. As an interesting contrast with our experience of typhus, I may say that no case of enteric fever has ever arisen either among the staff or among the patients beside whom cases of enteric fever are treated. These latter have, however, in a very few cases caught typhus."

The conditions essential to the propagation of the specific poison of typhus fever are mainly as follow:—(1.) Overcrowding, co-existing with deficient ventilation; (2.) Personal squalor and filthy apparel saturated with cutaneous exhalations; (3.) A deteriorated state of the constitution, such as may result from protracted starvation, continued underfeeding, general destitution, scurvy, and other debilitating causes; (4.) A moderate temperature—dry heat being a powerful disinfectant.

(c.) *The Morbid Anatomy* of cases of typhus fever has been carefully investigated by Gerhard and Pennock, A. P. Steward, John Reid, Thomas Peacock, Sir William Jenner, Felix Jacquot, Barrallier, and Murchison. All are agreed that there is no constant nor characteristic lesion; and the morbid appearances may be summed up generally as,—“A fluid condition of the blood; hyperæmia of the cerebral membranes with increase of intra-cranial fluid; bronchial catarrh and pulmonary hypostasis; soften-

ing of the heart, liver, spleen, and pancreas; hyperæmia and hypertrophy of the kidneys" (MURCHISON, p. 245). The tissue of the heart is "flabby, soft, and easily torn"—independent of the duration of the disease, and the voluntary muscles are softened. The general muscular system is of a dark colour, showing signs of molecular degeneration when examined microscopically. Inflammation of the parotid gland is not uncommon in some epidemics. All the solid organs are more or less hyperæmic, soft, and friable—the spleen especially is soft and pulpy.

The microscopical characters of the blood show a marked deviation from its normal state. Amorphous heaps of red discs replace the normal rouleaus, and the adhesion of the red discs to each other, in the imperfectly-formed rouleaus, is far less complete and long-continued than in healthy blood. The red discs part with their colouring matter more easily, and dissolve more rapidly than they do in their normal state, as shown by the red serosity found in almost every serous cavity, the deep dusky-red hue of the flesh and of every structure in contact with the blood. The blood drawn during life, or found after death in the vessels, is loosely coagulated or absolutely fluid (Sir WM. JENNER). It is also more apt to become putrid, when taken from the body during life, than healthy blood, or than blood in other diseases; and hence the body decomposes speedily, and the maculæ persist after death. The salts are increased, and there is good reason for believing that the unnaturally fluid state in typhus fever results from an abnormal amount of ammonia, possibly derived from the decomposition of urea; and there is evidence (as Drs. Richardson and Murchison have shown) that the blood of typhus fever contains an increased amount of ammonia.

In an uncomplicated case of typhus fever the body loses flesh rapidly, owing not only to diminished ingress of food, but also to increased egress of bodily structures in the form of excretory products. The metamorphosis of tissue, as judged by the urine, is augmented. In an uncomplicated and undoubted case of *typhus fever*, when no medicine whatever was given, "the urine was that of ordinary pyrexia. The water was lessened; the urea was increased one-fifth; the uric acid was in large amount, and spontaneously, or on the addition of an acid, deposited. The chlorides were entirely absent; there was no diarrhoea or sweating; the sulphuric acid was rather high; the phosphoric acid was not determined. The free acidity was very slight; and (differing from many pyrexia) the pigment and extractive matters were throughout in small amount. The urea continued large, and the chloride of sodium small in amount, and for some days after the temperature had fallen to below the normal limit. The excretion of urea was remarkably regular in amount from day to day; for during ten febrile days its range was only 15 grains (1 gramme) below the mean of the ten days, and 20 grains ($1\frac{1}{3}$ gramme) above it. And this took place with great alterations of temperature. It then, as usual, fell during convalescence, and rose again to the healthy standard in three or four days. The chloride of sodium was clearly retained in this case, for there was constipation, and the skin was dry, so that none could have passed off by the intestines or surface. It would seem also that the urine in typhus is much more frequently albuminous than in typhoid fever" (Dr. PARKES, *Urine in Disease*, p. 258). In three cases of typhus fever associated with jaundice (which is extremely rare in typhus cases), Dr.

Murchison examined the urine, which was also jaundiced. There was no reaction on testing for the bile acids; but in two of the cases *tyrosin* and *leucin* were found. In one of these cases the urine was almost devoid of urea. At the autopsies of two of them there was no derangement of the biliary ducts (*Path. Society*, Feb. 3, 1863). The following general pathological conclusions are noted from cases carefully observed by the late Dr. Parkes:—(1.) In spite of the many pints of fluid drank, a small quantity of water left the system by the kidneys and skin, and none at all by the bowels. This retention of water is not peculiar to typhus, and its cause is quite unknown. (2.) The amount of urea was greatly increased. The normal amount of urea excreted by active men on good diet, between twenty and forty years of age, weighing 145 lbs., is 491 grains in twenty-four hours. A boy ill of typhus, aged seventeen, weighing not more than 129 lbs., excreted not less than 532 grains daily, although he was on fever diet, and taking scarcely any nitrogenous food. (3.) The chloride of sodium is excreted in health at the rate of 180 grains daily. In this case of typhus fever it was present only in traces, the amount being too small to be determined. Like the water, this retention is common to the pyrexia. (4.) Metamorphosis of tissue was more active by one-fourth daily.

Symptoms.—(a.) *General Phenomena.*—Typhus fever attacks persons of both sexes, from early infancy to extreme old age, and its advent is generally somewhat sudden. After a longer or shorter duration (generally a few days) of unpleasant sensations—in which general soreness, uneasiness, and fatigue without cause, loss of appetite, and disturbed sleep, are the prominent phenomena—the disease begins and advances gradually. It is not possible in all instances to fix the precise time of commencement of the attack; but in the majority of cases the patient is seized with chilliness, which sometimes amounts to a rigor, usually followed by heat of skin, and occasionally by sweating, pains in the back and limbs, and frontal headache. This headache is a constant symptom, which ceases usually about the tenth day, and always before the fourteenth. During two or three days the chilliness and rigors occur at irregular intervals. The patient alternately hovers over the fire, or desires to move from it; and although the skin at the time may be felt hot and burning, he still lingers near the fireplace, and yet again soon complains of the heat of the room; so that he feels when near the fire hot and oppressed, and when away from it chilly and uncomfortable. Loss of appetite, and more or less thirst, exist from the first; the tongue is white, large, and pale, but is afterwards covered with a yellow-brown fur; and is sometimes tremulous, indicating the early loss of muscular power and control. At the termination of the first, or commencement of the second week, it has a large and swollen appearance, grows dry in the centre, and at the same time its white fur is replaced by pale dirty-brown mucus. The limbs tremble on movement. The bowels may be confined; the urine is scanty and high coloured; and nausea with vomiting are often among the earliest symptoms. If sleep is obtained, it is disturbed by dreams, or by the occurrence every few minutes of sudden starts. It is consequently unrefreshing; and although the patient may have appeared to sleep for hours, yet he feels that he has not slept, and declares that he has never

closed his eyes. The *general appearance* of a typhus fever patient is also characteristic. Bodily weakness becomes extreme; there is a marked sense of depression and exhaustion, so that the patient takes to bed by the second or third, and not unfrequently on the first day. While there is absolute loss of muscular power and control, there is at the same time an amount of great exhaustion, disproportionate by its severity to the muscular action. He lies prostrate on his back. The expression of his countenance betokens weariness. The eyes are dull and heavy, sometimes suffused and injected, or bloodshot, and a dusky flush overspreads the cheeks. There may be a constant tendency to heaviness and drowsiness. The attention cannot be fixed, and the mind ceases to think. A peculiar symptom may now become expressed, to which Sir William Jenner has given more appropriately the name of *coma-vigil*, than to that symptom which Chomel has so named. In the *coma-vigil* of Jenner "the patient lies with his eyes open, evidently awake, but indifferent or insensible to all going on around him." This symptom occurred in one-fifth of the fatal cases observed by him. Giddiness and noise in the ears are amongst the earliest and most loudly complained of symptoms. The debility increases rapidly, so that by the seventh day the patient can rarely leave his bed without some assistance. By this time also the want of control over the muscular movements becomes more decided; the gait is tottering, arms and hands shake when raised, and the tongue trembles when protruded. The impairment of the mental powers manifests itself in a variety of ways. Memory becomes deficient,—the ideas of time are such that it is always supposed to be prolonged. If an event is impressed upon the patient's mind, he will remember it, and it alone. This mazy state of the intellect soon passes into delirium, which becomes manifest first between waking and sleeping, then by night, and finally by day and night. When delirium first sets in, the patient is able to correct himself; if he is made to think, he becomes conscious of his mental error; but this power is soon lost, and delirium becomes predominant. About the tenth day of the disease, sometimes earlier, the headache ceases simultaneously with the commencement of delirium; and if it should continue with delirium, it suggests the probability of some commencing secondary lesion within the cranium, to which special attention must be immediately directed.

Delirium about the ninth or tenth day, or even earlier, becomes decided, sometimes violent, and always unquiet, although the attention may still be fixed by a sharp question. At this time the patient is in some cases violent, and unless restrained leaves his bed to wander about the room. His expression gradually comes to resemble that of a man unwilling to be roused from half-drunken slumbers. It now betokens complete stupidity, oppression, and decided prostration. The complexion, dull and dirty from the first, in the course of the second week becomes absolutely muddy, the conjunctival membranes injected, and the pupils contracted; and the danger of febrile coma, which may supervene, seems very much in proportion to the contraction of the pupil (W. T. GAIRDNER). The face is now often flushed—the flush being dingy and pretty uniform over the whole countenance, but occasionally somewhat more marked on the cheeks than elsewhere.

About the tenth or eleventh day somnolence sets in, which may

gradually pass into stupor, or even coma, and the expression indicates profound prostration. The patient lies on his back unable to turn himself in the slightest degree, and the urine is often passed involuntarily or is retained, requiring the use of the catheter for its withdrawal. The tongue is thickly coated, dry, and dark brown, or even black, appearing as if baked, and perhaps unable to be protruded. The teeth are covered with sordes, the patient is unable to be roused for more than a minute or two, and when so roused he mutters incoherently. The conjunctivæ are intensely injected, and the pupils contracted. The skin is cool and occasionally moist. Miliary vesicles, or sudamina, are sometimes observed about the end of the second week, usually in the groins, at the epigastrium, and under the clavicles. If such vesicles become hard at the summits, then black, and if then the mass drops out as a slough, leaving a circular ulcer, such a vesicular eruption forebodes an unfavourable result (STOKES). The abdomen continues flaccid and indolent throughout. The bowels usually act once or twice a day, the stools being somewhat relaxed.

The *Pulse* from the outset of the disease is quickened, and it increases in rapidity in cases which terminate fatally, ranging from 100 to even 150 in a minute; or, after reaching a certain point, its frequency as gradually subsides till health is restored. Cases in which the pulse is remarkably slow are usually cases in which the prostration becomes extreme. In the milder and uncomplicated cases the maximum rate of the pulse is reached before the eighth day, and continues for two or three days at least, at the maximum rate—commencing to decline gradually about the tenth, eleventh, or twelfth day. In the more severe cases the pulse keeps up very high till the thirteenth or fourteenth day, when, if the disease is about to end favourably, there is a *sudden* and marked fall (say from 120 to 96, or from 100 to 84), indicating a decided crisis (PERRY).

(b.) *The Eruption of Typhus Fever*.—About the fourth to the seventh day, usually on the fourth or fifth day, the characteristic eruption appears on the skin in 95 per cent. of the cases. It consists of—(1.) Distinct spots; (2.) A subcuticular rash.

(1.) The distinct spots are *maculæ*, *measly* or *mulberry spots*, or a *rubeoloid rash*. On the first appearance of this eruption it consists of very slightly elevated spots of a dusky pinkish-red colour, somewhat like the stains of mulberry juice. Each spot is flattened on the surface, irregular in outline, with no well-defined margin, fading insensibly into the hue of the surrounding skin. The spots *disappear* completely on pressure, resuming their distinctive appearances as the pressure of the finger is withdrawn; and they vary in size from a point to three or four lines in diameter. The largest spots appear to be formed by the coalescence of two or more smaller ones; and the shape of the larger spots is the more irregular. After one, two, or three days these spots undergo a marked change. They no longer remain elevated above the surrounding cuticle. Their hue becomes darker and more dingy than at their first appearance. Their margins become more defined, especially on the posterior surface of the body; and when the finger is firmly pressed on them, they grow paler, but do not entirely disappear. Thus they are said “to fade under

pressure;" but they cannot be entirely obliterated, a stain of the cuticle remaining to indicate where they are. A still further change may take place in severe cases. The centres of the spots may become dark purple, unaltered in appearance by the firmest pressure, although their circumferences may fade; or the entire spot may change into a true petechia, becoming of a dusky crimson or purple colour, quite unaffected by pressure, with a well-defined margin, and level with the surface. The spots of such an eruption are generally very numerous, close together, and occasionally almost covering the skin. Sometimes, however, they are very few in number, and situated at some distance from each other, and not to be distinguished at first from the *rose spot* eruption. The *mulberry eruption* usually occupies the trunk and extremities, but is occasionally limited to the trunk, and may now and then be observed to extend to the face. When the eruption is apparent, no fresh spots appear after the first, second, or third day, and each spot remains visible from its first eruption till the whole rash vanishes—that is, till the termination of the disease. When very abundant, the eruption, viewed as a whole, has not an equal depth of colour. Some places are much paler than others, and the spots have a dull appearance, as if seen through the cuticle. A mottled aspect is thus sometimes given to the skin on which the darker spots are seated, and hence,—

(2.) A *Subcuticular rash* has been also described, which is deepest coloured on the most dependent parts of the body. From this circumstance the eruption sometimes resembles *measles* so closely as to be distinguished with difficulty from the eruption in that disease. When the spots on the back are of a much deeper hue than those on the anterior surface of the trunk, the skin is at the same time so much congested at the back, that slight pressure with the finger leaves a white mark, which slowly returns to its dusky-red colour. The eruption of the mulberry rash usually appears from the fifth to the eighth day of this disease, and subsides between the fourteenth and twenty-first days (Sir WILLIAM JENNER). "As a rule, however, the eruption of typhus fever appears on the fourth or fifth day; it may be met with, however, as early as the third, and rarely is delayed as late as the seventh. It comes first on the backs of the wrists, the borders of the axilla, and about the epigastrium; and in many cases it covers the whole trunk, and frequently also the arms and legs" (Dr. GEORGE BUCHANAN). Age seems to exert a considerable influence in modifying the eruption. In 100 typhus patients under fifteen years of age the rash will be absent in 25. In 100 typhus patients between fifteen and twenty-two years of age the rash will be absent in 14. In 100 typhus patients above twenty-two years of age the rash will be always present. The eruption gradually becomes darker in hue, the centres of many of the spots, towards the termination of the second week, are unaffected by pressure, and here and there are to be seen some spots with well-defined outline, quite unalterable in appearance by the firmest pressure of the finger. These are true petechiæ. The posterior surface of the trunk is considerably congested, and the spots are there much darker and less affected by pressure than on the anterior surface. Desquamation of the cuticle is never observed as a consequence of the eruption; but the skin throughout the whole course of typhus

fever is often particularly sensitive, the slightest touch occasioning pain. The spots of typhus fever continue ineffaceably persistent after death.

(c.) *Temperature in typhus fever* gradually and steadily rises till it reaches its highest point—generally on the fourth or fifth evening. The heat of the skin conveys a burning pungent sensation to the hand, the temperature ranging from 102° to 107° Fahr. Dr. Cheyne, of Dublin, has recorded 109° Fahr. as the highest temperature he observed, and a few days before death it fell to 95° Fahr. Differences in the range of temperature are probably due to the varying types of different epidemics; and hence the seeming inaccuracies, or rather discrepancies, in the records of different observers; certain symptoms which accompany a high range of temperature may be more marked and frequent in some epidemics than in others. The type of typhus fever varies as head symptoms, heart symptoms, or symptoms of pulmonary congestion predominate in epidemics; and as the rates of mortality in different epidemics also show. The highest temperature recorded by Dr. T. J. MacLagan was in a girl aged seventeen, with severe head symptoms; and who, for thirty-six hours before death, had complete suppression of urine. Death took place by coma on the fourteenth day; and a few hours before death the thermometer indicated a temperature of 106.4° . In cases which terminate favourably Dr. MacLagan never noted a temperature higher than 105.2° ; and in 13.7 per cent. of Dr. MacLagan's cases the thermometer rose above 104.7° . These were all severe cases. The highest range of temperature occurs in cases which prove fatal by acute head symptoms, and in which there is partial or complete suppression of urine. In Dr. MacLagan's experience, in the Dundee Hospital, in 1865 and 1866, the average maximum range in typhus was 104.3° Fahr.; the highest temperature being 105° Fahr., and the lowest temperature 103° Fahr.

The temperature shows little or no tendency to a morning fall till it reaches its highest point; it then falls somewhat, and does not again reach the same height. On or about the sixth day the morning falls of temperature commence, which characterise the remaining course of uncomplicated cases of typhus fever. These morning falls are occasionally slight, but generally appreciable. If the eruption is delayed beyond its usual time, the maximum of temperature is not reached till a later day than the fifth, and the cases are so much more prolonged. Thus the febrile disturbance, as measured by the thermometer, reaches its height about the time at which the rash is fully developed—i.e., by the fifth day. From observation of fifty-eight cases which recovered, and in which no complications existed, nor disturbing influences prevailed to lower temperature prematurely or abnormally, the diagram (Fig. 84) on the following page may be given as typical of the cases of typhus of that time and place contrasted with the range of temperature in a severe case of typhus fever, as given by Wunderlich. The dotted lines are Wunderlich's records of a severe case. The continuous lines are MacLagan's, typical of an ordinary case of typhus. By several critics the records of Wunderlich in typhus fever, which I have previously taken as my authority and model, have been considered too high; but I believe it will be found that epidemics of typhus fever vary very much as to their type; and that in some epidemics cerebral symptoms run high, while in others their occurrence is more rare.

TYPICAL RANGE OF TEMPERATURE IN A CASE OF TYPHUS FEVER (Wunderlich and MacLagan).

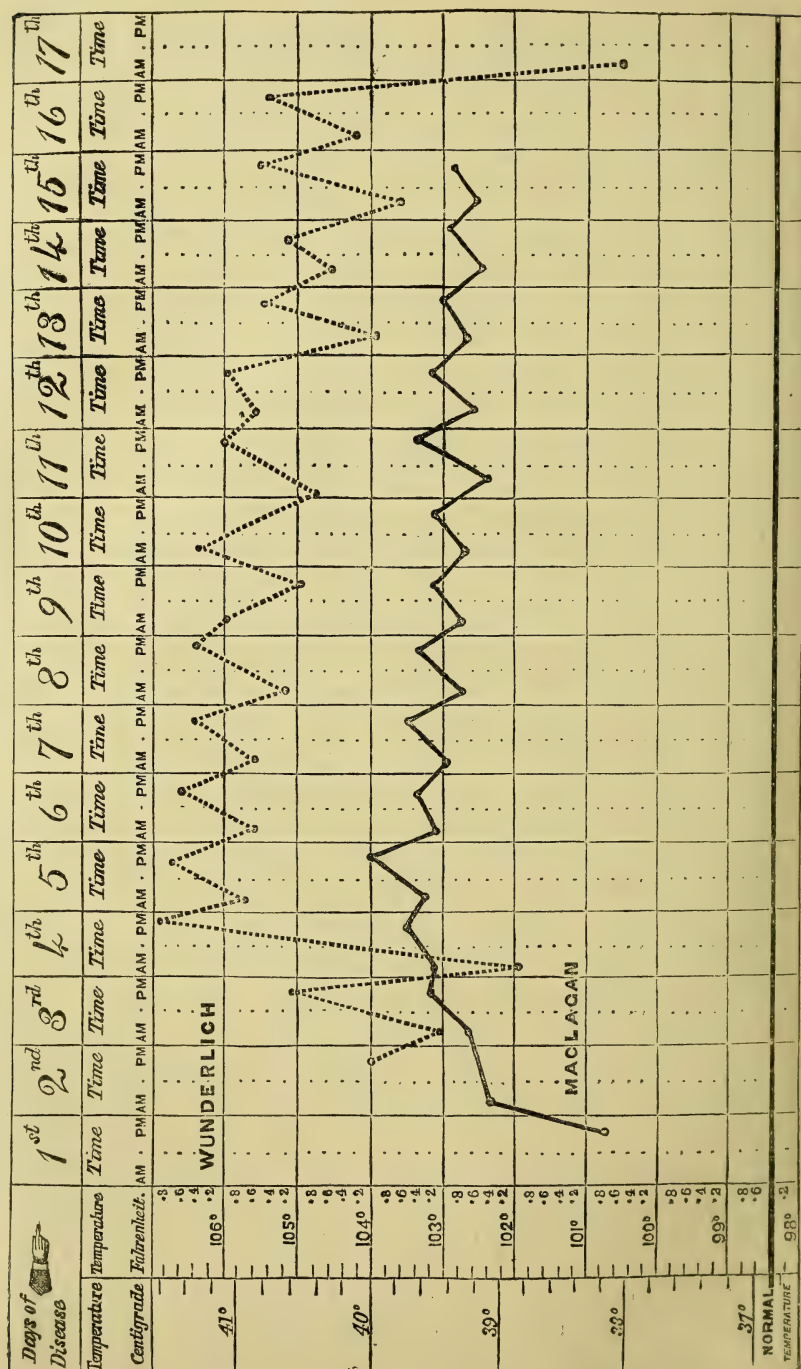


Fig. 84.

Observations are still required as characterising different epidemics in the way already indicated, before we are justified in setting down the records of any one observer as inaccurate or not trustworthy. In the ordinary cases of Dr. Maclagan, and in the severe cases of Wunderlich, the morning temperature was always lower than the evening. The average of morning temperature in Dr. Maclagan's typical cases was $102\cdot7^{\circ}$ Fahr., and evening $103\cdot2^{\circ}$ Fahr., showing a daily average range of *six-tenths* of a degree between morning and evening; in Wunderlich's severe case the average morning temperature is $104\cdot5^{\circ}$, and the evening $105\cdot7^{\circ}$ Fahr., with an average daily range of one degree and one-fifth, Fahr. In both the fever maintains the characteristic continuousness of typhus without interruptions till the crisis.

If the temperature at the commencement before the fourth day does not exceed on any evening $103\cdot5^{\circ}$ Fahr., the fever may be expected to run a mild course; and more especially if the increase of temperature takes place moderately, and is of limited daily duration during the beginning of the second week. On the other hand, in *severe* cases the fever continues with great intensity, at least to the twelfth day, and mostly throughout the whole of the second week. Defervescence rarely takes place before the first half of the third week. In severe cases the temperature may rise above $104\cdot7^{\circ}$ Fahr., and it may reach 106° Fahr. or more on the third or fourth evening (E. LONG FOX). The maximum of temperature is usually attained *before* the ninth day (GRIMSHAW), or *on* the ninth day (WARTER); and according to Grimshaw the extreme height of the thermometer in typhus seldom exceeds 104° , occasionally reaches $104\cdot5^{\circ}$, and may, in exceptional cases, rise to 105° —a conclusion nearly coinciding with the observations of Dr. Perry of Glasgow. But no just conclusion can be drawn from Dr. Grimshaw's records as to what may be the *maximum* temperature in the simple cases of typhus terminating in recovery, of which he gives the history (cases 1, 9, 12, 17, 18, 19, and 21, p. 15, of his *Thermometric Observations*, *Med. Press*, 1866); because none of them were observed before the seventh day, two not till the eighth, and one not till the twelfth. Moreover, his observations seem to indicate only *one* daily observation, which renders them valueless for scientific comparison. The same must be said of Dr. Compton's cases (p. 14 of his *Temperature in Acute Diseases*). That considerable diversity of experience is on record regarding the temperature in typhus, is undoubted; and, as yet, the experience of any one man hardly justifies him in setting down the records of another as "quite erroneous." According to the experience of Dr. George Buchanan, at the London Fever Hospital, the maximum temperature reached is rarely less than $104\cdot9^{\circ}$ or 105° Fahr., and in many cases it reaches $106\cdot5^{\circ}$ to 107° , sometimes even higher. This high maximum is, as a rule (in favourable cases only), attained once or twice throughout the fever, and generally of an evening; the highest morning temperature very rarely exceeds 106° Fahr. The temperature begins to rise at the commencement, and has been observed as high as $103\cdot8^{\circ}$ to $104\cdot9^{\circ}$ Fahr. the first evening; it continues rising till the third day, when it often reaches 105° to 107° Fahr. The maximum is generally attained in the middle of the first week, between the fourth and sixth days—generally on the fourth day (*A System of Medicine*, Vol. I., p. 533).

From these and other observations it is obvious that no just conclusion can be arrived at as to the *maximum* of temperature in typhus fever where observations are not made at least twice daily; and where the observations do not commence so soon as to include, at least, the third day of the disease. Observations delayed till the fifth or sixth day are obviously valueless, so far as a determination of maximum records are concerned. The differences between the morning and the evening temperatures amount, during the fastigium (from the middle of the first to the middle of the second week), rarely to more than 1° Fahr.; and from the middle of the second week to a difference of about 1.5° Fahr. Greater differences happen only temporarily—a character which distinguishes typhus from enteric or typhoid fever, so far as ranges of temperature are concerned. But the difference between typhus fever and enteric or typhoid fever is rendered still more striking by their respective modes of defervescence. In typhus fever the defervescence shows less frequently the gradual remissions seen in enteric or typhoid fever. The defervescence of typhus fever is generally sudden, by crisis—the temperature falling rapidly and continuously until it reaches the normal point, in from twenty-four to forty-eight hours. Sometimes the fall on the thirteenth day is very intense and rapid; but is followed by a rise of three degrees the next day, defervescence extending over a period of several days (E. LONG FOX). This is similar to the experience of Wunderlich, as in the severe case of typhus given in the diagram (Fig. 84, p. 558, *ante*). After the great and sudden fall on the evening of the sixteenth, a rise of at least two degrees followed, and defervescence was extended over five days more (not shown in the diagram). Thus defervescence may sometimes be gradual, commencing from thirteenth to seventeenth day, and extending over ten days; but it differs from enteric fever in the absence of the evening exacerbations. A period of critical perturbation precedes the fall, the temperature rising above the highest point of the day before. The observations of Fox and MacLagan show that the temperature falls *below* the normal during convalescence, and especially in some cases where the previous range has been excessive. This is also the experience of Mr. G. Stevenson Smith, in the Royal Edinburgh Hospital for Sick Children. When the fall is great (3° to 5°) as well as sudden, critical diarrhoea may be severe and serious, as in some of the cases detailed by Dr. Grimshaw; and a sudden fall of temperature to a range much below the normal, other symptoms being serious, is a bad sign, and may forebode death.

(d.) *Relation of Pulse to Temperature.*—There does not seem to be any exact relationship between the pulse and temperature in typhus fever. The pulse may remain low all through the disease, even when the temperature is very high. More commonly, however, there is a tolerable relation between them, the pulse rising as the temperature rises (Dr. E. LONG FOX). Typhus seldom shows the low pulse, with gradually increasing temperature, that is met with during the first week of enteric fever. It is quite clear, however, that observers are not at one as to the relation between pulse and temperature in typhus fever. Dr. J. W. Miller's experience, as Physician to the Dundee Royal Infirmary, shows that the correlation between pulse and temperature is not con-

stant. (See last edition of this Text-book for details.) The average pulse was observed to rise, though very unequally, with each degree of temperature, from 77 with 98° Fahr., to 120 with 105° Fahr. The range of pulse, however, corresponding with any one temperature is very wide, from 50 to 120, for example, with 98° Fahr., and from 84 to 156 with 103° Fahr. In some cases the disparity between the height of the temperature and the frequency of the pulse is very remarkable, and continues for several days, sometimes throughout the whole case.

Influence of Age on Temperature.—Here, again, observers differ. Dr. Maclagan does not think that age has much influence in increasing the range of temperature. The pulse is undoubtedly more frequent in children during the fever as well as during health, but this difference is not apparent after puberty has been reached. In cases which recovered from typhus, under fifteen years of age, the mean temperature in the morning was 102·8°, in the evening 103·8°, the pulse at the same time being 116 and 117. Dr. Perry's experience in Glasgow shows that in severe cases, in children, it is not unusual for the temperature to reach 107°, or even a little higher, from the fifth to the ninth day. According to Dr. Grimshaw's opinion, age has a considerable influence in modifying the thermometric range in typhus. In the young the range is higher than in those advanced in life; but no observations are recorded.

Prognosis.—(a.) *General Indications in Typhus.*—Cases of typhus fever are always anxious cases; and the thermometer enables us to form a definite idea of the actual state of the patient, adding certainty and precision to our knowledge, which, without its aid, would be wanting. But other grounds for prognosis must be taken into account, more especially, (1.) General aspect of the patient, condition of the pupil, injection of the conjunctiva, and expression of the eye; (2.) State of the pulse; (3.) Condition of the heart; (4.) Condition of nervous system; (5.) Condition of respiration—character given to breathing by the expirations—short, hurried, or forcible breathing. A knowledge of the probable course of a case, and of the mode in which death is threatened, is necessary to give significance to thermometric observations in typhus as to prognosis. Coma, asthenia, or a mixture of both, are the usual modes of fatal termination in uncomplicated cases of typhus fever. Thus, death may commence at the brain, or at the heart (see p. 295, *ante*); the former mode being more common in the young, the latter in older people. Pulmonary affections are still more apt to complicate the cases, when asphyxia will then have a share in producing the fatal issue. In severe cases, where the head affection is the source of danger, the thermometer is the earliest certain indicator of the unfavourable nature of the case; and also the earliest indicator of any improvement. If it continues high on seventh or eighth day, with an absence of morning fall on ninth or tenth, the continued persistence of this high temperature is a most unfavourable sign. The pulse is not so good an index of danger as temperature, because, when a patient is restless, excitable, and frequently endeavouring to get out of bed, or performing such like muscular feats, the rapidity of pulse and respiration is apt to be excited. The temperature, on the contrary, is not apt to be so affected. With a high range of temperature from the first, and continuing beyond the ninth day, in

typhus fever, head symptoms are generally severe. The urine ought to be examined for albumen, and although there may be nothing in the general aspect, pulse, or respirations, to suggest more than usual anxiety, the persistent high temperature will give the first warning note that the case will be severe. It will give also indications of commencing defervescence at least three days earlier than any other symptom.

Where nervous symptoms do not predominate, but where the chief source of danger is failure of the heart's action—in which the general symptoms are those of depression rather than of excitement—when the pulse may be even rapid, but the cardiac sounds (instead of yielding a clearly perceptible impulse and audible systole) give a very feeble impulse, and a first sound faint and indistinct;—in such cases the thermometer may not of itself be a good guide to prognosis, but is a reliable index of the decline of the fever. The temperature is then often equable, and not at all high; and, therefore, it is the general aspect, the feeble compressible pulse, the stethoscopic examination of the heart, which indicate danger from another source, namely, from failure of cardiac action. The thermometer, if trusted to alone, may tend to mislead in such cases, by giving a false idea of the state of affairs. It may be low, even to below the normal standard (the result of depression), or it may never reach 100°; yet the danger may be extreme.

From the observations of Wunderlich, Perry, MacLagan, Geo. Buchanan, and E. Long Fox, a summary may be given as to prognosis from the combined value of temperature and pulse in conjunction with other symptoms:—(1.) No two cases are exactly alike, and the temperature varies too much for being alone a safe guide to prognosis or treatment. (2.) The period at which temperature remains continuously stationary varies in duration in proportion to the severity of the attack. In slight cases there is often a temporary remission between the seventh and ninth day, which sometimes seems to usher in a gradual subsidence of the fever. The extent and continued duration of fever distinguishes typhus from all other fevers; while its mode of defervescence distinguishes it from enteric fever. (3.) Cases are slight in which a remission takes place on the fourth day, whilst temperature does not rise much after this period; but in some ordinary cases the acmé of temperature may not be reached till the seventh or eighth day. (4.) In general terms, in ordinary uncomplicated cases the temperature generally attains its maximum about the time of complete development of the eruption; and equability of temperature is a usual characteristic of a mild case. (5.) In cases of great severity the evening temperature will remain at a maximum point until the eleventh or twelfth day, and defervescence will then begin with greater or less rapidity. (6.) In cases the most severe the high evening temperature, with small morning remissions, will persist until the thirteenth or fourteenth day, and even longer; and in cases the most likely from their gravity to attract especial notice, the temperature will seldom diminish before the thirteenth evening. (7.) In uncomplicated cases likely to be fatal, there is no remission of temperature between the seventh and ninth day; the temperature usually keeps more or less high until death, in the second or third week; and, just before death, it may rise to a very extreme point. Wunderlich notes a case in which temperature, *at the time of death*,

rose to above 109° Fahr. (8.) If a high temperature persists into the third week, the prognosis is bad. On the other hand, there are cases in which death is preceded by a temperature of collapse, as in those recorded by Dr. Grimshaw, with a temperature of 95° or 96° Fahr.; and it is a very bad sign if the temperature falls rapidly at any period of the disease, whilst all the other symptoms remain as they were. Such fall may be due to enfeebled cardiac action, or to pulmonary obstruction from deficient oxygenation of the blood, leading to extreme depression. (9.) A high temperature during the first week—*e.g.*, a morning temperature of 104° Fahr., or an evening temperature of 105° Fahr. during the first week; and a morning range of 103·8° Fahr., with an evening one of 104·5° Fahr. after that time, continued into the second week, is the earliest indication of danger from head symptoms. (10.) The highest range of temperature occurs in cases which prove fatal by acute head symptoms, and in which there is partial or complete suppression of urine. (11.) Irregularities and abnormalities in range of temperature sometimes characterise severe cases, and are of specially unfavourable import when occurring in those cases in which a high temperature has been noted at the end of the first week. (12.) A rise of temperature at midnight, and a *continued* absence of a morning fall are (when the range is high) indications of dangerous nervous symptoms. (13.) The thermometer is of no value in estimating danger from probable failure of the heart's action. The state of the heart, as revealed by the stethoscope, is then the best guide to the condition of the patient. (14.) When pulmonary complications exist to so great an extent as to threaten death by asphyxia, the records of the thermometer will not indicate the risk, which can only be measured by the general symptoms, and by stethoscopic examination of the chest.

The disease, if it proves fatal, generally does so from the twelfth to the twentieth day; and before death the prostration increases to an extreme degree. The average duration of Jenner's fatal cases was fourteen days; of Reid's, thirteen days. *Subsultus tendinum*, or involuntary twitchings of the muscles of the face and arms, make their appearance. The face becomes dusky or even livid, the breathing very quick, and the pulse so rapid and feeble that it scarcely can be felt. Some want of resonance of the most depending part of the chest may often be observed at this stage of the disease. The respiratory murmur at the same part becomes muffled, as if heard through a covering, and there is sometimes a little coarse unequal crepitation. The urine, which is *now* secreted in large quantities—from three to four pints daily—is restrained, or passed into bed with the stools involuntarily. The skin is bathed in a profuse sweat, and the temperature is apt to fall below the natural standard. The patient lies on his back unable to move, or he sinks towards the bottom of the bed if his head be in the least elevated. Towards the middle or end of the second week a slough may form on the lower end of the spinal region, or on the region over the posterior spine of the ilium (see Bed Sores, page 311, *ante*). For a day or two before the fatal termination, the condition termed *coma-vigil* may come on. In this condition the patient never sleeps. He lies on his back with his eyelids widely separated, his eyes staring and fixed in vacuity, his mouth partially open, his face pale and expressionless. He is totally incapable of being roused to give a sign of consciousness,

the breathing is often scarcely perceptible, the pulse rapid and feeble, or unable to be felt, the skin cool, perhaps bathed in perspiration. Life is only known to have ceased by the eye losing its little lustre, and the chest ceasing to effect its slow and feeble movements. Sir William Jenner has never seen recovery from this condition. Death generally takes place without any return to consciousness, and by syncope rather than coma. If the disease should terminate in recovery, the improvement in the condition of the patient is frequently sudden. Some time between the thirteenth and the seventeenth day he may fall into a profound quiet sleep, lasting for several hours; and generally after from twelve to twenty-four, or even more hours, he awakes decidedly improved in all respects—indeed, quite another man. At first he is bewildered or confused, and wonders where he is; but he may recognise his attendants and friends, and is conscious for the first time of extreme debility. The complexion is clearer, delirium has disappeared, the pulse has fallen in frequency and gained in strength, the conjunctivæ are no longer injected, the tongue is moist at the edges; there is perhaps a little appetite, the skin is softer and moist, and the spots of eruption are paler. His limbs retain their sensibility; but when he attempts to move them, they seem at first as if separated from the body, so great is the prostration induced by typhus fever (MURCHISON). In a few days the tongue cleans completely, the appetite becomes ravenous and insatiable, and the patient rapidly regains strength. The *duration of the disease* is to be measured by the duration of the eruption; and the average duration of cases that recover is from fourteen to twenty-one days, although, not unfrequently, in very mild cases the fever terminates before the fourteenth day (JENNER). After twenty-one days, local lesions sufficient to cause death were always discovered in fatal cases of *typhus*. In other words, after the twenty-first day death does not then occur *from the fever alone*, as may be the case before the twenty-first day. There are cases of *typhus fever* which appear to die within a limited period after the outset of the illness, from the direct action of the poison on the blood or nervous system, and with the entire absence of local lesion, so that even the skin is not affected with eruption.

But it is the occurrence of certain phenomena, or the presence and succession of certain symptoms, indicating the existence of complications, which are the most important general guides as to danger. These may be arranged in the following summary under three heads, namely:—

(b.) *Combinations of Symptoms and Phenomena which are of extremely Unfavourable Import.*—(1.) A presentiment of death on the part of the patient; (2.) A pulse of 120, which at the same time is soft and compressible, small, fluttering, irregular, intermittent, reduplicate, or imperceptible; (3.) Complete absence of cardiac impulse, and an audible systolic sound; (4.) An excited or thumping action of the heart, associated with a very feeble radial pulse; (5.) Hurried respiration, whether “cerebral” or due to pulmonary lesion; (6.) Sleeplessness, associated with delirium, both of which are persistent; (7.) Severity of cerebral symptoms, and these symptoms coming on early, with no remission of temperature, between seventh and ninth day, the temperature keeping more or less high till death in second or third week; (8.) The occurrence of complete “coma-

vigil;" (9.) Extreme contraction of the pupil; (10.) Extreme prostration occurring early; (11.) Muscular tremor, picking and catching at the bed-clothes, subsultus, and spasmodic twitchings of the muscles of the face; (12.) Urgent and protracted hiccup; (13.) Rigidity of the muscles of the limbs, and squinting; (14.) Relaxation of the sphincters before the tenth day; (15.) Retention of urine; (16.) Tympanites, with extreme nervous prostration; (17.) A dry, brown, hard, retracted, tremulous tongue; (18.) The more abundant and darker the eruption the greater the danger and severity of the case; (19.) Great lividity of the face and extremities, and a dusky erythematous condition of the skin on the dependent parts of the body; (20.) Continuous profuse sweating, coldness of the surface, cold breath, and a rapid weak pulse; (21.) A sudden diminution in the amount of the excretion of urea; (22.) The occurrence of blood or albumen in the urine before the tenth day, especially when associated with casts of the uriniferous tubes; (23.) High temperature in third week; (24.) Pulmonary hypostasis and bronchitis, pneumonia, gangrene of the lungs, convulsions, pyæmia, erysipelas, parotid swellings, inflammatory swellings, bed sores, gangrene, renal disease, scurvy, the gouty diathesis.

(c.) *Combinations of Symptoms or Phenomena which may be regarded as of Favourable Import.*—(1.) A sudden fall in the frequency of the pulse; (2.) When a patient, after lying for days on his back, helpless and motionless, manages to turn himself round and sleep on his side, or if he is able to draw up his leg and rest it on the foot in the flexed position in the bed; (3.) Cases without rash, or in which the rash is scanty; (4.) When the excretion and elimination of urea and uric acid continue free and copious; (5.) Sudden cessation at the end of the second week of several of the unfavourable symptoms and phenomena; (6.) Diminution of the rapidity and increase in the strength of the pulse; (7.) A slight return of appetite, while the tongue becomes clean and moist at the edges; (8.) A diminution of the dusky tinge of the face, a less stupid appearance of the countenance, and a less injected state of the conjunctivæ, with signs of returning intelligence.

(d.) *Modes of Fatal Termination.*—(1.) Death during the primary fever may occur from syncope or from coma. In the former case the heart's action is enfeebled from paralysis or disease of its muscular tissue. In the mode of death by coma, the blood has undergone such modifications as render it incapable of supporting the changes essential to existence. Its contamination seems mainly due to the admixture of urea and other products of the retrograde metamorphoses of tissue, and from the diminution and destruction or solution of its red corpuscles; (2.) Death is for the most part due to a combination of syncope and coma; and, as a rule, the patient is quite unconscious for a considerable time prior to death; (3.) Death may occur from one of the many complications which happen before or after the cessation of the primary fever. Of these the following may be noticed as influencing prognosis:—

(e.) *Occurrence of Convulsions and Cerebral or Head Symptoms.*—The most formidable, and fortunately a most rare complication in *typhus fever* (1 in every 100), is the occurrence of convulsions. When they do occur, the case almost invariably proves fatal, unless in a patient who has suffered from

epilepsy. There are rarely any cerebral lesions sufficient to account for them; and their occurrence may be fairly referable, in the present state of our knowledge, to the morbid condition of the blood in *typhus* fever, and the altered condition of the nervous system which ensues. With rare exceptions they have an uræmic origin. In most cases there is albuminuria; and the kidneys are found diseased after death. Convulsions do not usually appear before the middle or the end of the second week. The cerebral complications are generally attended with what are commonly called "head or nervous symptoms," and are preceded by long-continued high temperatures. Sir William Jenner emphatically calls attention to the fact, that the continuance of the headache complained of spontaneously after the commencement of delirium is generally indicative of increased vascular action within the cranium. It may also be noticed that the headache which precedes the delirium is often in such cases of a very severe and constant kind, the face being sometimes pale and sometimes red, and greatly expressive of the distress the patient suffers. The eye, haggard or brilliant, with its conjunctiva injected and its pupil contracted, is painfully sensible to the light, and is therefore generally closed. The least noise is insupportable, and the patient is troubled with noise in his ears. His temper is altered, and his answers short and fretful. This condition is that of increased excitement, but not as yet of delirium, which supervenes at periods varying from two to ten days. "There is no relation between the headache and delirium; and in most cases the headache has ceased before the commencement of delirium,—a feature of no small importance as regards diagnosis from cerebral inflammation" (MURCHISON, l. c., p. 160, 2nd Edition). It does not usually come on until the end of the first or beginning of the second week, and continues more or less till death or convalescence. Delirium may assume every character,—joyous or melancholy, furious or tranquil; and in some cases the patient wanders from subject to subject, while in others he incessantly recurs to the same theme, and even to the same few words. In others, though the cases are few, the disease assumes every character of insanity; and, if permitted, the patient presents the extraordinary spectacle of being able, in typhus fever, notwithstanding prostration, to walk about the wards. This acute form of delirium is apt to be followed by profound prostration or fatal collapse; and the commencement of cerebral effusion is indicated—by the active delirium changing into a low muttering (*typhomania*); by the patient no longer requiring restraint; by his muscles becoming spasmodically affected with slight twitchings or *subsultus tendinum* (showing how rapidly the nervous power is exhausted, and how feebly supplied); also by the pupil of the eye becoming expanded or contracted; by the fæces being passed involuntarily; by the urine being retained; and by the rapid grouping of those other symptoms so happily described by Shakespeare as "the stony coldness of the feet creeping upward and upward," "the babble of green fields;" and the "fumbling of the bed-clothes"—all indicative of approaching death. Intra-cranial serosity is generally decidedly increased; and hæmorrhage into the arachnoid occurred in one-eighth of Sir William Jenner's fatal cases. When the patient recovers, however, from the stage of cerebral complications, the appetite improves, the pulse becomes fuller

and steadier, the countenance more tranquil, the mind firmer, and the sleep natural, till at last convalescence is fully established.

The respiratory movements in typhus are influenced by this cerebral condition. In the first week they may not exceed twenty or twenty-four in the minute; but when delirium supervenes and the pulse increases in frequency, they often rise to thirty or more, without any pulmonary lesion. In cases of great cerebral disturbance the respirations become sighing, irregular, spasmodic or jerking, and then coma is apt to supervene. The "nervous respiration" of Dr. Corrigan (or what is sometimes also called "cerebral respiration") is denoted by a blowing or hissing sound in breathing, the lips being kept closed; the cheeks are distended, the nostrils dilate with each expiration, and the breath is forced through the closed lips with a puffing, blowing noise. Such breathing is irregular, a long pause being followed by a deep inspiration, and subsequently by short respirations in rapid succession. In some cases the action is entirely diaphragmatic, the respiratory muscles of the trunk being paralysed (MURCHISON). The air expired has a disagreeable odour, most marked in the advanced stage of severe cases. It resembles the *typhus odour* exhaled by the skin, which has been compared to the "odour of rotten straw," to the "smell of mice, deer, and certain reptiles," or to the smell of "the leaves of rue when rubbed between the fingers." By some it is spoken of as "pungent, ammoniacal, and offensive;" but it must not be confounded with the smell from urine passed in bed. It seems to be, however, a smell *sui generis*, as Murchison very justly observes; and nurses experienced in typhus fever are quite familiar with it, and are able to distinguish cases of typhus fever by this peculiar typhus odour alone, which is always strongest in damp weather, and when the ventilation is bad. It is highly probable that the typhus poison is contained in this odoriferous substance. The expired air of typhus patients contains a smaller quantity of carbonic acid and a larger amount of ammonia (MURCHISON, pp. 134-137).

(f.) *Secondary Pulmonic Complications* are not uncommon in *typhus fever*, in the form of *pneumonia*, and so far influence prognosis. The congestion of the blood in the posterior parts of the lungs may give a tendency to this; and its presence may be suspected from the livid expression of the face, the existence of cough with rusty expectoration, the diagnosis being confirmed by the usual auscultatory means. It is seldom that pain is complained of. The part of the lungs affected for the most part is that which rests against the hollow of the fourth, fifth, and sixth ribs, between their tubercles and angles, the position of the patient appearing to determine the place of consolidation. Such consolidations are not to be regarded as analogous to what we see in an ordinary pneumonia, occurring in an otherwise healthy person. The incubation of the lesion is latent, the symptoms are masked, and the nature of the complication is only to be recognised by careful physical examination and determination of temperature, which suddenly rises and continues high. The full expression of the morbid state is often for some time undecided; the exudation, being of a serous nature, is slow to solidify on the one hand, and yet the symptoms of resolution do not appear on the other. Dr. Hudson, of Dublin, attaches some importance to a certain tympanitic

resonance, which becomes manifest over the diseased lung, as a sign of the existence of the pulmonic lesion. He describes it as "a tympanitic clearness over the solidified lung, without air being present in the pleura." Dr. Lyons explains this abnormal clearness as the result of the increased pressure of the respiratory column of air in the permeable portions of the pulmonary lobules, which become expanded beyond their natural volume, and thus a condition of temporary emphysema is produced, which yields a clear sound on percussion (Dr. STOKES, in *Medical Times and Gazette*, May 26, 1855). In some cases of pulmonary lesions there appears to be a combination of circumstances which leads to a fluid or purulent state of the diseased part, resembling the third stage of pneumonia as described by Laennec. The conditions which lead to this form may be stated to be,—(1.) A sudden exudation and abundance of fluid matter; (2.) A great amount of tissue involved; (3.) Diminished vascularity, and consequent (4.) Abeyance of absorption, tending to (5.) Increased fluidity of the diseased part; (6.) Breaking up or solution of the young and growing elements. A lung in this condition seems to have passed, as it were, at once into this state, without any well-marked hepatisation. *Gangrene of the Pulmonary Tissue* is by far the most formidable of the thoracic secondary lesions of *typhus fever*. The hepatisation of the lung is not obscure, but the consolidation is at once sudden, complete, and extensive, involving perhaps the greater part of a lung, and coming on without any marked physical signs different from what are to be heard in the simple congestion of *typhus*. A gangrenous cavity forms in the substance of the solidified mass, and is only indicated by the fetid expectoration and the accompanying physical signs of a cavity. Large eschars are apt to form towards the pleural surface, surrounded with well-defined lines of demarcation where separation of the slough proceeds. In this gangrenous slough every element of the pulmonary tissue becomes disintegrated, almost perfectly liquescent. Sometimes it happens that the gangrenous cavity does not communicate with the bronchial tubes; then the morbid state is difficult to diagnose, and its existence is often unknown till after death. With such physical signs of gangrene, the expression of the countenance of the patient is often highly suggestive. It suddenly becomes small, pinched, contracted, ghastly, miserable, and death-like. The eyes are sunk and void of lustre; and, along with languor, the patient feels nausea, and sometimes vomits. There may be several distinct gangrenous centres, as if the lesion had been, from the first, disseminated or lobular.

(g.) *Secondary Cardiac Lesion* assumes the form which Dr. Stokes has called "typhus softening of the heart." The muscles of the larynx and the circular muscles of the trachea are sometimes similarly affected, as well as the involuntary muscles generally. This complication has for the most part occurred when there was a great amount of secondary bronchial disease. The wasting of the involuntary muscles is always great in typhus. In the heart it is more obvious than in the arterial or systemic portion. The cardiac phenomena (adynamic) of typhus are chiefly indicated by a diminution of the impulse, and an impairment or loss of the first sound—the impulse diminishing progressively from the fifth or sixth day to the termination of the disease—while the systolic sound becomes

daily more feeble or quite inaudible, leaving the second sound clear and distinct. The poison, however, does not necessarily make itself manifest through all the series of local secondary affections already referred to. Thus, in one year or epidemic the lungs will be attacked in every case; in others the bronchial membranes or the membranes of the brain; while in other epidemics, parotid swellings, symptomatic parotitis, and inflammation of the ear will prevail. At other times such attacks will be rare,—the exception, and not the rule of the disease.

General Indications for Treatment.—These have been already fully stated at page 302 (and *typhus* and *enteric* fevers are not exceptions). They may be summed up as a combination of measures to *reduce excessive heat*, to insure *proper excretion*, and to *act on the semi-paralysed nerves*; and, “every remedial agent which shall be found to promote the elimination of urea, without increasing the destructive metamorphosis of tissue, will deserve a trial in typhus” (MURCHISON l. c., p. 268). To reduce heat and to regulate elimination are but secondary indications in the treatment of *typhus* fever, compared with the influence which must be exercised over the nervous system; and one of the greatest objects of therapeutics at the present day is to find substances which will act on the nerves and the blood, and restore them in some way to their normal action.

Special Indications for Treatment.—Our objects in the treatment of typhus fever should be,—(1.) To neutralise the poison and to correct the morbid state of the blood; (2.) To promote elimination of the poison and the products of the destructive metamorphosis of tissue; (2.) To reduce the temperature; (4.) To sustain the vital powers, and to obviate the tendency to death; (5.) To relieve the distressing symptoms; and, (6.) To avert and subdue local complications (MURCHISON, p. 265).

1. In the belief that the morbid condition of the blood in typhus fever may be due to the presence of ammonia in some as yet unknown combination, the use of mineral acids has been recommended by many physicians. Murchison considers their beneficial effects in typhus as undoubted, and in this opinion he is confirmed by the experience of Huss of Stockholm, Haller of Vienna, of Mackenzie, Chambers, and Richardson, in this country. Huss recommended *dilute phosphoric acid* in doses of twenty-five to forty drops every second hour, believing that the phosphorus exerts a special influence on the central organs of the nervous system. But in the advanced stage, and especially if sweating, numerous petechiæ, or ecchymoses be present, he has recourse to *dilute sulphuric acid* in doses of fifteen to twenty drops every hour or every second hour. *Dilute hydrochloric acid* is preferred by Drs. Murchison, Richardson, Mackenzie, and Chambers. It may be given to the extent of half a drachm of the dilute acid, mixed with a like quantity of the *tincture of syrup of orange* every three hours. Dr. A. P. Stewart has used with advantage the *tinctura perchloridi ferri*, in doses of thirty minims every three hours. Dr. Murchison recommends *nitro-muriatic acid*. He prescribes twenty minims of *hydrochloric acid* with ten minims of *nitric acid* every three hours, each dose being diluted with the patient's drink, such as barley water sweetened with *syrup of ginger* and flavoured with *lemon peel*. But if the “typhoid state” (see pages 78 and 29, *ante*) is

developed in a marked manner, *dilute sulphuric acid*, in doses of fifteen to twenty minims every three hours, in combination with *ether*, and small doses of *quinine*, are to be had recourse to, as in either of the following formulæ:—

R. Acid. Hydrochlor. dil., ℥xx.; Acid. Nit. dil. ℥x.; Spt. Æther-Nit., ℥lx.; Liquor. Cinchonæ, ℥xxx.; Decoc. Scopar. comp., ℥i.; *misce*. A draught so composed may be administered every third hour.

Or, R. Quiniæ Sulph., gr. $\frac{1}{2}$; Acid. Sulph. dil., ℥xx. ad ℥xxx.; Æther. Sulph. ℥xv. ad ℥xxx.; Syrup Aurant., ℥lx.; Decoc. Scopar. comp., ℥i.; *misce*. A draught so composed may be administered every third or fourth hour.

When the acids are cautiously administered in smaller doses, in conjunction with a few minims of *solution of muriate of morphia* (if the bowels be irritable), sweetened with *syrup of orange peel* and diluted with water, the draught is generally relished, and the tongue from being dry, and hard, and brown, becomes moist and clean (PERRY).

2. Perhaps the best general method to insure proper excretion is to supply the system with abundance of diluents containing *alkaline salts*, which are not given in the food, and to maintain the action of the kidneys, the bowels, and the skin. Plenty of pure drinking water tends to increase the flow of urine, and so helps to wash away the products of metamorphosis. *Chloride of sodium*, the *alkaline salts of potash*, and probably also those of *soda*, tend to aid the formation of urea and its elimination. Dr. Murchison has been in the habit of ordering large quantities of salt to be mixed with the patient's beef-tea. In most cases it was greatly relished, and apparently beneficial.

Purgatives tend to insure a proper excretion, probably by removing from the blood some of the abnormal products formed in fever. The great relief which sometimes follows their use, as well as the fall of temperature, seems to show this. Where there is retention of urea, they aid its elimination, because we know that urea passes off sometimes by the mucous membrane of the stomach and bowels. The patient should be allowed to drink freely of water; and five grains of the *nitrate of potash*, or a small dose of the *tincture of digitalis* (℥iv. to ℥x.), may be given with each dose of the *nitro-muriatic acid* already mentioned. Dr. Murchison recommends *nitre whey*, prepared by boiling ℥ii. of *nitre* in a pint of milk, and straining; or a drink prepared by dissolving ℥i. to ℥ii. of the *bitartrate of potash* in a pint of boiling water, flavoured with *lemon peel* and *sugar*; but if the patient be very prostrate, or if the bowels be relaxed, 15 minims of the *spirit of nitrous ether* is to be substituted for the *nitrate of potash*.

Tea and *coffee* have been recommended in the stupor of typhus; and it is probable that their good effects are due to their power of eliminating the urea already formed in the blood (Dr. PARKES). The *coffee* may be given as an *extract*, or as a *strong infusion of the powdered berry* made in the ordinary way. *Tea* has been recommended as an *infusion of the green tea leaf*. As beverages or common drinks in fever, both *tea* and *coffee* have been found to relieve the headache, the pulse becoming fuller and stronger under their use. Böcker, L. Lehmann, and Hammond, all agree in

showing that in health they greatly lessen the urea (PARKES *On the Urine*, p. 76). To restrain excessive metamorphosis it has been proposed to administer *coca*—the dried leaves of the *erythroxylon coca*—which, when indulged in by a healthy person, prevents for a time the feeling of fatigue, and diminishes the amount of urea (*to be?*) excreted. Hence its possible value as a therapeutic agent (M'BEAN, *Brit. Med. Journ.*, March 10, 1877, p. 291). A drachm to a drachm and a half of the *tincture* is to be given in water every four hours; or an *infusion* of the leaves may be taken when thirsty.

The action of the bowels is to be maintained by emetics and laxatives. In the first instance, if the patient is seen early—*i.e.*, before the sixth day—an emetic of *ipecacuanha* (one scruple), and of *antimony* (one grain), or of *carbonate of ammonia* (two scruples), in place of the *antimony*, is to be administered. If the bowels remain confined after the emetic, a mild laxative of *rhubarb* and *calomel*, or of *castor-oil*, is to be given; and failing these, or in place of them, a simple enema is to be administered (MURCHISON, p. 277, 2nd Ed.). The advantages of emetics are, that they relieve the patient to some extent by mitigating or removing headache and general pains. They also reduce the temperature, abate thirst, and quiet gastric disturbance. Emetics, however, are contra-indicated if the patients are unusually weak, or if the disease has advanced beyond the first week. Laxatives and enemata, however, ought to be repeated daily, if required, so as to secure a motion of the bowels once a day. In this respect the treatment is different from the treatment which ought to obtain in enteric fever. Excrementitious matters in the intestines must be removed by gentle aperients. The dark offensive matters accumulated in the intestinal canal in typhus fever may have a secondary deleterious effect on the system if they are allowed to remain. Purging, however, is to be avoided, and *fresh-made compound rhubarb pill mass*, which *tends to stimulate the peristaltic action of the intestines*, is as good a medicine as can be given, followed, if necessary, or alternated, by a small dose of *castor-oil*, or by a simple enema.

Diaphoresis is not to be encouraged beyond the insensible transpiration of the skin, to remove which the wholesome detergent of tepid water sponging is most beneficial. It ought to be used twice or three times daily, and quantities of *Condy's fluid* or of *muratic acid* (ʒi. ad Oj.) may be mixed with the tepid water (MURCHISON). The measure is a good one in a hygienic point of view; and it contributes—

3. To reduce temperature, the external application of cold water, as was once practised to an extreme degree by Currie (and originally recommended by Dr. Robert Jackson), has been again recently advocated. In health such an application as that of cold water has a great effect in reducing temperature, and tends to increase metamorphosis of tissue (LEHMANN, SANDERSON). Its therapeutic uses have been fully considered at p. 302, *ante*.

4. The vital powers are to be sustained by food in the first instance. For this purpose, nourishment ought to be given often, and at stated intervals—at least once every three or four hours after the fourth day of the fever. Even if the patient is asleep, or seems to be so, he must be roused at these stated intervals (not oftener) to take his food or his

stimulants. But if, towards the period of the crisis, the patient appears to be in a sound sleep, he ought not to be disturbed. The indications for treatment just described apply to the earlier stages of the fever, up till about the fourteenth day. Beef-tea, broths, meat-juice, bread pudding, arrow-root, jellies, milk, eggs, and alcoholic fluids, are the foods on which the typhus fever patient must be sustained. *Alcohol* in small quantities, as well as *tea*, *coffee*, *lemonade*, *soda-water*, water poured from standing over oatmeal, and taken cold or after boiling and then cooling, and other fluids, have a directly stimulant action on the nervous system and on the organs of circulation; at the same time, alcoholic drinks diminish the metamorphosis of the tissue-elements. Few remedies, however, require more discrimination in their use; and the following guides for their administration are compiled from the careful observations of Dr. Murchison (l. c., p. 288, 2nd Ed.):—(1.) Patients under twenty years of age do best without alcohol; but most patients over forty are benefited by alcohol from the commencement of the second week of the illness, or earlier; while persons of intemperate habits require alcohol earlier, and in greater quantity than others. (2.) In individual cases the indications for alcoholic stimulants are mainly derived from the state of the organs of circulation; and the profession is indebted to Dr. Stokes (1839) for pointing out the importance of cardiac and radial pulses as guides for the use of alcohol in fever. These indications are,—(a.) Extreme softness and compressibility of the pulse. An undulating, irregular, intermitting, or imperceptible pulse more imperatively demands stimulants than a merely rapid pulse. So also an abnormally slow pulse—*e.g.*, 40 to 60—is a stronger indication for stimulation than a quick pulse. (b.) When the cardiac impulse becomes weak, and when the first sound is impaired or absent, a liberal allowance of stimulants is demanded; and in every case where there are doubts as to the propriety of giving stimulants, the heart must be examined with the hand and with the stethoscope, because the state of the pulse alone is not sufficient to judge from. The impulse may be found to diminish progressively from the fifth or sixth day to the termination of the disease; and for several days prior to death or recovery it may be entirely absent. The systolic sound of the heart becomes daily more feeble, and ultimately may be quite inaudible, leaving the second sound clear and distinct; and before the first sound is altogether lost, it may become so short that it is difficult to distinguish it from the second sound. If the action of the heart be rapid, its sounds may thus come to resemble closely those of the *fœtus in utero*. A violently excited heart all through the disease, with cold surface, cold breath, and feeble pulse, demands wine from the first; but even with its judicious use the prognosis in such cases is extremely doubtful (STOKES, GRAVES, MURCHISON). Other indications for stimulants may be stated as follow:—(c.) If by raising the patient to his semi-erect position a tendency to syncope is induced, or great prostration is manifest, with diminished strength and volume of the pulse, then stimulation must be commenced. (3.) The darker and more copious the eruption, the more is the necessity for stimulants, especially if petechiæ are numerous. (4.) Profuse perspiration, with no improvement in the general symptoms, requires an increased supply of stimulants; but a burning dry skin is in

itself an indication against alcohol. Coldness of the extremities is an indication for alcohol, especially when at the same time the temperature of the trunk is considerably elevated. (5.) Stupor, low delirium, subsultus, involuntary evacuations,—symptoms generally of the typhoid state, are indications for the liberal administration of alcohol; but the propriety of giving stimulants in delirium depends on the state of the pulse. If, on the trial of stimulants, the patient becomes tranquil, they do good, and may be continued; if the reverse, their use must be suspended. (6.) A dry brown tongue is an indication for wine or brandy; and if it becomes clean and moist at the edges under the use of either, such stimulation is beneficial. (7.) Complications, as a rule, increase the necessity for stimulation; and large quantities of stimulants are called for if *pyæmia*, *erysipelas*, *bronchitis*, *pulmonary hypostasis*, *pneumonia*, *inflammatory swellings*, *bed sores*, or *local gangrene* should supervene. (8.) Alcohol, as a rule, is contra-indicated if there be severe darting or throbbing headache, or acute noisy delirium, especially when these symptoms co-exist with great heat and dryness of the skin, flushing of the face, suffusion of the eyes, and little or no impairment of the cardiac and radial pulse. When alcohol is given under such circumstances it should be restricted to the intervals of the paroxysms of delirium. (9.) Scanty urine of low specific gravity, containing little urea or much albumen, and suppression of urine, are in themselves indications against the use of alcohol.

The effects of alcoholic stimulation require to be most carefully watched throughout the whole period of their administration. Four ounces of wine in the twenty-four hours is enough to begin with; for if the blood be overloaded with the products of alcoholic ingestion, further alcoholic stimulation will lead to increased contamination, and it is rare that more than eight ounces of brandy in twenty-four hours are necessary. There are differences in the demand for stimuli in the typhus of different countries, and in the fever of different epidemics. Dr. Wood tells us that in America cases requiring wine or brandy are extremely rare. Dr. Stokes says that the *typhus* in Ireland demands large quantities of wine. In Scotland, also, wine is the great mainstay in the treatment of *typhus fever*, requiring often to be administered largely. Port, sherry, marsala, madeira, brandy, gin, or whisky, possess no peculiar advantages apart from the alcohol contained in each. Spirits contain 50 to 60 per cent. of alcohol, sherry and port from 17 to 24 per cent., and malt liquors from 6 to 8 per cent. Two fluid ounces of spirit will thus be equal to five or six of wine, and spirits ought to be given diluted; and if the prostration is great, and when the skin is cold and covered with perspiration, the best stimulant is brandy or whisky punch, given as hot as it can be taken, in small quantities at a time, frequently repeated. In urgent cases stimulants ought to be given every hour; and, as a rule, a larger quantity will be required during the night and early morning than in the daytime, for it is usually towards morning that temperature tends to get low, and the vital powers are at their lowest ebb (MURCHISON). At the same time it must ever be remembered, as Sir Wm. Jenner justly observes, that “in no disease is the advantage of refraining from meddling more clearly displayed than in typhus fever; and in no disease is the prompt use of powerful remedies more clearly indicated. It is in deter-

mining when to act, and when to do nothing, that the skill of the physician as a curer of disease, in the case of fever, is shown. Interfere by depletion or stimulation when nothing should be done, and the patient is lost, who, if it had not been for you, would have been safe. Refrain from depletion or withhold stimulants when the one or the other is required, and the patient sinks into that grave from which judicious treatment might have saved him."

A large well-ventilated apartment, fresh air, a cool, but not a cold atmosphere, quiet, abstinence from solids, and a free supply of water, milk and water, coffee, weak broth, beef-tea, according to the discretion of the physician, are the conditions and remedies on which a large majority of cases will recover. But the patient must be constantly and carefully watched. There is no disease where the attentions of a well-instructed nurse are more demanded; and there is no class of patients in hospitals so apt to be neglected by the attendants, especially as to the regular administration of the remedies prescribed. It is not uncommon to find that the wine allotted for the day has been administered at a draught, when it ought to have been given in small quantities at regular intervals, with care and watchfulness. How often do we see almost hopeless cases recover under the careful nursing of an intelligent person, regulated by the dictates of common sense and conscientious solicitude, guided by the judicious directions of a physician who knows well the nature of the disease with which he has to deal! The nurse ought to note down the hours at which food or medicine has been given, or any remarkable change in the symptoms. She might also, if she were instructed, take observations with the thermometer, for the information of the physician at each visit. Dr. Murchison recommends that, in urgent cases, food and alcoholic stimulants must be persisted in as long as the patient is able to swallow; and even when he can no longer swallow, the case is not to be given up; for he has seen cases where life appeared to be saved by frequent enemata of beef-tea and brandy after the patient had ceased to take anything by the mouth.

Of special symptoms which call for relief, the most urgent is generally headache. If headache should persist after delirium sets in, with a rapid pulse (*e.g.*, 120), attended with nausea, some saline effervescing mixture, with four drops of *dilute hydrocyanic acid*, may be given every six hours. In the persistence of headache, dry cupping, such as has been recommended by Dr. Sieveking, might furnish an aid to guide the treatment, by determining whether it may not depend upon repletion or upon emptiness of the cranial vessels. When applied to the nape of the neck, dry cupping may afford relief, if repletion has to do with the continuance of headache. Under such circumstances the face is generally flushed, the conjunctivæ red, and the skin dry and hot. If the dry cupping does not relieve such symptoms, the hair must be shaved off the head, and the scalp covered with crushed ice inclosed in a bullock's bladder, or recourse may be had to cold affusion. The application of cold water is best effected by bringing the patient's head over a basin at the edge of the bed, and having a vessel arranged so that the cold water (at 40° or 50° Fahr.) may drip continuously from a height of two or three feet upon the head (MURCHISON). A skein of worsted arranged in the water, with the ends overhanging the basin, will maintain a constant flow of water from the basin, which may

be directed to fall upon the scalp. Dr. Murchison recommends that in young subjects two or four leeches may be applied to the temples; and in aged or infirm persons warm fomentations to the head are advisable (GRAVES and MURCHISON); a double fold of lint moistened in warm water and vinegar, is to be laid over the scalp, covered with oiled silk, and the application renewed every three hours. But if anæmia is the cause of the headache, as may be suspected from the state of the vascular system, then stimulants are called for. Four to six ounces of wine may be given in divided doses during the day and night of twenty-four hours. If the pulse continues to get weaker, the wine must be increased. It must be borne in mind that the headache of typhus naturally abates about the eighth day; but it is sometimes rendered worse by sleeplessness; and if the remedies for headache do not relieve it, nor tend to induce sleep, then opiates may be given, combined with *antimony*, if the skin be dry and hot and the pulse of good strength. Dr. Murchison thinks that the employment of *opium* in typhus is more dreaded than it ought to be. The dose of *opium* should be given about 9 P.M., followed in two hours by half the dose if the patient does not sleep. The form of the opiate and dose may be ten to twenty minims of *Battley's solution*, or fifteen minims of the *solution of the bimeconate of morphia*, or five grains of the *opium pill* or *compound soap pill* of the *British Pharmacopœia*. Dr. Murchison teaches us to distinguish two forms of delirium as a guide to the administration of *opium*, combined with *antimony* in the one form, and with *ethereal stimulants* in the other. When the condition of the patient approaches more to that of *delirium ferox*, the cardiac and radial pulses being of good strength, after trying the cold affusion, and remedies already mentioned, then *opium* combined with *antimony* ought to be given without delay, as in the following prescription:—

R. Liq. Opii. Sedat. (Battley's), ℥℥.x.; Antim. Tart., gr. i. ad gr. ii.; Aquæ Camph., ℥vi.; *misce*. A large spoonful of this mixture is to be given every hour until sleep is induced.

On the other hand, if the delirium approaches in its character that of *delirium tremens*, the radial pulse is usually quick and feeble, the cardiac impulse diminished, and the first sound of the heart more or less inaudible, then the *opium* must be combined with alcoholic or other stimulants, the amount being regulated by the state of the pulse and heart. Dr. Murchison suggests the following prescription:—

R. Liq. Op. Sed. (Battley's), ℥ss.; Spt. Ætheris, ℥℥.x.; Aquæ Camph., ad ℥iii.; *misce*. Commence by giving two table-spoonfuls of this mixture, and repeat it every hour till sleep is obtained.

Or *opium* to the amount of half a grain may be combined with three grains of *camphor* in a pill, and such a pill may be repeated, if necessary, every two hours.

Cases requiring such treatment ought to be seen at least three or four times daily. If dyspnoea is urgent, and lividity of the face betoken pulmonary lesion, defective arterialisation of the blood and venous congestion of the brain, *opium* in any form must be withheld; and it must likewise

be discontinued if any tendency to stupor supervene, or if there be any marked contraction of the pupil—e.g., “the pin-hole pupil” of Dr. Graves. This physician proposed the use of *belladonna* in such cases, and he, as well as Dr. Benjamin Bell and Dr. Murchison, bear their united testimony to its usefulness. Dr. Graves prescribed it as follows:—

R. Ext. *Belladonnæ*, gr. i.; Ext. *Hyoscyami*, gr. vi.; Pil. *Hydrar.*, gr. xxx.; *misce*. This mass being divided into six pills, one may be given every three hours.

Dr. J. Harley recommends 15 to 20 minims of the tincture every four hours; or to inject hypodermically from $\frac{1}{8}$ to $\frac{1}{4}$ of a grain *sulphate of atropia*. But *digitalis* is the remedy on which Dr. Murchison places most reliance. From 15 to 20 minims of the *tincture*; or from 6 drachms to 3 ounces of the *infusion* may be given in the twenty-four hours (l. c., p. 284). *Musk* and *camphor* are stimulants of very great value, which have fallen into unmerited neglect. *Camphor* may be given in emulsion in doses of five grains every two hours; or in the form of an enema in doses of a scruple. Huss and Graves also bear testimony to the good effects of these remedies. In a case of complete sleeplessness Dr. Graves gave the following combination of these medicines with the best results:—

R. Antim. Tart., gr. ss.; Pulv. Moschi, gr. x.; Camphor, gr. v.; Tinct. Opii, ℥x.; Aquæ dil., ℥i.; *misce*. A similar draught may be given every two hours; and after the third dose the patient will generally fall into a quiet sleep.

The *hydrate of chloral* has been found to be preferable, in certain circumstances, to *opium* in typhus fever. As a sedative and hypnotic, it is successful in every form of cerebral excitement; but it seems to have a more immediate and permanent curative action on cases of acute delirium at the acmé of typhus, than in the *delirium tremens* of the second and third weeks, when the blood is loaded with the products of the fever. It is also of use where there is bronchitis and congestion of the lungs, when *opium* may not be given. With regard to the dose, 40 grains is found to be a poisonous dose in typhus, producing depression and irregularity of the heart's action. All the benefits without the dangers may be obtained with 20 grain doses for adults; 2 grains for children of one to two years; 3 grains for three years; and 10 grains for children from nine to fourteen years. It is to be given in an ounce of water, sweetened with half an ounce of syrup (Dr. J. B. RUSSELL).

When there is danger of stupor passing into profound coma, the best effects may result from a small cupful of a strong *infusion of coffee*, given every three or four hours, employing at the same time such measures as have a derivative action on the kidneys—e.g., dry cupping; mustard poultices to the loins; wet compresses of thickly folded flannel, wrung out of hot water, passed round the loins, and covered with a piece of water-proof cloth, retained in its place by a bandage or a towel. These remedial agents are all the more necessary if the urine contain either blood or albumen. At the same time free evacuation from the bowels should be secured by a purgative, or by a turpentine enema. If the lethargic

state supervenes early, and before there is great exhaustion, the *douche* has been found to be of great service as a stimulant, provided there be considerable elevation of temperature, and little irritability of the nervous system (TODD, ARMITAGE, MURCHISON). (See p. 302, *ante*.)

The region of the bladder should be examined by the physician at least two or three times daily, by manipulation and percussion, and if there be the slightest doubt as to its containing urine, the catheter must be introduced.

CEREBRO-SPINAL FEVER—*Syn.*, MALIGNANT PURPURIC FEVER;
EPIDEMIC CEREBRO-SPINAL MENINGITIS.

LATIN EQ., *Febris Cerebro-spinalis*—Idem valent, *Febris Purpurea Pestifera*, *Meningitis Epidemica Cerebro-spinalis*; FRENCH EQ., *Fievre Cerebro-spinale*—*Syn.*, *Fievre Purpuree Maligne*; GERMAN EQ., *Epidemische Meningitis*—*Syn.*, *Epidemische Meningitis Cerebro-spinalis*; ITALIAN EQ., *Febbre Cerebro-spinale*—*Syn.*, *Meningitide Cerebro-spinale*.

Definition.—*A fever believed to be of an acute specific character, of sudden invasion, attended by painful contraction of the muscles of the neck and retraction of the head. In certain epidemics it has been accompanied by a profuse purpuric eruption; and, occasionally, by secondary effusions into certain joints. Lesions of the brain, the spinal cord, and their membranes, are found on dissection. The course of epidemic cerebro-spinal fever is rapid, attended with great prostration of the powers of life, severe headache, and pain along the spine. The variations of temperature are so great that no typical range can be given; and, the result is generally fatal.*

Pathology.—(*a.*) *Historical Notice.*—There is reason to believe that this disease has prevailed as an epidemic in Europe at different periods and different places during the fourteenth, sixteenth, and seventeenth centuries, and in the eighteenth century in France, Germany, Italy, England, Ireland, and Scotland. In the present century it has prevailed at Geneva, in Switzerland, and in some parts of North Germany, and in Ireland, and in the United States of America. The name of the disease is apt to fix the attention on local structural changes (which are probably only secondary, and may not always be present), to the exclusion of a consideration of the real nature of the malady. It has been regarded, therefore, by some as a form of simple acute cerebro-spinal meningitis; but its rapid course, greater severity, and clinical history, clearly show that it is more than a simple meningitis. The whole of the nervous system is gravely implicated from the first, so that it generally kills at an early period, leaving no evidence of local structural change. On the other hand, there seems to be no constant relation between the severity and duration of the illness and the extent of the lesions seen after death. There are also not a few, and especially the classical authority of Murchison, who regard the disease as a variety merely of typhus fever; and cases are met with, particularly when cerebro-spinal meningitis and typhus prevail together, which favour this view. The exact nature of the disease seems therefore to be still undetermined; and pathologists have rather stated

what it is *not* than what it is. So far it seems to be a substantive specific disorder, with constant symptoms, and therefore ought to be considered here under the general diseases of a specific febrile nature. That the disease is a peculiar form of typhus fever, the late Dr. F. Niemeyer considered to be completely disproved during the late epidemic in Germany; and he separates it altogether from the group of infectious diseases to which typhus belongs, on the following grounds:—"In the latter (namely typhus) the severe constitutional symptoms, especially the fever, for the most part depend immediately on the reception of the infecting material into the blood, and the anatomical changes in the organs, caused by the infection, are very peculiar; they are induced only by infection with the specific poison. In epidemic cerebro-spinal meningitis, on the contrary, the fever and all other symptoms depend solely on the local disease induced by the infection, and on its injurious effects on the body, just as they do in croupous pneumonia or erysipelas; and the changes in the meninges of the brain and spine are just the same as those sometimes induced in other ways" (NIEMEYER, Vol. II., p. 219). Trousseau seems to be also of the same belief, as he points out, from several *post-mortem* examinations, the similarity of lesions from cerebral fever and lesions extending to the meninges from local injuries. The cause of the disease is unknown, but the phenomena of its repetition as an epidemic are those which belong to the nature of a specific poison infecting the body. The disease extends from place to place, just as other epidemic diseases appear to do; although there does not appear to be any transfer of the disease by actual contact; hence there is no evidence that it is contagious. During the early part of 1865 a disease of this nature prevailed epidemically about the Lower Vistula, and in other parts of North Germany. It had some characters of being a specific fever like *typhus*, but its characteristic lesions were chiefly seen on the surfaces of the meninges of the brain and spinal cord.

(b.) *Morbid Anatomy*.—The *pia mater* was found in fatal cases to be infiltrated with serum, or perhaps suppurating and disorganised. The cranium contained much blood; the longitudinal sinus was engorged; and the *dura mater* alike of cranium and spine was more or less tense with purpuric or hæmorrhagic spots upon it. Lymph was more rarely effused, yet it was occasionally found organised, uniting the opposite surfaces of the serous sac together. Exudation was generally found in the subarachnoid space, serous or sero-purulent. The *pia mater* and the *arachnoid* were also found adherent after effusion of lymph; and instances occurred in which the layers of the spinal membranes were found united to each other. The base of the brain was usually the most severely affected; and generally the lesion extended over the convexity of the cerebrum. The exudation filled up the interspace between the gyri and fissures of Silvius, the subarachnoid space at the base of the brain and cerebellum; so that the nerves as they emerge were imbedded in sero-purulent exudation. These lesions on the surface were associated also with more central and deeper-seated lesions. The brain was more vascular, soft, and almost pulpy in the vicinity of the ventricles, within which the fluid contents were generally purulent, rarely clear serum. Between the *pia mater* and *arachnoid* of the spinal cord there is generally some purulent fluid; and the tissue of the *pia mater* is infiltrated with the

sero-purulent exudation, mostly in the posterior surface, commencing in the dorsal region, and extending downwards to the *cauda equina*, the upper part of the cord being generally free. When infiltration is not obvious, there is usually thickening of tissue or cloudy opacity of the membranes. The substance of the cord is, like the brain, generally increased in vascularity, infiltrated or soft; and Ziemssen has seen the central canal of the cord dilated and filled with purulent fluid (NIEMEYER).

Symptoms.—Many phenomena of this disease are capable of explanation from the nature and position of the specific lesions already noticed. Premonitory symptoms are rare, but when they do occur are usually slight headache and pain in the back, or weariness and a general sense of uneasiness for a day or two before more acute symptoms set in. The invasion is generally sudden. Most usually chill suddenly seizes the patient and shivering prevails, followed by intense vertigo, headache of intolerable severity, obstinate vomiting, and painful muscular stiffness (which soon develops into tetanic contraction), particularly of the neck and back. While consciousness lasts, the distress in the head is incessant; and, during delirium or stupor the patient's instinctive movements show that the head is the chief seat of pain. The eyes are expressive of wild distress, the face generally pale, pupils contracted, and conjunctiva red. Excessive restlessness and general muscular agitation are soon added to other symptoms. Often sensibility of the surface is so great that every touch or movement causes agony. The neuralgia increases, and muscular contractions become more and more uncontrollable and convulsive, affecting, like tetanus, all parts of the body about the third or fourth day of the disease. Deglutition is affected; respiration becomes irregular and imperfect; the head is dragged tightly backwards upon the neck, even at the end of the first or second day, and the features are fixed in the characteristic grin of *trismus* or lock-jaw. Consciousness becomes effaced, and delirium tends towards stupor, the patient passing into coma, or into a depression on the confines of death. By this time emaciation has greatly progressed, and goes on, from the profuse sweating which is apt to attend the typhoid state. Anæsthesia, muscular paralysis, drooping of the eyelids, or squinting, are not unfrequent phenomena—the pupils being dilated, or motionless, or unsymmetrical. When death ensues, it is generally from the fifth to the eighth day; otherwise the duration of the disease may extend over three or four weeks, and convalescence may be of many months' duration. After a few hours to one or more days, an eruption or marking becomes visible upon the skin of the neck, abdomen, back, arms, legs, and face. It is of a hæmic or purpuric appearance, and made up of distinct dark-red or purple spots, about the size of a pin's head or a little larger. The spots are not raised, nor do they fade on pressure, and in some cases are not visible till after death. The tongue remains moist and creamy till the spasmodic stage becomes developed, when it becomes dry, dark coloured, or even black, fissured and swollen, or covered with sordes. Herpetic eruptions on the lips are not uncommon. The respirations become slow and laboured, and as a fatal issue approaches they become hurried, irregular, infrequent, or stertorous; the heart beats and the pulse becomes quick, feeble, and tremulous, or weak and slow, with cardiac blood-murmurs (DA COSTA).

The body temperature is so extremely variable that no typical range can be indicated (ZIEMSEN, WUNDERLICH, NIEMEYER, FOX). Generally a remittent type of fever prevails, with exacerbations of half a degree to a degree, expressed in sudden leaps, each of short duration. The highest temperatures are generally seen in the most rapidly fatal cases; and in most cases the temperature does not rise above 103° Fahr. (NIEMEYER). Based on thermometric records, Wunderlich recognises three varieties:—(1.) One form, rapidly fatal, shows a very high temperature after the commencement of illness, which is maintained for several days, and is still further raised towards the moment of death, when 108° Fahr. or more have been recorded. It is in these cases that temperature continues to rise for some hours after death. (2.) A slight form, with fever of short duration, often of considerable intensity and very irregular course, with exacerbations and defervescence by crisis. (3.) A protracted variety, in which the temperature varies considerably,—the great irregularity mainly depending on successive complications of bronchial, pulmonary, intestinal, or cerebral lesions. On the other hand, Ziemssen believes that during convalescence the intermittent character of the fever is associated with *reabsorption*; while, during the early period, the fever betokens the uninterrupted progress of the meningitis. The intermittent character of the fever has also been made the ground of distinguishing three forms of the disease by Hirsch:—(1.) In one form the intermittency of the fever occurs in the first stage. Repeated attacks of premonitory symptoms commence and pass away, till at last the outbreak of the disease is fully expended. (2.) In another form the symptoms suddenly remit; but next day the case becomes worse,—exacerbations, which occur several times, assuming a more or less irregular quotidian type. (3.) The third form is characterised by perfect intermissions during convalescence. The headache and stiff neck usually increase, generally every day for a considerable period, the patient having daily intervals of comparative comfort.

The pulse shares in variation like the temperature. It may be at or below the normal, frequent or irregular, 80 to 100, with the bodily temperature moderate, and with inspirations increasing from 30 to 40 per minute. The pulse and respiration become more frequent as the disease continues through the third day. A pulse over 120 may prevail, and respirations over 90 a minute, while bodily temperature may be low or over 103° Fahr. The frequency of the pulse does not at all accord with the height of the fever. With moderate fever the pulse may be very high, and slowness of the pulse is rarely observed at the commencement of the fever (NIEMEYER). Besides great irregularity of temperature in this disease, the maximum point is generally below that of typhus, enteric fever, relapsing fever, and scarlatina, and is lower than in any other typhoid condition (FOX).

Diagnosis rests mainly on the irregularity of temperature, the cerebral character of the respiration, the vomiting (cerebral) and peculiar position in which the patient lies, the nature of the delirium, and the eruption. But the main difficulty is to distinguish the fever from typhus on the one hand and acute tuberculosis on the other, with *tubercular meningitis*, also from *tetanus*. There are also certain individual symptoms which challenge

attention as marking certain cases in epidemics,—namely, cervical and dorsal pains, painful sensations in the extremities of a neuralgic character, which (caused by irritation of the posterior roots of the spinal cord) are aggravated on movements of the spine; by hyperæsthesia and anæsthesia of the skin; by tetanic spasms of cervical and dorsal muscles; by epileptiform convulsions; paralysis; disorders of intellect; incessant jactitation at the commencement; blindness from keratitis, and deafness. Death is generally by coma, or from paralysis of the heart by lesion of the *medulla oblongata*.

Prognosis.—The duration of attack varies from a few hours to many weeks. A fatal issue is recorded in as short periods as three, four, five, six, twelve, and thirteen hours; but more than half of the fatal cases die between the second and the fifth day. Convalescence may begin from the fifth day to the fourth week, or later, and is always tedious. Health is not restored for many months. Relapses are not infrequent, and often fatal. The most frequent complications are—congestion of the lungs; sero-purulent effusions into serous cavities, especially the pleura and joints; erysipelas; sore throat; swelling and suppuration of the parotid gland. The disease has been marked by great fatality; and no case can be regarded without anxiety. Nevertheless some cases recover, and the favourable signs are less jactitation, clearness of intellect, while pain in head and back continues, as well as tetanus of dorsal muscles. Improvement sometimes goes on up to a certain point, but advances no farther. In such cases paralysis is apt to set in, and death finally takes place from gradually increasing wasting and anæmia.

Treatment.—At the outset, stimulating *embrocations* to the spine and extremities, with moist or dry heat to the limbs, and wrapping in blankets, give some relief. *Opium* in large doses is the only drug which has appeared to be of any service in subduing the virulence of the symptoms. *Quinine*, in cases where malaria aggravates the disorder, is also well spoken of. *Ether* and *chloroform* inhalation have been of use as sedatives; and *tincture of cantharides* has been of service in cases marked by extreme depression. Counter-irritation, by the actual cautery applied along the spine, or by blisters, has been followed by alleviation of the pain and other symptoms. The energetic use of cold, as of *ice compresses* to the head, of *leeches* behind the ears, and of *calomet*, as in *sporadic meningitis*, are measures to be recommended.

ENTERIC FEVER—*Syn.*, TYPHOID FEVER.

LATIN EQ., *Febris Enterica*—Idem valet, *Febris Typhoides*; FRENCH EQ., *Fièvre Typhoïde* ou *Dothiëntentérie*; GERMAN EQ., *Abdominal-typhus*—*Syn.*, *Ileo-typhus*; ITALIAN EQ., *Tifo Enterico*—*Syn.*, *Febbre Tifoide*.

Definition.—*A continued specific fever, having an incubation-period of about two weeks, with an eruption on the skin of isolated, elevated, rose-coloured spots, chiefly on the abdomen, appearing generally from the seventh to the fourteenth day, occurring in crops, each spot continuing visible about three days and vanishing on pressure. Languor and feebleness are prominent from*

the first, attended by headache, abdominal pains, and (early) by spontaneous diarrhœa; but absolute prostration does not come on till late, the patient rarely taking to bed before the seventh or tenth day. With the advance of the disease the diarrhœa increases, the discharges being for the most part liquid, copious, of a bright yellow colour, devoid of mucus, occasionally containing altered blood. In reaction the discharges are alkaline, and contain a large proportion of soluble salts with some albumen. The fever may terminate favourably by a gradual restoration to health during the fourth week. The average duration of the illness is about twenty-three days. Death in the majority of fatal cases occurs towards the end of the third week. There are special symptoms sometimes associated with the characteristic lesion of this fever—namely, fulness, resonance, and tenderness of the abdomen; more or less tympanites; with gurgling in the iliac fossæ; and increased splenic dulness. The specific lesions are enlargement of the spleen and the mesenteric glands, with enlargement, ulceration, or sloughing of the glands of Peyer and the minute solitary glands of the small, and sometimes also of the large, intestine.

Pathology.—(a.) *History.*—This form of fever has been described under a great variety of names by various writers, such as *typhus metior*; *nervous fever*; *abdominal typhus*; *common continued fever*; *entero-mesenteric fever*; *dottiën-enteritis*; *follicular enteritis*; *bilious fever*, and *gastric fever*. The continued fevers (of which enteric is one form) were at one time embraced (without being clinically distinguished) under the single name of “Common Continued Fever,” of which it was believed there were several varieties. But since about the year 1840 specific differences have been gradually becoming more and more obvious, so that now at least SEVEN DISTINCT FEVERS of the continued type can be recognised, allied by certain common characters, and not less distinctly separable by peculiar and distinctive marks (see Nomenclature and Definitions as given by The College of Physicians, p. 339, *ante*). The plurality of continued fevers must now therefore be generally admitted.

Much has been spoken and written about the identity and non-identity of *typhus* and *enteric* fevers; but proofs of numerous and remarkable differences of a specific kind between *typhus* and *enteric* fever have been slowly but surely accumulating since the beginning of the present century. It was in Paris, by the dissections of Prost in 1804, that the foundations of our knowledge were laid which turned the attention of pathologists in the direction which has led to such definite results. In more than 150 dissections of Parisian fever patients Prost always found “inflammation,” with or without ulceration of the mucous membrane of the intestines. Petit, Serres, Pommer, and Bretonneau followed up the investigation; but the celebrated treatise of Louis, in 1829, was the first to give a complete and connected view of symptoms as well as of *post-mortem* lesions in the fever then common in Paris. Although Prost asserted the connection of a certain intestinal lesion with a definite series of symptoms, still it was Louis who described this intestinal lesion in terms sufficiently precise, and indicated with scientific exactitude the symptoms with which it is concurrent. The views of Louis were subsequently adopted by Chomel and Andral in France. In other parts of Europe, however, and especially in England, bodies of numerous fever patients were opened without finding any disease of Peyer’s patches, although differences in the symptoms

detailed regarding the fevers of France and England were not then so obvious. Hence arose at once two opinions, based on a *post-mortem* distinction, namely, the "anatomical sign," described by Louis. The first opinion was that this "anatomical sign" was an incidental occurrence; or, that its occurrence was in some way connected with locality, the cases of fever being everywhere considered identical. A second opinion, however, soon began to gain ground, especially when the intestinal lesion was not found by the most careful observers in some cases where it was intentionally looked for (as in the epidemic of Toulon in 1829-30)—namely, that there were in fact *two diseases* which were indifferently named *typhus* and *typhoid fever*; that one prevailed only at Paris, and the other in England, in Germany, and elsewhere; and that it was also sometimes more or less mixed up with the Parisian fever, as measles may be with scarlet fever. Louis subsequently (1841) adopted this view.

In 1835 the "Académie de Médecine" formally proposed the question, "What are the analogies and the differences between the *typhus* and *typhoid* fevers?" The question excited considerable interest in France, but less so in England, where a strong bias has always prevailed towards a belief in the doctrine of a single fever—a belief entertained and taught by the most eminent observers and teachers of that day. But dissenters arose. Scotch, English, and American physicians, practically familiar with the fever of their own countries, began to visit Paris to study fever there; and they were not long in learning to recognise the chief points of difference between the two fevers. Gerhard and Pennock, of Philadelphia, in a systematic treatise, were the first to indicate (1836) these differences, it having been already determined by Jackson and Gerhard that the fever described by Louis under the name of *typhoid fever* existed in America, and presented there the same assemblage and development of symptoms, and the same *post-mortem* lesions, as the Parisian fever. In 1836 M. Lombard, of Geneva, after visiting London, Edinburgh, and Dublin, ultimately came to the conclusion that two different fevers had been confounded together. During this year (1836), also, Dr. A. P. Stewart commenced his observations in the Glasgow Fever Hospital, where he continued his inquiry for two years. His attention was first especially directed to the study of fever by Dr. Peebles, who, during a long residence in Rome, had observed the maculæ of typhus in the contagious fever of Italy, and who first showed the difference between the characteristic eruption of fever and the cutaneous affection to which the name of "*petechiæ*" is given (*Edin. Med. and Surg. Journal*, 1835). He pointed out this eruption to Dr. Perry (then physician of the Glasgow Fever Hospital), "and who," Dr. Stewart states, "was the first to maintain the complete difference of the two eruptions—namely, those of *typhus* and *typhoid fever*. Dr. Stewart subsequently went to Paris and examined the fever there. The result was a complete recognition of the existence of two fevers and of their differences—an account of which he published in the *Edinburgh Medical and Surgical Journal* for 1840, p. 289. In 1839 Enoch Hale published an account of the fever of Massachusetts, and distinguished among them two perfectly different forms of fever, one of which agreed with the Parisian fever, while the other might be held to represent the fever described by most English writers,—which we now know

to have been "typhus." Soon after this the characters of the prevalent fevers of England were noted by Shattuch (another American pupil of Louis), who published his results in the *Boston Medical Examiner*. The appearance of these papers, and of others about this period (1840), gave rise to an elaborate discussion of the whole question (in the pages of the *British and Foreign Medical Review*, Vol. XII., p. 293); and the conclusion the reviewer arrived at seemed to favour the opinion "that the French and English fevers were varieties, that is, different developments of a common stock, but not specifically distinct diseases." I understand the eminent physician who wrote that review now believes in the specific distinction of the two fevers; and it is since that period that the differences have been carefully looked into. In America the doctrine of a specific distinction between the two fevers has been generally adopted, as represented in the treatises of Dr. Bartlett, of Philadelphia, in 1842 and 1847, *On Typhus and Typhoid Fevers* and *On the Fevers of the United States*. In Germany three opinions were entertained. The typhoid fever of Louis received from the Germans the name of "abdominal typhus;" thus regarding the disease as a variety of typhus fever, the latter being the simple, and abdominal typhus (enteric) being a complicated form and higher grade of the same disease. By some, however, it was regarded as a disease distinct from the "*typhus exanthematicus*." A third opinion also found followers—namely, that this *abdominal* typhus was the only form of continued fever. All these opinions were the result of a too limited and narrow field of investigation.

Up till 1846 opinions were thus divided, crude, and, in many schools of Medicine, quite unformed. Relapsing fever was distinguished by some, but not by all; and all other forms of continued fever were considered in this country as identical. Under these circumstances the inquiry was taken up in 1846 by Dr. (now Sir William) Jenner, and at that time Professor of Pathological Anatomy in University College, and worked out by him systematically in the London Fever Hospital. There he patiently accumulated case after case of fever, until he had nearly 2,000 accurate reports before him. From these he separated all cases of relapsing fever, and then instituted a rigorous comparison of the remaining cases. He selected the fatal cases which had been examined after death, and the diagnosis of which had been confirmed. He found that he had sixty-six such cases and *post-mortem* examinations. Of these sixty-six cases twenty-three had the intestinal and mesenteric lesion—the "anatomical sign" (according to Louis) of typhoid fever; and forty-three cases were without this appearance. The following question then remained for solution; namely,—Did these forty-three cases (in which the intestinal lesion was *not present*) differ so much in symptoms and *post-mortem* appearances from the other cases (in which the "anatomical sign" referred to *was present*) as to render it impossible to suppose that they were cases of the same disease? Or, contrary to the opinion of Louis,—Were the symptoms of the two sets of cases so similar as to lead to the belief that the presence or absence of the intestinal lesion (the "anatomical sign") was a matter of little consequence? On comparing these two groups of cases, Dr. Jenner found that while the symptoms and *post-mortem* appearances of the twenty-three cases were exactly the same as those

described by Louis, the symptoms, course, and *post-mortem* appearances of the remaining forty-three cases were entirely different—so different, indeed, as to render their separation from the other cases a matter of absolute necessity, if accuracy was to be maintained in the description of these diseases, or certainty arrived at in their treatment.

Causation, as a ground of distinction between *typhus* and *enteric* fevers, is a condition upon which much stress has been properly laid by Sir Wm. Jenner, and subsequently by the late Dr. Murchison. Dr. Jenner was the first to argue that the material media by which the two fevers are propagated are specific and different from each other, according as they are generated by the bodies of those affected with the one or the other form of fever. This argument he based upon the circumstance, that because *certain local foci* sent *enteric* cases to the hospitals, and *certain other local foci* sent *typhus* cases there, he inferred that different specific causes existed in each focus. Dr. Murchison has also clearly stated the evidence of many other observers in his great work on *The Continued Fevers of Great Britain*, which goes to prove that the two fevers have no community of origin. The late Professor Niemeyer has also recorded his experience that the *contagion of enteric fever* is not so intense as that of *typhus* fever. It clings more particularly to the dejections, but he considers it doubtful if the contagion is capable of transference by exhalations from the skin and lungs of *enteric* fever cases. On the other hand, exhalations from the skin and lungs are certainly vehicles for the contagion of *typhus* fever. Infection of the nurses and physicians is rarely seen with cases of *enteric* fever, and when it occurs it is generally traceable to the influence of the dejections. At all events, using the bed-pans, night-stools, and privies, where the dejections of enteric fever patients have been emptied, appears to be more dangerous than being brought in contact with the patient. The opposite is the case with *typhus* fever (PARKES, *Med.-Ch. Rev.*, July, 1851).

Since 1851 proofs of differences between the *typhus* and *enteric* fevers have been still accumulating in many different directions. They especially result from the observations of Dr. William Budd, of Clifton, near Bristol; of the late Dr. Murchison, of St Thomas's Hospital, London; of the late Professor Wunderlich, of Leipsic; and of Von W. Greisinger, of Zurich. The observations of these two latter physicians are especially valuable, as showing the ranges of temperature in the two fevers to be distinctive of two diseases. The excellent lectures given by Dr. Peacock, of St. Thomas's Hospital, in 1855, and published in the *Medical Times* of 1856, "on the varieties of continued fevers and their discrimination," must also be mentioned as influential in forwarding these modern views; for they appeared at a time when the specific distinctions between typhus and enteric fevers were less generally admitted than they are at present. Thus the evidence has slowly but surely accumulated; and when the whole subject has been re-examined in all its relations, the conclusion irresistibly forces itself on the mind, that a belief in the identity of *typhus* and *enteric* fever is no longer tenable. They are distinct and different diseases. In common with many, I had, as a student, been taught to recognise the striking similarity between the two fevers, in outward aspect, in many respects; and therefore I was unduly biassed by the *resemblances*, rather than led to give sufficient importance to the

numerous and remarkable *differences* between them to which I would now direct attention.

With regard to their most prominent points of resemblance and difference, it may be shortly stated here, that all the points in which the two fevers agree are common to them and many other diseases, and therefore are of no value as *indicia* of a species. The resemblance is in no respect perfect—not more than is seen between typhus and cases of measles which are attended with typhoid symptoms (*typhous measles* of NIEMEYER). On the other hand, the points in which they differ are all of a very special nature. The points in which they agree may all be summed up in the phrase “typhoid symptoms”—a set of symptoms which are met with in a great variety of diseases, and therefore are of no specific value in the question at issue. These so-called typhoid symptoms occur not only during the course of *typhus* and *enteric* fevers, but are also found to occur and to group themselves in a similar manner in *measles*, *scarlet fever*, *small-pox*, *pyæmia*, *uræmia*, some forms of *pneumonia*, and in many cases of *acute tubercle* (W. BUDD). Such diseases are then said to pass into the “*typhoid state*”—*i.e.*, they resemble *typhus*, especially in the malignant and putrid characters. Such so-called *typhoid symptoms* are made up of the following phenomena:—“A quick small soft pulse; a dry brown tongue; teeth covered with *sordes*, the symptoms and physical signs of hypostatic congestion of the lungs; great impairment of the mental faculties; extreme prostration, stupor, and more or less unconsciousness, passing into coma; delirium, which at one time is acute and noisy, at another low and muttering, and not unfrequently associated with muscular tremor; involuntary evacuations, and occasionally subsultus, carphology, or even general convulsions. The precise grouping of the symptoms varies in different cases even of the same disease (MURCHISON, W. BUDD). The *post-mortem* appearances met with in such cases are a dark fluid condition of the blood; hypostatic congestion and oedema of the lungs; old disease or recent congestion with epithelial engorgement of the tubes of the kidneys; enlargement and softening of the spleen, and, unless the typhoid state has been of very short duration, an accumulation of serous fluid in the lateral ventricles and at the base of the brain, the veins and sinuses of which are usually full of dark blood (MURCHISON). This typhoid malignant or putrid condition is developed in the following diseases, namely,—true typhus fever, enteric or typhoid fever, malarious remittent fever, yellow fever, and cholera; cases of severe pneumonia where symptoms of the local lesion are masked by those of the general blood poisoning; cases of acute rheumatism, acute tubercle, pyæmia, uræmia, and especially in those forms of disease where the kidney is contracted, granular, or “gouty,” and where albuminuria is often absent. Such “*typhoid symptoms*” are now found dependent not so much on the primary poison, as upon the blood becoming contaminated by the *debris* of the disintegrated tissues, in addition to the original fever poison, especially if there be any impediment to excretion. Hence these “*typhoid symptoms*” are closely assimilated in the advanced stages of all these diseases, although the primary poisons have been perfectly distinct (MURCHISON).

The disease is chiefly met with in youth and adolescence—the mean

age of patients being about twenty-one. The lesions of enteric fever have been considered rare after fifty years of age. Persons under thirty are more than four times as liable as persons over thirty—a very remarkable difference as contrasted with *typhus* fever (MURCHISON); but the proportion exceeding fifty years of age (1·37 per cent.) is much larger than at first appears, seeing that only one-seventh of the entire population of England and Wales is above fifty years of age; and many who survive that age may have acquired an immunity from the disease by a previous attack (MURCHISON). It is necessary to remember, however, in connection with the age of enteric fever patients, that the solitary and the aggregate glands of Peyer are known to be most fully developed and most active in youth, up to the age of early manhood; after that time they are obviously less active. Structure and function seem to be alike impaired by age, till at length after forty or forty-five years, traces only of the existence of Peyer's glands are apparent, or they have altogether disappeared. The gland substance no longer exists; the glands have become atrophied, and the places where the patches of Peyer once were may be detected only after careful examination,—a mark of varied form and character being all that indicates the place of the patch. Twenty-seven cases have been noted at the Fever Hospital above sixty, and two above seventy-five, in one of which characteristic typhoid ulcers were found in the ileum after death. Trousseau records having also seen the characteristic lesions in a woman sixty-four years of age; and they have been seen by Wilks in a woman aged seventy, and by others in cases aged respectively seventy-two, eighty-six, and even ninety years of age (MURCHISON). These exceptional cases are explicable when it is known that the existence and functional activity of these glands are sometimes prolonged for an indefinite term of years beyond the usual period of their existence. On the other hand, it is in childhood, early life and manhood that Peyer's glands are most obvious, and their functional activity the greatest; while “more than one-half of the cases of enteric fever occur between fifteen and twenty-five years of age; more than one-fourth are under fifteen. The average age of enteric fever cases is 21½. In very early life the proportion of cases of enteric fever would be greater, were it not that many children labouring under this disease are described and treated at their own homes as cases of “*Infantile Remittent Fever*,” so that few young children are admitted into the Fever Hospital (MURCHISON).

The following records with regard to the age of enteric fever patients, contrasted with those of typhus, and collected by Dr. Murchison, from the records of the Fever Hospital, are of striking interest :—

PERCENTAGE OF CASES OF TYPHUS AND TYPHOID FEVER AT
DIFFERENT AGES.

	Typhus.	Typhoid.
Under Ten Years,	7.88	10.42
From Ten to Fifteen Years,	12.06	18.16
From Fifteen to Twenty-five Years,	29.39	46.55
Twenty-five Years and upwards,	50.66	24.87
Thirty " " " " " "	41.14	13.30
Forty " " " " " "	24.70	4.54
Fifty " " " " " "	10.68	1.37
Sixty " " " " " "	3.87	.44

It is thus pre-eminently a disease of childhood and adolescence.

(b.) *Morbid Anatomy and Special Lesions in Enteric Fever, with reference to the Phenomena and Progress of the Disease.*—Complications of enteric fever, as they are sometimes called, are mainly due to lesions of the solitary and aggregate glands of Peyer, and to enlargement of the mesenteric lymphatic glands. These lesions in the small intestine and in the mesenteric glands are especially recognised as the “anatomical sign” of enteric fever. Of these the most noticeable are to be seen in the small intestines (hence the German name of *ileo-typhus*). They may be considered in the following stages:—

I. *A generally congested state of the mucous membrane of the small intestines, especially expressed in the vicinity of the solitary glands, which are surrounded by vascular rings, and clustered groups of agminated glands which constitute Peyer's patches.** This vascularity seems to be very general, involving most of the abdominal viscera. Sensations of heat and abdominal distress are associated with this morbid state, and the lineaments of the belly are obliterated.

II. *Associated with this congestion, the adenoid or gland tissue of the mucous membrane becomes obviously prominent.* The proper structure of the glands becomes enlarged by increased growth of the cell-elements (proliferation), and as the process advances the surrounding connective tissue becomes implicated, until at last the whole patch is converted into a cutaneous mass of altered gland tissue (MURCHISON). This condition is sometimes described as *intumescence* of the glands, and with the congestion just noticed constitutes the stage of “*infarction*,” as described by the older authors. Symptoms denoting intense irritation of the mucous membrane—*catarrhal* and *gastric* symptoms—prevail; and the mucous membrane of the small intestine generally is swollen, relaxed, and turgid, especially the villi, which are particularly distinct, imbedded in a thick layer of dirty-yellow gelatinous mucus. Although these conditions seem to involve the whole of the mucous membrane, yet they are more expressed towards the lower end of the small gut at an early period of the disease than in any other part. “The morbid process, as well as the process of reparation, always commences at the extremity of the ileum nearest the cæcum, and proceeds upwards” (MURCHISON). The time at which the increase of cell-growth commences in the glands is not yet well defined in relation to the day of the fever. Nor are observers at one as to the constancy of *congestion*. Rokitsky maintains that the enlargement of the glands is preceded by a “*congestive stage*.” On the other hand, Murchison considers there is no evidence that the enlargement of the glands is preceded by increased vascularity. He has never found increased vascularity without enlargement of the glands when death has occurred at an early stage of the disease; and slight enlargement without any increase of vascularity

* It has now been shown that these glandules are not closed vesicles which periodically discharge their contents into the bowel, as represented in previous editions, but they are in reality small lymphatic glands. Each glandule is composed of a delicate fibrous reticulum, inclosing lymph corpuscles in its meshes (adenoid tissue). Their function is that of absorption, and not that of secretion or elimination—their period of greatest activity corresponding to that of intestinal absorption into the blood (KOLLIKER). They have been injected from the lacteals; so that a Peyer's patch is really a lymphatic gland spread out in the coats of the intestines.

he believes is not uncommon. But although mere increase of vascularity of the agminated and solitary glands without any enlargement will not justify the opinion that a patient has died of enteric fever, however short may have been the duration of the illness, yet there is evidence in the general and abdominal symptoms during life that hyperæmia pervades most of the organs in the abdomen, and my experience is in favour of increased vascularity of the mucous membrane of the ileus upwards from the cæcum, as accompanying the increased growth of the gland tissue. This growth of gland tissue commences with the disease, and continues to progress till about the ninth day. A case is described by Dr. Sankey, in the first volume of the *Pathological Society's Transactions*, in which dissection showed the growth in the glands as early as the fifth day. Cases fatal on the fifth day with similar enlargement of the glands are described by Bretonneau, Forget, Bristowe, and Hoffman; and Murchison recently recorded a death at the end of the second day in which there was considerable enlargement of the solitary glands; and in some cases at Clapham considerable enlargement was found at the end of the first day. The bulging of Peyer's glands and the extent of intumescence vary considerably in different patches; and simultaneously with these conditions the mesenteric and bronchial glands begin to increase in size. They, too, are supplied with an increased quantity of blood, and the increased tissue of the gland becomes unusually soft and elastic, vascular and dark coloured.

III. *A subsidence of the general congestion, and of the generally turgid state of the mucous membrane, takes place after the gland-growth has been fully developed, round which also the general redness and swelling concentrates.* The growth continues actively, and progresses rapidly within the gland-structures till the patches of Peyer become so thick as to be elevated three or four lines above the surface of the surrounding mucous membrane. The growth reaches this point by the eighth or tenth day of the disease. A beautiful vascular halo encircles them, stopping short at their margins; and a contracted border surrounds the margin of the patch, which gives it a sessile fungiform aspect, with an umbilicated-like depression on its surface. Growth is now confined within narrow limits, pressing on the muscular coat below and the mucous coat above. The patches assume various aspects as to colour; and, when vascular, they have an appearance which has acquired for them the description of being like "fleshy lumps," their tawny colour showing through the peritoneum of the gut. Varicose vessels abound in the vicinity—a fact of some importance in connection with the formation of thrombi, leading to hepatic or pulmonary embolism. The specific gravity of the gland tissue of Peyer's patches is obviously changed by such increased growth. I have found it to range from 1.032 to 1.044. The solitary glands at the lower end of the ileum are generally also affected in the same way as those in Peyer's patches. The size of the swollen solitary glands varies from that of a millet seed to a split pea. They have thus been considered to resemble the pustules of variola. Sometimes, as towards the lower end of the ileum, Peyer's patches and groups of solitary glands will be found to coalesce, so that the lesions may almost entirely cover six or seven inches of intestine. These changes are pathognomonic of enteric fever. Occasionally the increased growth (sometimes called "*medullary infiltration*") extends beyond the

gland structures into the surrounding connective tissue of the mucous membrane—a cellular new growth which originates from the connective tissue corpuscles (GOODSIR, VIRCHOW).

IV. *Softening of the tumid gland-growth* seems to be the next event in the series of disease processes, and which would appear to be preliminary to one or other of the following conditions under which the softened glands subside or disappear:—(1.) *Elimination without ulceration*—i. e., simply by re-absorption. This is the usual way in which the gland tissue disappears, just as some kinds of buboes do. For many reasons I am induced to believe that this is the natural, the most common, and the most frequent mode by which the softened new growth in cases of enteric fever is got rid of—namely, by the reabsorption of the softened contents of the glands. The membrane covering the patches is of a pink grey or purple colour, and sometimes softened; between the patches the colour may be normal, or showing every grade of vascularity up to the most intense injection (MURCHISON). The glands collapse, and assume the appearance of little pits, depressions, or follicles, and so give rise to that “reticulated indistinctly pitted surface” so often seen after all evidence of gland-structure has disappeared. This view is also consistent with the observation of Wedl, when he says that “the glands in question not unfrequently burst; and the capsules also may collapse, in consequence simply of absorption of their contents. Owing to one or other of these occurrences, the Peyerian patches acquire the well-known reticulated aspect, since the mucous membrane surrounding the individual capsules assumes the form of a projecting border; and entire patches present the appearance of a fine sieve” (*Pathological Histology*, p. 221). The description also agrees with the experience of Niemeyer, when he says that “not unfrequently the process becomes retrogressive, without the occurrence of destruction of the wall of the follicle, or of the mucous membrane covering it, the swelling of the glands subsides, while their contents are reabsorbed after the cellular elements have been destroyed by fatty metamorphosis;” and “in still other cases,” he has written, “the individual glands composing Peyer’s patches rupture and empty their contents outwardly, without the covering sloughing. As a result of this, the surface of the plaques looks as if full of holes, or it has a net-like appearance (*plaques à surface réticulée*).” The ordinary peristaltic action of the intestines may assist this mode of elimination by absorption or by rupture of the glands, if the softening is complete. A case I dissected at Scutari led me first to the conclusion I have stated. My original idea was that the material was got rid of by the glandules (then believed to be vesicles) rupturing and discharging their contents into the bowel,—a view since shown to be erroneous; and which I have now modified in accordance with more recent anatomical observations. In this case a process of growth and elimination seemed to have gone on for at least one month previous to death; and the elimination of the material from the patches took place without ulceration. The man died suddenly from aneurism of the aorta. A lull in the febrile symptoms had led to his premature discharge from hospital; and the day on which he suddenly died was to have witnessed his embarkation for England. Peyer’s patches were all greatly enlarged and obvious. In some parts of them the glandules were greatly distended with the material of new growth, in a milky-like con-

dition; while other parts of the same patch were completely bare, and dotted over with minute points of black pigment (the probable remains of previous intense congestion). These parts were quite bare of all gland-structure, and had a reticulated appearance. In corroboration of this view, it is to be observed further, that Dr. Friedrich, of Dresden, regards the elimination of the deposit from the Peyerian patches in the enteric fever of children, by the formation of sloughs and ulceration, as extremely rare. For the most part, only single follicles in the glandular assemblage are infiltrated, and these, either from reabsorption of the infiltrated material, or more often from rupture of the follicle within the intestinal canal, *revert to a normal condition, without the formation of any cicatrix* (*Brit. and For. Med. Chir. Review*, July, 1858, p. 162). Murchison considers that this reabsorption is what probably occurs in cases where the disease is mild and of short duration. The cases which do not go on to ulceration or sloughing, and where the whole of the presumed poison is absorbed into the blood, are always the shortest and the mildest.

(2.) *Elimination by ulceration of the swollen gland tissue* occurs in groups of various sizes, involving more or less of surrounding tissue in ulceration, and tending to induce perforation of the gut or peritonitis. Considering the severe nature of this lesion, and taking into account the fact that a large proportion of cases of enteric fever recover, it seems to me that this is a mode of elimination which occurs much less frequently than the mode already described. Of course it is the state most frequently met with after death, and of which specimens are preserved in museums—for ulceration of Peyer's patches is the characteristic "anatomical sign" of enteric fever. The time of commencing ulceration of the mass appears to be about the ninth or tenth day; but it may commence earlier or later. Dr. Murchison has seen it as early as the eighth day. He refers to other cases earlier, and considers we have reason to believe that in rare cases ulceration may commence as early as the first or second day; and the softening of the new growth which precedes ulceration is associated with a return of the violent congestion to the small intestines, when the veins especially are filled with dark-coloured viscid blood. The outbreak of ulceration is always characterised by an aggravation (recrudescence) of the original symptoms, after it may have been sanguinely supposed that convalescence had decidedly taken place. But in such deceptive convalescence the abnormal temperature is maintained, showing with absolute certainty that the fever is not at an end. The ulceration therefore is usually denoted, (*a.*) by a reaccession of febrile phenomena, with or without diarrhoea; (*b.*) by abdominal pains and tenderness. Judging from *post-mortem* examinations, the ulceration seems to commence at the lowermost patches of Peyer, in the glands nearest to the cœcum, and the ileo-cœcal valve is often implicated in the destruction. The ulcers vary in number and in extent; and although there is a tendency to perforation of the gut in fatal cases—the most important and dangerous complication of enteric fever—yet peritonitis may supervene without perforation having actually taken place. Various statements have been made concerning the tendency of enteric ulcers to perforate the gut, and the frequent association of this lesion with peritonitis. Perforation is said to be rare in the northern parts of Europe (Huss); but, from the

records of Louis, Murchison, and Bristowe, it appears to be a more frequent mode of fatal termination than has been commonly supposed in this country. It seems also more common in England than on the Continent; and from the extensive statistics given by Dr. Murchison in the second edition of his great work *On Continued Fevers*, it appears that in England of every thirty-three cases of enteric fever one case dies of perforation, and that perforation occurs about one in five fatal cases. Perforation may occur in three ways (MURCHISON), namely:—(1.) By molecular disintegration, or extension of the ulcerative process. One or more minute small rounded openings may then be seen at the base of the ulcer, each just large enough to admit a pin or a stocking wire; and this, according to the extensive experience of Dr. Murchison, is the most common mode. (2.) A considerable portion of the peritoneum may slough, and perforation result from its partial or complete detachment. The opening may then be of considerable size,—indeed, sloughs corresponding to several entire patches of Peyer and including peritoneum, have thus fallen out, leaving large oval apertures. (3.) Perforation may result from rupture of denuded peritoneum, a mode of perforation presumed to be common in those latent cases where the patients have never been so prostrate as to confine them to bed. Perforation does not occur in the course of any other acute disease, except in cases of dysentery or tuberculosis, and rarely in them. It generally takes place through the ileum, near the valve, although it has been known to occur in the colon, and also as high up as the jejunum. *Post-mortem* examination often discloses vigorous attempts on the part of neighbouring structures to limit by union and adhesion the results of perforation, obviously indicating, in practice, the necessity of absolute rest throughout the disease.

The characters which distinguish the ulcers of typhoid fever from other ulcers of the intestines may be stated as follow:—(1.) They have their seat in the lower third of the small intestine, their number and size increasing towards the ileo-cæcal valve. (2.) They vary in diameter from a line to an inch and a half—from the size of a hemp seed or a pea to a crown piece; but a number of ulcers may unite to form a mass of ulceration several inches in extent. Such extensive masses of ulceration occur close to the cœcum. (3.) Their form is elliptical, circular, or irregular—elliptical when they correspond to an entire Peyer's patch, circular when they correspond to a solitary gland, and irregular when they correspond to a portion of a Peyer's patch, or when several ulcers unite to form one. (4.) Elliptical ulcers are always opposite to the attachment of the mesentery. (5.) The ulcers never form a zone encircling the gut, as may sometimes be seen in the case of the tuberculous ulcer, but their long diameter corresponds to the longitudinal axis of the intestine. (6.) Their margin is formed by a well-defined fringe of mucous membrane, detached from the submucous tissue, a line or more in width, and of a purple or slate-gray colour—an appearance best seen when the bowel is floated in water. (7.) After separation of the slough there is no thickening or induration of the edge of the ulcer, as in the case of the tuberculous ulcer. (8.) Their base is formed by a delicate layer of submucous connective tissue, or by the muscular coat, or occasionally by nothing more than peritoneum. (9.) There is no deposit of morbid tissue or new growth at the base of the

ulcer; although sometimes fragments of yellow sloughs may be seen adhering to both the base and edges (MURCHISON, p. 619, 2nd Edition). The ulcers also are known to heal. Their cicatrices have been seen four, five, and thirty years after known attacks of enteric fever (ROKITANSKY, BARRALLIER). And in cases where death occurs during a relapse, the cicatrices from the first attack may be found co-existing with the fresh growth in the vesicles, and with the recent ulcers of the relapse. As a rule, the reparative process does not commence till the end of the third week of the disease, and in one case, where the primary fever lasted three weeks, and where death occurred from complications about the fortieth day, all the ulcers in the ileum were cicatrised (MURCHISON). Cicatrisation commences by the growth of a thin, delicate shining layer of new growth which covers the base of the ulcer, and is also attached to the basement membrane of the mucous coat. The fringe of mucous membrane becomes adherent to this new tissue, from the circumference towards the centre, until the healthy mucous membrane merges insensibly into a serous-looking lamina. The new film of membrane cannot at first be moved upon the subjacent coat, but after a time it becomes movable, and, according to Rokitansky, even becomes covered with villi (MURCHISON). There is no evidence of the gland-structure ever being restored. The resulting cicatrix is slightly depressed, firmer, less vascular, and smoother than the surrounding mucous membrane. The bowel appears thinner at this part when examined by transmitted light. The depressed spot is never surrounded by any puckering, nor does it ever cause any stricture or diminution in the calibre of the gut.

(3.) *Elimination of the gland growth by sphacelus of large masses of Peyer's patches.* The whole gland substance, with the superimposed mucous membrane, is generally involved in the destruction, as well as the morbid material in the submucous tissue. The cell-growth in the glands suddenly becomes so excessive that a condition is at last reached which is incompatible with the maintenance of life. The growth actually chokes itself; and the whole mass, or a great part of it, softens and dies. It becomes detached in the form of a slough, leaving an ulcerated surface behind. Such sphacelus has been known to happen as early as the twelfth day of the fever; but the process is generally more slow. A dirty yellow-brown slough forms, varying in thickness, and sometimes extending as deep into the substance of the gut as to expose its muscular layer on separation of the mass. There is a tendency to bleeding on separation of the sloughs; and such hæmorrhage occurs in about one-third of the fatal cases. The frequent repetition of such hæmorrhages during life has a marked influence in modifying the febrile phenomena. For example, in a case described by Dr. Parkes, in which the temperature was very carefully recorded three times daily, it was observed to fall *below* the standard of health on the fourteenth and fifteenth days slightly, on the sixteenth day to the extent of 4° below 98°, on the seventeenth day to the extent of 5°, on the twentieth day to the extent of 2°. These falls of temperature were all traceable to the influence of repeated hæmorrhages from the bowels. The occurrence of hæmorrhage is always a most alarming symptom, and is most frequent during the third and fourth weeks of the disease. It varies in amount from a mere stain to a large quantity of blood, sometimes discharged in

clots, and generally of a red colour, in consequence not only of the rapidity with which it is passed out, but also, as Dr. Parkes has shown, in consequence of the alkaline reaction of the contents of the intestine. It may cause immediate death by syncope; or, by reducing the temperature and strength of the patient, he may sink exhausted, unable to cope with the disease. Whenever, therefore, blood appears in a case of typhoid fever, it is certain that the lesions of Peyer's patches are severe.

V. The mucous membrane of the intestines having existed for several weeks in such a state of irritation as has been described, and the catarrh being more or less excessive, *an atrophic condition of the intestine at last supervenes*. The mucous tubes become wasted, irregular in form and size, as seen on microscopic examination, sometimes separated by an interstitial growth of a granular nature, and their bulbous ends disappear. The whole substance of the gut then becomes so thin that it resembles a portion of thin paper rather than intestine. Hence some cases never recover from this atrophy, but die of marasmus.

VI. The mesenteric glands are invariably enlarged. They begin to enlarge at the very commencement of the disease, gradually becoming soft, vascular, and dark-coloured, and sometimes attain a very large size, and their stages of congestion, of swelling, and of subsidence, go on simultaneously with the similar changes in Peyer's patches, increasing day by day till about the fourteenth day. They decrease and become softer when the material begins to be detached from the intestinal glands; but they have been found considerably larger than natural as late as the thirtieth day. When death is so late as the fifty-sixth day I have found them small and shrunken, and of a greyish-blue colour, in a case of unusual severity with continued recrudescence of the disease. In cases of relapse, on the other hand, they are found enlarged at so late a period in the history of a case.

VII. The spleen is usually greatly enlarged, varying from five or six to fourteen ounces (twice to six times its normal size), with a specific gravity varying from 1052 to 1059. Its Malpighian sacculi (glandular) are also intumescent; its parenchyma puffy, and of a dark violet or blackish-red colour. The splenic pulp, in addition to the ordinary lymph cells, contains many other cells of larger size and multi-nuclear; found in largest number during the first three weeks of the disease, while the organ is still firm. When the organ becomes soft, the small lymph corpuscles become more numerous, mixed up with much granular matter, while yellowish brown pigment granules are found in the cells and amongst the trabiculæ (MURCHISON). In some cases (rare) it has ruptured, and so caused death by hæmorrhage into the peritoneum. Infarctions or embolic masses are sometimes met with.

VIII. The liver is sometimes hyperæmic, but generally normal as to colour, or pale; soft in consistence, with an indistinctness in the mapping out of lobules. The microscope shows the liver-cells undergoing marked disintegration—generally fatty; and in advanced stages of the disease the cells have been seen of large size, and containing several nuclei. Pyæmic deposits and infarctions due to embolism are also recorded. In a large proportion of cases, at third or fourth week of the disease the bile is thin, watery, and almost colourless, and of an acid reaction.

IX. Thoracic lesions occur, (1.) as infiltrations of the lungs; or, (2.) as the consolidation of pneumonia; or, (3.) as portions of lung which have become carnified; (4.) as enlargement of bronchial glands similar to those of the mesenteric; (5.) bronchial catarrh; (6.) laryngeal or bronchial ulceration. In the first-mentioned form of lesion the growth seems to commence in the terminal air-vesicles, ultimately assuming the form of a miliary deposit, with a semi-transparent gelatinous appearance. It is the irritation set up by this sudden growth which generally gives rise to pneumonic consolidation. Softening and friability of the pulmonary texture is thus a very constant *post-mortem* state in protracted cases of enteric fever. Such lesions usually supervene during the later period of the fever, and when the ulcerations of the intestines are extremely spread (HUSS). In this respect only it differs from the consolidation of the lung described in typhus fever. This lesion has been also termed *non-granular consolidation*; and it may be observed not only in the course of *enteric fever*, but in *measles*, *scarlet fever*, and *small-pox*. It is of the catarrhal form of pneumonia. A portion of the lung in this condition has a mottled aspect. There are patches in it here and there, varying in size from a single lobule to half, or more than half, of a lobe, of a deep bluish-chocolate, violet, or purplish-slate colour, bounded by a well-defined angular margin, and crossed and mapped out into smaller patches by dull, opaque, whitish lines. These are seen to be thickened lobular septa. Scattered in the midst of the larger patches one or more comparatively healthy lobules are frequently found, of a pale brightish-pink colour, contrasting strongly with the hue of the surrounding tissue. The pleura which covers the part may have a slight milky-like aspect (Sir WILLIAM JENNER). It is also extremely probable that many of these thoracic lesions in such cases may be due to the direct passage of fibrinous particles from the large veins surrounding the diseased intestinal glands, for clots thus tend to form in the blood-vessels, near the site of irritation; they break up, the blood becomes contaminated, and the phenomena of *embolism* supervene in the branches of the pulmonary artery. The circumscribed patches of lobular pneumonia then become converted into small abscesses, or pass into gangrene. Such dangerous phenomena may be looked for from about the fourteenth day to the twenty-first day. The tissue of the darker portion of the lungs appears tougher than in health, presenting nearly a uniform section: there is no appearance of granules, and the part sinks in water. Sir William Jenner has injected such morbid lungs, and found that occasionally the centre of the lobule is really the point at which the diseased action is first set up. The development of the new material appears to be very deficient; molecular granular matter and delicate minute cell-forms compose its structure; and the specific gravity of the part is greatly increased (1.040 or more). Its colour is generally slate-grey or flesh-like; and the lesion is commonly limited by a vascular boundary, forming something like a distinct line of separation between comparatively healthy texture and local lesion. Carnification of the pulmonary tissue occurs often in considerable portions (WALSHE). The general debility of the typhoid state seems to favour the occurrence of pulmonary collapse—a state of *atelectasis* from collection of secretion or swelling of the mucous membrane of the bronchi leading to the collapsed air-vesicles, and rendering the passage impervious to air—a condition

which must not be confounded with the hepatisation of pneumonia. The bronchial glands are swollen and vascular, having an appearance similar to that of the mesenteric gland lesions.

X. The tracheal glands, and those of the bronchial mucous membrane, are also affected.

XI. There is generally a great tendency to ulceration of mucous membranes in typhoid cases; ulceration of the pharynx occurs in about *one-third* of the cases; of the larynx and œsophagus in one out of every fifteen cases; and the mucous membrane of the colon becomes implicated in seven out of twenty cases. Louis found the colon affected (*colotyphus*) by the second week in two out of fourteen cases, by the third week in six out of twenty-three cases, by the fourth week in four out of fifteen cases, and between the fifth and tenth week in one out of two cases. The solitary glands of the colon then undergo the same changes (*pari passu*) as those of the small intestine. Far more rarely the process extends to the jejunum and solitary glands of duodenum and pylorus. There is also a tendency to pericœcal abscess, preceded by the phenomena of the morbid state known by the name of *perityphlitis*, or inflammation of the areolar connective tissue surrounding the *caput cœcum*.

XII. Softening of the texture of the heart, as in typhus, is common also in enteric fever; but the softening is less often extreme.

XIII. The minute arteries of the brain, kidneys, and other organs are in a state of extreme fatty degeneration (HOFFMANN).

XIV. The blood during the first week of enteric fever resembles that of plethora, the corpuscles and solids of the serum, especially albumen, being increased; but about the ninth day the corpuscles and solids of the serum commence to diminish rapidly proportionate to the extent of the intestinal affection and diarrhœa. The number of white corpuscles is then increased, and this in proportion to involvement of Peyer and mesenteric glands, while fibrine is diminished. Reddish-black bodies, pigment granules, also abound, which are probably red corpuscles in disintegration (LEHMANN, VIRCHOW, HOFFMANN).

It will be seen that the essential lesions in enteric fever all belong to or involve parts of the lymphatic system. The glands involved are in a state of inflammation, due to the irritation of some poison absorbed from the bowel—the actual poison of the fever being usually swallowed with the ingesta—so that the more normally and actively the functions of the glands are performed, the more will they absorb the poison. Hence, enteric fever is most common in persons under thirty years of age. It is through involvement of Peyer's patches and the mesenteric glands, that the spleen and system generally become infected and excite intestinal catarrh and diarrhœa, either as the result of such contamination or of extension of inflammation to the glands of Lieberkühn, *i.e.*, to the mucous membrane generally.

Growth of Tubercle during Enteric Fever.—There is still another pulmonary condition which frequently occurs in enteric fever, and which may either complicate the progress of the case, or come on subsequently to the fever. It is the development of tubercle—not only in the lungs but elsewhere—which is much more common after enteric fever than after typhus. Usually when recovery takes place from enteric fever it is

complete; but in some cases, especially where there is hereditary predisposition to scrofula, an impetus or tendency seems to be given to the development of tubercles in the lungs. If the physical signs of bronchitis continue beyond the thirtieth day, or fourth week, combined with hurried and difficult breathing, and with the signs and symptoms of irritation of the lungs, with hectic fever, then there are good grounds for suspecting that the deposition of tubercle has commenced in the lungs. Dr. Stokes gives two sets of cases in which this deposit takes place. In one set a great quantity of tuberculous matter seems to be formed during the existence of the fever; and although, sometimes, such an occurrence may not have been suspected, yet the expectoration of pulmonary calculi, at periods of different duration after the convalescence, furnish strong proofs that such a lesion had taken place. In other cases, again, the cure may be effected through absorption, or by suppuration of the minute tuberculous points over the mucous surface of the bronchia. A doubtful convalescence, a quick pulse, and a hectic state, suggest such a state of things, especially when combined with persistent bronchitis. Erysipelas, phlebitis, parotitis, and such-like local inflammations, are not uncommon in cases of enteric fever. Such lesions may be excited by cold simply; but the absorption into the blood of putrid substances, from the ulcerating patches of Peyer or other diseased parts, may be usually, and probably correctly, considered to be the cause of most of the secondary inflammations already noticed to occur in cases of enteric fever. Dr. Parkes considers it probable, however, that deficient urinary excretion may have a share in their production (PARKES *On the Urine*, p. 254).

Such are the more obvious lesions which may develop themselves during the natural progress of *enteric fever*, the derangements and symptoms to which they give rise constituting new phenomena in its course. In some severe cases, however, the fever may destroy the patient in a few days; but, even at the earliest date at which death ever occurs, some of the lesions of enteric fever are capable of recognition. The lesions just noticed all arise after the fever has existed some time; and it appears now to be pretty well established that the intestinal lesion at least is a growth, which, in cases of recovery, follows first a progressive or developmental course, and afterwards retrogrades. Specific characters of the elements composing the growths are difficult to demonstrate by microscopic examination, although it is certain that the poison of enteric fever has a specific *vital* attribute peculiar to itself. The nature of this so-called "typhoid deposit" has been the subject of much discussion ever since 1842. It is a new growth rather than a deposit or infiltration; and is, in the first instance, confined to the gland-elements, and seems really to consist in a directly continuous development of the pre-existing cell or germinal elements of the diseased glands. Eventually it pervades the submucous areolar tissue as a yellowish-white substance, deposited or infiltrated in a layer beneath the gland-tissue, and above the muscular coat. The late Professor John Goodsir, in his descriptions of the morbid anatomy of the cases he dissected at Anstruther, in Fifeshire, was the first to point out that the new growth was in the first instance confined to the interior of the gland-tissue, which became much distended thereby. It has been held that the new growth up till recently has no specific

structure to distinguish it from other morbid growths (WEDL, VIRCHOW, and others); and although a specific "typhus cell" has been described and figured by Gruby, Vogel, Bennett, and others, its existence till recently has not been proven as microscopically characteristic. The material was not known to have a specific structure. The development of the new growth in the glands begins like a simple hypertrophy (or hyperplasia rather) of the natural cell-elements of the gland tissue. Nuclei and cells exist in great abundance, which afterwards degenerate into the abnormal diseased products which constitute the masses of new growth so pathognomonic of enteric fever lesion. No forms arise capable of sustained existence; but a directly continuous development from pre-existing cell or germinal elements of the glands and the connective tissue furnishes the material of the mass (GOODSIR, VIRCHOW). An increase in the colourless corpuscles takes place in the blood, and deposit of pigment in the ganglia of the sympathetic nerve-system (VIRCHOW). When the enlarged gland substance begins to die, the exuberance of new growth gives a fungating appearance to the part; and when the rose-red tumor is cut into, a milk-like turbid juice exudes: and in this juice many new-formed elements may be seen, consisting of cells, mostly oval or angular, with single eccentric nuclei—sometimes with many nuclei. The cell-contents are finely granular, and fat globules may conceal the nucleus. The growth must be examined before ulceration commences; for, as softening advances, a viscid fluid with a bloody tinge, containing fine molecular elements, is all that remains, with decaying blood and blood-crystals. Definite and positive statements have been made recently by Dr. Klein as to the minute pathology of the specific lesions found in the intestines. "*Peculiar organisms*" have been described by him as abundant in the lymphatics and venous vessels of the mucous membrane over and round Peyer's glands. These appearances, so minutely described by Dr. Klein in sheep-pox (*Trans. R. S.*, CLXV., p. 233) and in enteric fever, have been since shown by Mr. C. Creighton (*Proc. R. S.*, June 15, 1876, p. 140) to be altogether deceptive; and to be the result of coagulation common in mucus or albuminoid fluids. "Although Dr. Klein thought that he had before him in his preparations the various conditions of a fungus, to which he gave a generic and specific name; and although he professed to find the various conditions of spore, mycelium, and fructification occurring in their natural sequence, and that natural sequence to correspond with the regular advance of the pathological process, there is no doubt that this circumstantial account rests on erroneous observation and on defective evidence; and that the appearances found in the skin of the sheep (and in enteric fever) are none other than those exceptional forms resulting from the coagulation of albuminoid fluids under particular circumstances. Mistakes of this kind, as Mr. Creighton justly observes, greatly contribute to the spread of a reactionary and superficial pathology.

(c.) *Causation, Origin, and Propagation of Typhoid Fever.*—It is now nearly half a century since M. Bretonneau related to the French Academy of Medicine a series of cases in which the communication of this disease from person to person, and its modes of propagation in this way, were so evident as to admit of no reasonable doubt. Nevertheless, the conclusion arrived at has

not been generally accepted by the profession, so that the communicability of typhoid fever has not met with general belief. But "in so vital a question it is of the highest importance that the actual truth should be generally known" (BUDD). And Sir Thomas Watson, whose authority is undisputed, justly remarks that, "if this fever be really contagious, it is not only erroneous but dangerous to hold the contrary opinion." "To what extent it is dangerous," continues Dr. Budd, "may be best measured by the fact, that in this country alone 20,000 persons die annually of this fever, and 140,000 more are laid prostrate by it." Thus, vast is the field for the operation of preventive measures. And when the discovery and success of such measures must depend in a great degree on our insight into the real mode of propagation, it is at once seen what importance the question assumes. To what extent it is contagious there is abundant evidence to prove that typhoid fever is at once *specific* and *infectious*, while the question as to its spontaneous origin has been already fully considered at p. 358, *ante*, from which it is clear that the question of proof is a very difficult one to solve.

The immediate exhalations from the patient do not appear to contain much of the contagium, so that there is little danger from merely coming into the vicinity of patients suffering from enteric fever—a fact which justifies the admission of typhoid patients into the wards of general hospitals. In this respect it contrasts favourably with typhus, which cannot be so admitted. The probability of enteric fever spreading directly from the conditions surrounding the patient appears to be getting less and less, such as infection from breath, skin emanations, and fresh stools; but—given the enteric fever case—the surroundings (especially as to drains, water supply, and the like) are the media of its spread. The virulent part of the specific poison by which the disease is communicated is doubtless contained in the diarrhoeal discharges which issue from the diseased and *exanthematous* bowel. These discharges drying up, the germs of disease are thus preserved as effectually as the crusts of small-pox preserve the virus of that disease. If, therefore, through atmospheric or other agencies, these germs obtain access to the living body, diarrhoea is brought about in the usual course of events, and the commencement of the disease thus communicated takes place. The discharges from the bowels of the person so infected, which are at once copious, numerous, and liquid, are thrown into the water-closet or the privy, and the drains, or systems of drains, become at once saturated with the specific poison of the disease in its most concentrated and virulent form. Regarding, therefore, the drain, or system of drains, as a channel directly continuous with the diseased intestine of the infected person, the specific virus of typhoid fever may be propagated amongst healthy persons in one or more of the following ways, namely—

- (1.) By percolation through the soil into the wells which supply drinking-water to the inhabitants—the most common channel of conveyance.
- (2.) By issuing through defects in the sewers into the air of the inhabited area; or,
- (3.) By exhalation through the aperture of small, ill-trapped water-closets or privies, which are at once the receptacles of the discharges from the sick and the daily resort of the healthy.
- (4.) By overflow water-pipe connections between drinking-water cisterns and the drain.
- (5.) By contamination of milk from polluted water supply. When the

specific poison thus issues into the air, the *atmosphere generated is immeasurably* more likely to communicate the disease than that which immediately surrounds the fever patients. "There is reason to believe, however, that the duration of the period of incubation varies considerably, partly with the nature of the medium through which the specific poison finds admission to the living body, partly by reason of the conditions it meets with there, and still more so *in virtue of the greater or less intensity of the state of change* in which the poison itself may be at the moment of its reception." Hence the simultaneous seizure of a large number of persons within a definite interval after the occurrence of a single case points to some one or other of these modes of propagation. Such modes of propagation are thus the exact counterpart of what has been oftentimes observed (especially in schools) in the case of measles, scarlet fever, and, in former times, of small-pox also.

The hypothesis, on the other hand, of the spontaneous origin and indefinite propagation of enteric fever has assumed a definite form of expression in the doctrine which teaches that enteric fever is often actually caused by the products of common putrefaction—a doctrine which has been ingeniously embodied in the nomenclature of the subject by the late Dr. Murchison;* and thus a degree of precision and permanence has been given to the opinions he has so ably advocated in his classic work on *The Continued Fevers of Great Britain*—a volume which is unsurpassed for its erudition and its practical importance. The term "pythogenetic fever," or fever "born of putrescence," is the name by which the late Dr. Murchison at once designates enteric fever and theoretically implies its origin, believing that even in the specific stools the poison is always a product of decomposition—thus arguing strongly in favour of its origin independently of the disease which it generates. Without, however, doubting the fact that animal and vegetable substances in some states of decomposition have the power of inducing ill-health; and that there is now acknowledged to be a connection between putrid states of the air and the prevalence of pyæmia, erysipelas, puerperal fever, and cholera; yet there does not seem to be sufficient evidence to show that any of these causes can *produce* a disease which is of so *specific* a nature as to be maintained and propagated by a specific poison generated in the body alone. Undoubtedly, the state of ill-health induced by the decomposing material of night-soil and the like, does produce *a state of the system favourable to the development*, not alone of typhoid fever, but of many other specific diseases, such as cholera, dysentery, yellow fever, and the like. Dr. Carpenter, also, long ago showed physiologically, what observation has since confirmed—namely, that decaying animal material, especially night-soil, seems to be for some poisons (*e.g.*, cholera and yellow fever) great centres or foci, where the specific germs or poisons are able to multiply; and for the propagation of which "foulness of medium is indispensable." An interesting question

* As these pages are passing through the press, it is with very great regret the author has read of the sudden death of this most accomplished and highly esteemed physician, to whose "magistral" work on *Continued Fevers* he is largely indebted. Long unwearied study, and extreme care in recording results, enabled Dr. Murchison to do an amount of work in his life-time which is unsurpassed, if it is equalled by that of any other man (*Brit. Med. Journal*, April 26, 1879, p. 648).

or inquiry is thus opened up as to whether the germs of enteric fever, cholera, and the like, could be made experimentally to grow or increase upon or about organic matter, just as the germs or spores of many fungi are induced to grow in collections of manure (Dr. LANKESTER). In such collections on the earth's surface there is reason to believe that the contagia of diseases like cholera, and enteric fever, and yellow fever, may find a resting-place—that thus they are always extant somewhere; although it may be only now and then, when season and other conditions conspire, that they display their full power as epidemic diseases. As such, they seem to occur every now and then in “mysterious cycles,” the existence of which we admit, but do not understand. The experiments of Dr. Barker on cesspool air prove that long inhalation of an atmosphere charged with the gases evolved from decomposing organic matter is capable of producing a series of symptoms of the following character—namely, increased heat of skin, thirst, irregular and feeble muscular contractions, and diarrhoea. These symptoms continue so long as the person is exposed to the influence of the foul air; but when the cause is removed, there is no continuance of symptoms, no recurrence or remittency, but a tendency to recovery; and no communicable disorder is induced. The poison of the foul air acts for the time as chloroform might act, and so soon as removed recovery progresses. The history of enteric fever, whose leading features have been described in the previous pages, is wholly inexplicable upon the “pythogenetic theory” of Dr. Murchison. On the contrary, it is emphatically the history of a specific disease generating a specific poison, and propagating itself by it. As the hidden sources of pollution in regard to the propagation of this fever are so extremely varied and extensive, there is a most urgent necessity for exploring every possible means by which enteric fever may be spread. Until such an exploration is made and all these hidden sources exhausted, the doctrine of spontaneous origin rests upon thin air, and one of the strongest motives for precaution is absent. Some cases of typhoid fever are in fact so mild or so ill-developed that they are not recognised—the malady is not diagnosed; and hence inquiry for sources of infection is baffled. It is necessary under all circumstances to keep in view the fact that there is one mode of contamination which may be said to take effect at what may be called a “long range,” by the formation of *soak-holes*, *swallow-holes*, *dead-wells*, *dry-wells*, or *puits d'absorption*.* Thus it happens

* These are illustrations of the futile attempt to get rid of sewage. Once on a time, solid and liquid sewage were received into cesspits, where the contents leaked away slowly, having little water with them, and were emptied from time to time. A suburban builder thought there was ample provision for getting rid of sewage and for water-supply if every house had a small yard, with just room in it for a pump and a privy. Then came the water-closet, with its indoor means of contamination from the absorption of gases by the cisterns, which, till lately, were used both for drinking and closet purposes; and the admission of reflux sewer-gas into the very centre of houses, through every sink and pipe by which water or slops might pass away,—this gas being nominally kept out by *traps*. The water-closet system generates an enormous bulk of polluted water, defying many of the attempts to deal with it, and spreading foulness wherever it goes. Given a country house, one of two courses is pursued with the sewage: either it is turned into the nearest ditch, to infest the air, and evaporate at leisure; or, if the soil be porous, it is turned into a dead-well, to soak away in the earth. Here, of course, it penetrates into any neighbouring well, and may travel long distances. Hence, in case of the “spontaneous” origin of typhoid in some simi-

that several severe outbreaks and propagation of enteric fever have been distinctly traced to the use of infected milk—notably the Marylebone epidemic, so courageously brought to light by the late Dr. Murchison, and ably investigated by Dr. Ballard; the epidemic at Jarrow, investigated by Mr. John Spear; at Glasgow, investigated by Dr. Russell; and the Easley epidemic, reported upon by Mr. Robinson.

Enteric fever is essentially an endemic disease; and is, in fact, *the endemic fever* of England, France, and America. Its prevalence in a place is quite independent of typhus fever; and it has formed no part of the great fever epidemics of this country. Cases have been met with in the epidemics, just as cases of measles and small-pox. The number of cases never increased towards the end of any epidemic of typhus, so as to take the place of typhus, and so continue the epidemic. For sixteen years before 1865 enteric fever had remained at about the same annual prevalence. From 1865 to 1870 there had been a great increase of cases in London—an increase which appears to have been contemporaneous with the development of the main drainage scheme. In Glasgow it is endemic, and its prevalence quite independent of typhus. It appears to have considerably diminished since the introduction of the excellent water supply to that town from Loch Katrine. It is similarly endemic in Edinburgh, and its prevalence is in no way affected by epidemics of typhus fever. It appears to be much more common in Edinburgh now than formerly—an increase which has followed the introduction of new sanitary arrangements—namely, the substitution for scavengers and night-men of drains opening into the interior of houses, and having a water-supply quite insufficient to prevent the escape of sewage emanations. The epidemics of enteric fever are local and circumscribed—sometimes confined to a single house, or district, or village. Hence it has often been named from localities where it has prevailed—as Croydon-, Marylebone-, Westminster-, Winchester-, Cowbridge-, Windsor-fever.

The disease in certain places seems never to be absent, and is invariably most prevalent during autumn, at the time that diarrhoea is most common; and it has been observed to be especially prevalent in seasons remarkable for their high temperature. In the winter months of December and January the cases are more numerous than in June and July. At the end of autumn the disease is at its maximum, and continues to decrease till April, when it is at its minimum; after which it tends again to progressively increase through the summer and autumn months. It is similarly prevalent on the Continent in autumn; and in America it is so much so that it has been there called the “Autumnal” or “Fall fever” (MURCHISON). Warm damp weather, when drains are most offensive, is the kind of weather apt to be followed by outbreaks of enteric fever. Its origin is quite independent of overcrowding.

ing rural village or detached country house, it will be expedient to explore not only the sewage arrangements of the infected place, but to cast an eye on the isolated farm-houses and country seats in the neighbourhood, more especially if the soil be chalk or sand. It will be expedient, too, that the insidious nuisance constituted by this substitute for drainage should be denounced and rendered illegal (*Med. Times and Gazette*, Oct. 30, 1875).

Symptoms, Course, and Duration.—Enteric fever begins gradually—often, indeed, so very insidiously that its commencement is not always able to be fixed. Hence the period of incubation is difficult to determine. It is most commonly about two weeks; but it is often less, and may not exceed one or two days. Instances of longer duration appear to be more common than in typhus or relapsing fever (MURCHISON in *St. Thomas Hospital Reports*, Vol. II.) The fever is preceded by indefinite feelings of general illness, mental depression, great dulness and relaxation, loss of appetite, indigestion, restless sleep disturbed by dreams, headache, dizziness, wandering pains in the limbs, usually considered rheumatic, and repeated epistaxis (NIEMEYER). When the commencement can be fixed, it is found that the fever proper may be ushered in with a distinct chill or rigors, or profuse diarrhœa. The chill is rarely so severe or continued as in the malarious fevers, or in pneumonia. Amongst the earlier symptoms, the most characteristic are the abdominal pains and diarrhœa, which continue to increase. The pulse is quickened, the skin hot; the tongue is furred and white in the centre and red at margins and tip. The countenance indicates anxiety, the mind continues clear; but delirium, when present, is generally active; the patients are vivacious and disposed to leave their beds. The conjunctivæ are pale, the pupils dilated, the cheeks somewhat flushed, and epistaxis not seldom occurs at repeated intervals during the first week. The nights are restless, weakness increases, and there is great disinclination for mental or bodily exertion; but the patient rarely takes to bed before the sixth day, and may even continue to walk about for a fortnight. Gurgling on firm pressure may commonly be detected in the right iliac fossa, and there is often tenderness in the same situation. The occurrence of diarrhœa, or gastric disturbance, with an evening temperature of 103° or 104° Fahr., and prostration in a young person ought always to make the practitioner suspect that enteric fever is the disease with which he has to do (MURCHISON). As a rule, the flushing of the face is more marked towards evening; but the complexion does not get muddy, as in typhus, and the flush of the cheeks is bright and pinkish—not dark-red—often circumscribed, and then strongly contrasts with the surrounding pale skin. From the seventh to the fourteenth day the characteristic eruption appears.

During the third week the abdomen becomes more distended; the diarrhœa (which is due to intestinal catarrh and not to ulceration) increases—the stools amounting to *five, six, eight, and ten*, or even *twenty* a day. Three to four stools a day is about the usual number in a favourable case. They are liquid, pale brownish-yellow, with flocculi of an opaque whitish-yellow colour floating through them, like coarse bran, or like badly cooked pea-soup, in which the meal is not thoroughly pulped, and sinks in particles to the bottom of the containing vessel. As the patient loses strength these stools are passed involuntarily. Pain is rarely complained of, unless perforation of the gut occurs; and hæmorrhage from the bowel is an occasional symptom during the third or fourth week.

The frequency of the pulse often varies much from day to day, a marked peculiarity of enteric fever, without any appreciable coincident alteration in the general or local symptoms. There may be a difference of even

ten, twenty, or thirty beats between the morning and evening, and these differences are most marked in mild cases, and in the earlier stages of more severe forms of the disease (MURCHISON). But enteric fever may run its course with a pulse little if at all above the normal frequency (sixty to eighty), the temperature however reaching 103° or 104° Fahr. It is generally soft, sometimes feeble and slow relatively to the height of temperature. This slowness of the circulation has no doubt to do with promoting the development of bed-sores. In such cases (which are generally prolonged) the healing of the intestinal lesions are retarded, and as the patient becomes weaker, he slips down in bed, and bed-sores may begin to form over the *sacrum*, or *trochanters*, or *elbows*, or *knees*, often causing great destruction, and they are even produced if the patient is laid on his belly (NIEMEYER). Emaciation in such cases becomes excessive, and the pallid skin is constantly bathed in perspiration. The gums and lips become pale and bloodless, and œdema of the limbs may supervene.

The tongue at first is red and fissured, but ultimately becomes dry and covered with a pale-brown fur. At first it is rarely thickly coated, but broad and moist, showing the impressions of the teeth along its edges (œdematous). The coating consists of a thin whitish epithelium, through which the papillæ may project as red points. Afterwards it becomes coated with a rough slimy mucus. The coating generally falls off after the fifth or sixth day, leaving "a moist, red, smooth tongue, that looks as if covered with goldbeaters' skin, or else is already inclined to dryness. If there be at first a thick, adherent coating on the tongue, it is usually detached from the point, and from the sides towards the middle, so that the whitish-yellow coating appears inclosed in a very red border, which constantly increases in width. But in some cases the detachment of the coating begins centrally, so that in the middle of the tongue there is a red stripe, that has a peculiar tendency to become dry, and at the sides two whitish-yellow, moist, slimy stripes. The central stripe is often broad anteriorly, and disappears posteriorly, so that on the point of the tongue we see a red triangle with the apex posteriorly" (VOGEL, NIEMEYER).

The splenic dulness is increased, the enlargement being generally well marked towards the end of the first week. The enlarged organ usually assumes the horizontal position. It rarely projects beyond the ribs, and is apt to be pressed upwards and backwards against the spinal column by the distended intestines. Hence the enlarged spleen of enteric fever is rarely to be reached on palpation, and even when it can be touched it is dangerous to attempt to define its outlines with any degree of pressure, for it is so soft. If the patient is laid on his right side, with the left hand over his head, then on percussing the lower ribs of the left side, a dull space may be detected, about six inches long by four inches wide, corresponding to the eighth, ninth, and tenth ribs, and reaching from the spinal column posteriorly to the margins of the ribs anteriorly. It is the region of splenic dulness; but only a small part of a very large spleen may be thus detected (NIEMEYER). Pulmonic complication is not uncommon.

The symptoms of typhoid fever cannot be said to be fully expressed until the appearance of the characteristic—

Eruption, which consists of the so-called *rose spots* peculiar to *typhoid* fever, the "*taches rosées lenticulaires*" of Louis. These spots begin to appear from the sixth or seventh to the twelfth or fourteenth day of the disease, very rarely later, and still more rarely at an earlier period than the sixth day. A very delicate scarlet tint of the whole skin, closely resembling the skin of a person soon after leaving a hot bath, sometimes precedes, by a day or two, the characteristic eruption of typhoid fever; and this is important to remember, because it may be mistaken for the rash of scarlet fever, especially if sore throat is present. The eruption consists of slightly elevated papulæ or pimples; but, to detect their elevation, the finger must be passed very delicately over the surface of the skin, because although pimples they are not hard, like the first day's eruption of small-pox. Their apices are neither acuminate nor flat, but invariably lens-shaped or rounded, and the bases gradually pass into the level of the surrounding cuticle. No trace of vesication can be detected on their apices. They are circular, and of a bright-rose colour. Their margin is well defined, and *they disappear completely on pressure*, resuming their characteristic appearances as soon as the pressure is removed. These characters they preserve from their first appearance to their last trace. They leave behind no pit, scar, or stain. They vary in size, but their usual diameter is nearly 2 lines, but varying from 1 to $2\frac{1}{2}$. The ordinary duration of each papula is about two days, but its existence varies from two to six days, and fresh ones generally make their appearance every day or two after the first appearance of eruption, and they continue to appear in successive crops till the twenty-first or twenty-eighth day of the disease. Sometimes only one or two are present at first, after which one or more fresh ones make their appearance. The eruption of such spots does not consist of a great number at one time—only from six to twenty, which occupy usually the abdomen, thorax, and back; but may be present on the extremities, sometimes, though rarely, so thickly seated that scarcely an interval of normal cuticle is left between. THIS SUCCESSIVE DAILY ERUPTION OF A FEW SMALL, VERY SLIGHTLY ELEVATED, ROSE-COLOURED SPOTS, DISAPPEARING ON PRESSURE, EACH SPOT CONTINUING VISIBLE FOR THREE OR FOUR DAYS ONLY, IS PECULIAR TO AND ABSOLUTELY DIAGNOSTIC OF TYPHOID FEVER.

The eruption is, however, often so scanty that the physician may justly hesitate for a day or two to make a diagnosis. The first crop of the eruption is rarely quite decisive; but as soon as successive crops, even of two or three spots each, appear, all doubt is removed. When the eruption is scanty, it is advisable to surround each individual spot with an ink line, in such a way as to distinguish accurately the period of its appearance (see p. 293, *ante*). It is the occurrence of this eruption which clinches the diagnosis;—a diagnosis which becomes absolute, as regards typhoid fever, when, in a febrile disease attended by diarrhœa, or simply looseness, unequivocal rose spots appear on the sixth or eighth day. If they do not appear then, the diagnosis cannot be said to be complete till the case has been watched for several days, and the age of the patient, and the history of the illness, have been fully and carefully studied. In children between *one* and *five* years of age the phenomena do not seem to be so easily observed as in adults.

It has been now clearly established that typhoid fever is by no means

an unfrequent disease amongst children, and has been often described under the name of "*Infantile remittent fever*." Boys seem to be more liable to attack than girls. It is most frequent between six and eleven years of age; and from five to nine seems the period of greatest liability. Its occurrence is rare during the first years of life. Nevertheless, it is on record at the following very early ages:—namely, between two and three months; three months; six months; seven, ten, and thirteen months (WUNDERLICH, HENNIG, FRIEDRICH, RILLIET). The author of a very interesting Review on the Enteric Fever of Children, in the *British and Foreign Medico-Chirurgical Review* for July 1858, p. 161, mentions, in his own experience, the occurrence of enteric fever in a girl one year and seven months old; and also in a boy two years of age. The chief symptoms of enteric fever in the child are,—splenic enlargement, diarrhœa, meteorism, gurgling in the course of the colon; associated with pyrexia, quickened respiration, bronchial catarrh; delirium, somnolency. The eruption already described and sudamina are nearly constant in children after five years of age. The rose-coloured spots are especially frequent on the back and the extremities, so that, if the abdomen and chest only are examined, their presence may often not be apparent.

The Temperature during Typhoid or Intestinal Fever.—Wunderlich has given a summary of results derived from the observation of 700 cases of typhoid fever investigated thermometrically (*Arch. der Heilk.*, Vol. II., 1861, p. 433; also, *Edin. Med. Journal*, Nov., 1862, p. 465). The course of the disease is typical, and the type is characteristic, distinguishing enteric fever from every other disease; and when irregular cases occur, irregularity may sometimes be traced to a special cause. The course of the disease shows two sharply bounded distinct periods, which correspond to the deposition and reabsorption of the new growth within the intestinal glands. These periods seem to agree with certain portions of time, and correspond to the first and last half of the disease. In mild cases the first period lasts about a week or a week and a half; in severe cases it extends to two, three, or three and a half weeks; so that the entire disease (measured by the records of temperature) lasts five, six, or occasionally from eight to ten weeks. The mode of accession is pretty nearly the same in mild as in severe cases. Increase of temperature to the extent of two degrees in the evening, and remissions to the extent of one degree in the morning, follow one another for about three days, the temperature every morning and every evening being about $2\cdot2^{\circ}$ Fahr. higher than on the preceding morning and evening, while the morning temperature is generally about $1\cdot1^{\circ}$ lower than that of the previous evening; or, according to the following formula:—*First day*, morning, $98\cdot5^{\circ}$; evening, $100\cdot5^{\circ}$; *second day*, morning, $99\cdot5^{\circ}$; evening, $101\cdot4^{\circ}$; *third day*, morning, $100\cdot5^{\circ}$; evening, $102\cdot5^{\circ}$; *fourth day*, morning, $101\cdot5^{\circ}$; evening, 104° Fahr. In the second half of the week the evening temperature is from 103° to 104° ,—the morning temperature about a degree lower. Between the fourth and sixth day the fastigium or height of the fever is attained (MURCHISON), when the temperature in the evening amounts to $103\cdot5^{\circ}$, 104° , or 105° , or even 106° Fahr.; but the diagnosis of enteric fever must not be excluded if it does not reach 103° Fahr., as Wunderlich teaches. By the end of the first week the patient becomes very weak and is much prostrated, with great thirst and no

appetite. From this time onwards the fever proceeds in regular stages of weekly and half-weekly periods. On the other hand, the disease may always be recognised when there is in the evening hours a persistent elevation of temperature; and a decided increase of temperature during the first week is generally an unfavourable sign, while a slight increase is favourable. During the second half of the first week both mild and severe cases follow the same course, so that, for the purposes of prognosis, the determination of temperature is of little consequence during the first week. It is only in the maxima of the temperature that sometimes a difference is visible between very mild and very severe attacks. In the mild cases there is now and then a large decrease of temperature observable towards the end of the first week—namely, from 105.8° to perhaps 102.5° Fahr.

The complications of enteric fever generally make their appearance about the third week, and threaten or tend to a fatal end up to the very beginning of convalescence. In the mild types the growths in the intestinal glands are no doubt such as are reabsorbed without any ulceration. The severe cases owe their severity partly to the more extensive growth of new material in Peyer's glands, partly to the mode of elimination of that material—the healing of the parts being accomplished under great excitement of vascular reaction, renewed hyperæmia, sloughing, softening, and final cicatrisation. Cases intermediate in severity between the mild and severe cases just described are not unfrequently met with. Many of them, although they show a course more or less irregular, nevertheless follow a pretty clearly defined type as to variations of temperature, and are capable of clinical recognition. There are still considerable evening exacerbations during the second week, yet with a tendency to abatements in the mornings. During the third week, great vacillations between morning and evening temperature continue, and sometimes also between single days. During the fourth or fifth week the normal temperature is reached in the morning; but it is only in the fifth or sixth week that the temperature becomes permanently normal—the evening temperature showing a complete freedom from fever; so that the beginning of convalescence can only be established with certainty by the use of the thermometer. In the majority of cases of enteric fever, severe as well as mild, a peculiar periodicity of weeks and half weeks cannot be mistaken. Each week shows a distinct character, which cannot be overlooked in a graphic representation. On the first and last days of each week changes generally take place which are either temporary changes or continue till the fever subsides. There are cases so mild that towards the end of the second or beginning of the third week the symptoms have all disappeared. Lebert has proposed the designation of “abortive” for those cases; and on this point Niemeyer makes some very important observations. He considers the designation preferable to such names as “*febricula*,” “*febris typhoides*,” because it better expresses the fact that these cases are only modified, benign, brief forms of enteric fever, and not a peculiar variety of disease. . . . Many of the cases which former writers call “gastric fever,” or “mucous fever,” are to be regarded as abortive typhoid. The old customary precaution of *waiting till the ninth day* of the disease, before saying whether the case was “one of gastric or nervous fever,” was and still is very justifiable. The designations *gastric* and *nervous* fever, in common use, exactly correspond to what

modern physicians mean by "*abortive enteric fever*," or, as the Germans call it, "*abortive typhus*." In the first week of the disease, the thermometer is the most certain means of distinguishing enteric fever from a genuine attack of febrile, gastric, and intestinal catarrh; so, in the second week, it is the most certain means of deciding whether the disease will be an abortive case of enteric fever or not—the other symptoms being far more deceptive, while the use of the thermometer is decisive. If we find that on the eighth or ninth day of the disease the temperature ceases to rise, but gradually falls, and especially if we find decided morning remissions at this time, we may be almost certain that the case is one of abortive enteric fever (NIEMEYER). The lesions in the intestines are got rid of as described at p. 590, without ulceration and by reabsorption. No opportunity is given for *post-mortem* examination; and the lesions cannot be shown in museums; they can only be seen if the patient dies of some other diseases, as in the case of the soldier (at p. 590) dying in the middle of an attack of enteric fever from rupture of an aneurism.

The temperature varies much in different cases of enteric fever; and no series of cases, however well marked, gives an average that can be considered typical of all. Among the most characteristic features are the following:—The period of ascent is gradual—a little each day, to the extent of a degree and a half, with a remission in the morning, to the extent of one degree or under. This gradual and general ascent is invariable for four days at least, or five, and is not influenced by remedies. Such being the rule, a temperature of 104° Fahr. on the second day is not enteric fever; a temperature which is not above 102·5° Fahr. after the evening of the fourth day is not enteric fever; and lastly, a patient after the first day, whose temperature has been normal *once* during the first week, is not a case of enteric fever. A stationary period may be observed from the seventh to twenty-third day, when there are no more ascents, and with slight morning remissions the evening temperature is variable. It may be lower by a degree or a degree and a half for a day or two, and again rise; and if maintained at 105° Fahr. for two weeks or more, the case is one of great severity. As the descending period begins, the morning remissions are more marked, while evening temperature remains the same. After three or four days the evening decrease begins, while morning remissions become greater. Oscillations occur to the extent of 1° or 1·5°, or 2° or 3° Fahr., extending over from seven to twenty-one days. The ascending and stationary periods of temperature correspond to the period of infarction or infiltration of the intestinal glands, with the new growth, and with the elimination of the products. The descending period of temperature corresponds to the period of repair.

Duration of Attack and the Mode of Recovery, or the transition into the feverless state, is peculiar and characteristic of enteric fever. With rare exceptions, the defervescence is a remittent one. The great vacillations of temperature between morning and evening recur for a longer or shorter interval. For weeks the evening temperature may amount to 104° Fahr. or more, whilst in the morning the patient is almost quite free from fever. At the same time the transition into the feverless condition may follow different courses. The remissions may either become longer and longer—the morning temperature decreasing and the evening remaining stationary;

RANGE OF TEMPERATURE IN A SEVERE AND PROLONGED CASE OF ENTERIC FEVER. THE RECORDS INDICATE MORNING (A.M.) AND EVENING (P.M.) OBSERVATIONS (Wunderlich).

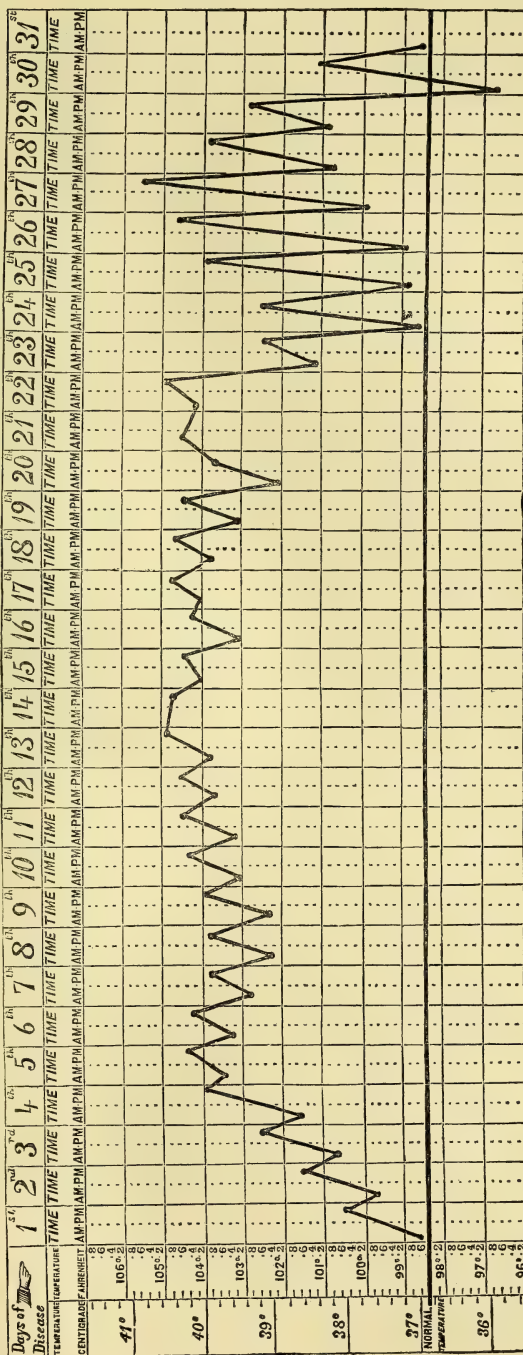


Fig 85.

or after some time the remission may become shorter and shorter—the evening temperature, together with the morning temperature, gradually descending. Again, the differences between the morning and the evening temperatures may remain nearly the same, while a relative decrease takes place at both periods; or the fever shows a sudden transition into the remissions with low temperatures—changes which generally correspond with the commencement of weeks. The period of development of the disease occupies two weeks, or a week and a half in slight cases; in severe cases it may occupy two and a half to three weeks. The initial stage (that is, the period when the growth of material in Peyer's patches takes place) lasts about half a week. The removal or elimination of the growth may take place in a week; but the process may extend over several weeks. In mild cases the disease continues at its height for only a week or a week and a half,—rarely for two weeks; so that the whole duration of a mild case of typhoid fever extends from eleven to eighteen days. The period of convalescence occupies from one to two weeks. The whole disease, therefore, in mild cases, may be gone through in from three to four weeks,—rarely in two weeks and a half. But it is extremely difficult to fix the precise date of the commencement of enteric fever, hence it is not less difficult to fix its entire duration. In severe cases the disease continues at its height from two weeks and a half to three weeks and a half. Then an undecided period of irregular duration succeeds, after which decided abatement is established, the defervescence occupying a week, followed by another week of convalescence. Consequently, the whole disease extends from four and a half to ten weeks, or even longer. Regarding the mean duration of illness in *typhoid fever* considerable differences of statement are to be found—a circumstance not to be wondered at when the nature and seat of the pathognomonic lesions of this form of fever are recognised as influencing the duration of the disease. Dr. Shattuck assigns the mean duration of *typhoid fever* to be 22 to 24 days; the mean duration of the Parisian cases of 1839-40 were 19·6 days; Dr. Jackson's experience in America gives 22 days; Dr. Jenner's experience in London leads him to give 21 to 30 days; and the late Dr. Murchison 24·6 days. The mean of these varied statements gives nearly twenty-three days.

It is now well known that during the progress of this form of fever there is a repetition of the development of new material in the individual gland-vesicles of the intestine, and consequently a succession of retrograde metamorphoses; so that, in many cases of *enteric fever*, it is not unusual to have the malady prolonged throughout a course nearly double as long as that of *typhus*; and that, undoubtedly, the influence of the secondary local lesions of *enteric fever* is great in protracting the disease. Thus it is that a very indefinite idea of its duration prevails; and, as Sir William Jenner has shown, it is of the greatest importance to know when the original fever ceases, after which we are to consider the subsequent symptoms as due to the effects produced by the local lesions. As long, then, as fresh eruption continues to appear, the fever cannot be regarded as having terminated.

True relapses are occasionally observed. They occur about ten days or a fortnight after convalescence from the first attack, and are marked by

a return of all the former symptoms; while the duration of the attack is usually shorter than that of the first; and, according to the experience of Murchison, it is more severe. Such relapses are most common in autumn; and are indicated by an elevation of temperature. In the *Australian Medical Journal* for August, 1867, Dr. P. H. Macgillivray records some interesting observations on enteric fever, and gives diagrams of temperature, one of which shows a fatal relapse, beginning on the twenty-ninth day, and ascending by a gradual continuous ascent of temperature during four days, exactly like a case originally commencing, till it reached 105.2° Fahr. The case terminated fatally on the forty-fourth day. During the first attack the eruption was scanty; in the relapse it was abundant. Diarrhoea and great prostration, with delirium, characterised the first attack. In the relapse diarrhoea was slight, deafness great, and no delirium. Death was due to bronchitis, which had scarcely existed in the first attack.

The influence of hæmorrhage from the bowels in reducing temperature has been also well shown in a case recorded by the late Dr. Parkes. It occurred in a female twenty-five years of age. Diarrhoea was considerable; and blood was largely passed in fluid stools the night before the seventeenth day of the fever. On the morning of the seventeenth day the temperature was as low as 93° Fahr., rising in the evening to about 101° Fahr. After the eighteenth day diarrhoea ceased; but the differences between the morning and evening temperatures continued to be very great; and it was not till the twenty-sixth day that these differences began to grow less and less. A sudden rise of pulse with a fall of temperature after the fourteenth day points to intestinal hæmorrhage, even though no blood may yet have been passed by the bowel.

Condition of the Urine in Enteric Fever.—It is not till the third or fourth day of the fever that the urine assumes any special characters. It is peculiar in the following respects:—

I. As to normal constituents:—(1.) *The water* is greatly diminished, generally about one-half or even to one-fourth or one-sixth. This lessening of the water is most marked during the first week; it then begins to increase gradually during the second and third weeks; and at the end of the fourth week, in favourable cases, it has reached its normal standard. (2.) *The whole amount* of the urine does not seem to stand in any close relation to the febrile heat; but when the temperature begins to fall permanently, the urine increases at once, or very soon after. (3.) *The specific gravity* of the urine is high in almost all cases where the urine is scanty; and at convalescence the specific gravity diminishes, sometimes before the amount of water increases—*i.e.*, at convalescence the lessening of the solids of the urine is often prior to the increase in the water. (4.) *The urea*, as a rule, seems to be augmented, during the febrile period, above the physiological standard proper to the individual; and it sinks again below this standard during convalescence. The amount of increase varies: Vogel has noted 78 grammes, or 1,200 grains, in twenty-four hours; while Parkes has noted 57 grammes, or 880 grains, in that time. In most of the cases observed by Dr. Parkes the average increase has been about one-fifth above the physiological standard proper to the individual; and the augmentation is most marked in the first week, when the water

and the chloride of sodium are at the lowest point; and if the fever be continued beyond the third or fourth week, the urea keeps up in amount. The relation of urea to temperature is yet uncertain. (5.) *The chloride of sodium* is diminished (indefinitely); the cause of the diminution being in part due to the lessened ingress of this substance on account of spare diet, or due to the elimination of large quantities of it with the stools or the sweat. (6.) *The uric acid* is uniformly increased in amount; and it is relatively greater than that of the urea. It is often doubled in amount; and the increase progresses up to the fourteenth day, when it is at its greatest. It then diminishes to the twenty-first or twenty-eighth day; and during convalescence falls below the normal amount. Spontaneous deposits of urates occur very frequently, and when there is no such deposit it may be brought about by a drop of acid; but as yet the fact has no particular significance. (7.) *The sulphuric acid and phosphoric acids* maintain their amounts very much the same as in health, and sometimes a little above that; and seeing that much of the former is derived from food, its abundance in typhoid fever would indicate active tissue-change, when little or no food is being taken. (8.) *The pigment* at first is sometimes enormously increased, measured after Vogel's method (by comparison with a scale of colours). It has sometimes amounted to 80 or 100 in twenty-four hours, the normal amount being 3 to 6 (VOGEL). This is to be referred to increased disintegration of blood-cells; it is therefore much more highly coloured than the mere concentration will account for, as seen by adding water. (9.) *The acidity of the urine* appears always great during the early period, simply from concentration; but by neutralisation with an alkali it is found actually to be below the average by one-fifth, or even by one-fourth. During the third week the acidity still continues to lessen; and ultimately the urine may even become alkaline from fixed alkali. It may also become alkaline from ureal decomposition, soon after being passed. Therefore it is necessary, in all observations on this point, to distinguish carefully between the alkalinity due to fixed alkali and that due to ammonia.

II. As to abnormal constituents:—(1.) Albumen occurred in 33·3 per cent. of the cases examined by Dr. Parkes. In 23 per cent. of these cases it was temporary, and entirely disappeared before the patients left the hospital. In the other cases it was permanent, and in one of these a very profound kidney lesion, which had not previously existed, was immediately excited by the fever. (2.) Renal epithelium, casts, and blood are sometimes seen in the cases with temporary albuminuria—the blood generally in microscopic quantities; although in bad cases it may be greater in amount. Different phenomena in the course of typhoid fever variously affect the urine. The effect of diarrhoea is to diminish both water and solids, the chloride of sodium especially. Non-excretion of urea, or deficiency in its solid matters, often coincides with the putrid, adynamic, or profound “typhoid” state, and with symptoms which imply more or less blood poisoning from retention. Local lesion in the kidney may lead to this, or, from failing circulation, less blood may pass through the renal vessels, or there may be, as Dr. Parkes suggests, some special condition or combination of urea which hinders transudation. Such non-excretion is most apt to supervene during the third or fourth week, when

the first stage of the disease is over, and when the growths in Peyer's glands, and in the mesenteric glands, are softening, when the secondary blood poisoning occurs, and when the heart's action tends most to fail. Judging from the urine alone, the febrile action appears strongest in the first week of enteric or intestinal fever (typhoid), although the temperature is highest in the second and commencement of the third week.

Diagnosis.—Dr. J. T. MacLagan has well shown that there are two classes of cases of enteric fever in which difficulties of diagnosis are apt to be experienced, namely:—(a.) Cases in which the head-symptoms are so prominent and so early as to cause the case to resemble typhus. In these cases the range of temperature may not be sufficiently characteristic (from its height and irregularity) to be of use in the discrimination of the two diseases. In such cases examination of the conjunctivæ may be suggestive. Cases of typhus with marked cerebral symptoms have the conjunctivæ injected, the eyes suffused, and the pupils small. Cases of enteric fever, on the other hand, with severe cerebral symptoms, do not have the conjunctivæ injected, but the sclerotics are clear and pearly, and the pupils are large. (b.) Cases of enteric fever in which the symptoms are so mild and equivocal, and of such short duration that a diagnosis is impossible. The thermometer gives little or no aid in such cases; but with commencing defervescence it supplies the only certain information that can be got regarding the true nature of the case. In typical cases of enteric fever, the mode of ascent of the temperature, and its persistence with the characteristic descending oscillations, distinguish enteric fever from all other febrile states. In *pneumonia* the ascent is much more sudden. In *ague* temperature becomes normal during some part of the day. In *gastric intestinal catarrh* the height of the temperature on the second day may be above that of enteric fever, and the remissions are much more considerable. In *acute tubercle* the oscillations of temperature are as irregular as in enteric fever they are regular, and there is no fixed point as to maximum of temperature. The morning remissions are always more considerable than in enteric fever; there is no stationary period, and no period of decline (Fox, *St. George's Hosp. Reports*, Vol. IV.)

The leading diagnostic points, between the range of temperature of typhus and that of enteric fever, are briefly and clearly stated in the following table (*British and Foreign Med-Chir. Review* for October, 1868):—

Typhus Fever.

The duration of elevated temperature is very rarely beyond eighteen days; it is generally shorter by several days, and may be even so short as nine days.

The evening temperature is frequently lower than that of the morning.

The difference between the morning and evening temperature, during the height of the fever, or from about the third to the tenth or eleventh day, is comparatively seldom above one degree, and although about the period of defervescence the difference is sometimes much greater, the oscillation is not continued over more than one or two days.

Enteric Fever.

The duration of elevated temperature is very rarely less than twenty-one days; it is generally longer, and may be protracted to thirty-five days or even more.

The evening temperature is almost constantly higher than that of the morning.

The difference between the morning and evening temperature is generally, throughout the case, greater than in typhus, and towards the end of the fever there occurs the very characteristic oscillation of temperature, during which the difference is frequently five, six, or even seven degrees, and which may continue from a few days to a week or more.

Typhus Fever.

A high temperature is, as a rule, accompanied by a high pulse.

Enteric Fever.

A high temperature is frequently accompanied by a pulse but slightly accelerated, and occasionally by a pulse slower than normal.

Many cases of enteric fever markedly resemble remittent fever by variations in the pulse and temperature between the morning and evening, and also especially when the remissions are attended by perspiration.

Prognosis.—The average mortality among cases of typhoid fever appears to be about 1 in $5\frac{1}{2}$ to 1 in 6. It is considerably less in autumn than in spring; and is least of all in winter. It tends to be greater among males than females; and the average age of the fatal cases appears to be about 23·5; while the mortality increases to a small extent as life advances. In severe typhoid fever the prognosis appears to be more favourable in proportion to the free excretion of urea and uric acid (PARKES). The excretion of these effete products is a most necessary point. There is more danger *in the retention* than in any amount of fever and *formation of them with elimination*. The greater the excretion in typhoid fever the better; and as long as 500 to 700 grains of urea in men, or 300 to 500 in women, are being passed in each twenty-four hours, the progress so far is favourable. But whenever, while the fever continues, the urea falls much below these amounts, we may anticipate a low typhoid condition, or some local inflammation, as pleurisy, which may relieve the blood for a time from some of the effete products, but which at the same time may kill the patient. The existence of slight albuminuria or hæmaturia is not of itself unfavourable; but if either be in large amount, or if there be exfoliation of epithelium or renal cylinders present in the urine, retention of urea and its consequences may be expected.

The temperature is generally high in fatal cases. During the stationary period of fever-heat the lower the maximum, the slighter the case; and, the greater the morning remissions, the more favourable the prognosis. A temperature of $105\cdot5^{\circ}$ to 106° Fahr. is always serious; and a persistent temperature of 105° Fahr. for several consecutive evenings always indicates an anxious case. A temperature above 106° Fahr. is highly dangerous, and 108° Fahr. always fatal. A sudden fall of temperature in the stationary period, from say 106° or 104° Fahr. to the normal or below it, is a fatal sign, indicating cardiac collapse, or intestinal hæmorrhage. It is also a bad sign if the exacerbations begin before midday and only end after midnight. It is a very serious case also if the morning temperature is higher than the evening (FOX); and recovery is rare after a morning temperature of 105° Fahr., or a temperature at any time of 107° Fahr. The approach of death may be looked for if the temperature remain for some time at 106° or 107° Fahr.; if it suddenly rise to $107\cdot6^{\circ}$ or 108° Fahr., or when it suddenly falls very low (as in collapse), to say 94° Fahr.

The pulse has less definite relation to the temperature in enteric fever than in any acute disease; but the pulse of fatal intestinal hæmorrhage is small and rapid. The co-existence of a slow pulse, or one slightly above the normal, with a high fever temperature, is not unusual in cases of

enteric fever (J. W. MILLER). Those also are the most severe cases in which the pulse is quickest; and if in an adult the pulse persistently exceeds 120 the prognosis is usually bad.

When the head-symptoms are severe, and death is threatened by coma, the range of temperature is generally high and irregular; but the more rapid onset of the head-symptoms in such cases does not permit of the thermometer giving the same premonitory indications as in typhus fever (J. T. MACLAGAN). Such a mode of death is apt to occur when cerebral symptoms are marked and prominent from the first, and such cases usually terminate fatally by the end of the second or beginning of the third week.

Death by asthenia may be threatened when the extent of abdominal mischief is great, as indicated by the presence of diarrhoea, hæmorrhage, tenderness over the *caput cæcum*, and small intestines. But the best indications of danger are to be got by a study of the thermometry of the case from the commencement.

At the beginning of the second week, or at the latest during its second half, severe and mild cases diverge so unmistakably that the course at that period is decisive as regards what the future progress will be. A favourable course during the second week permits us to anticipate that the third week will be still milder, with a favourable termination of the disease; while, on the contrary, a severe and unfavourable second week may lead us to expect that the subsequent course of the disease will be unfavourable. In mild cases (analogous to those of modified small-pox), although the evening temperature may reach 103° , and even exceed 104° Fahr., considerable abatements (1° to 2°) take place during the morning, which become more and more obvious toward the end of the second week. Such mild cases progress favourably when the exacerbations do not begin before ten o'clock in the morning, so that before midnight an abatement takes place; when these conditions remain daily the same, or when a diminution of temperature shows itself, although not more than half a degree; and, lastly, when there is an abatement on the eleventh, twelfth, and fourteenth days, so that the evening temperature becomes normal, the only certain proof that the fever has ended.

Among the *favourable* indications during the second week are,—An evening temperature between 104° and 105° Fahr.; a morning temperature one or two degrees lower, late occurrence of exacerbation (not before 10 A.M.); early occurrence of the remission (before midnight); regular and daily moderate decrease of temperature, as compared with that of the preceding day.

Among the *unfavourable* indications are:—Continued elevation of the morning temperature; increase of the evening temperature to 105.5° Fahr., or more; early occurrence of the daily exacerbations; late occurrence of the daily remissions; and very high temperature at any time. Before death the temperature may rise to 108° , or even 108.95° or 110.3° . A retardation of recovery until at least the fourth week is to be anticipated when in the second week the morning temperature is above 103° , and the evening above 104.5° Fahr.; when the exacerbations occur early in the forenoon and remain after midnight; and, lastly, when a fall in temperature about the middle of the week does not take place. A *permanent temperature* of 104° , or even 103° , is an unfavourable sign; so also is an

elevation of the morning above the evening temperatures. A severe form of the disease is to be expected when the morning temperature at the beginning of the second week is above 104° , and when the evening reaches nearly 106° Fahr., or exceeds 105° ; and when towards the end of the week a rise still takes place. The most unfavourable cases are those where, in addition to these unfavourable conditions, oscillations are added, even if these consist in diminution of temperature.

In the third week the patient enters upon those highly characteristic quotidian vacillations of 4° , 6° , and even more degrees, Fahr., between the morning and the evening temperature. In mild cases there are well marked great morning remissions, to the extent of three to three and a half degrees lower than the evening temperature, which may become normal towards the end of the week. From the middle of the week the evening temperature also decreases. If the case is mild, the evening exacerbations gradually decrease in intensity, and the morning temperature is regularly at first from 3° to 4° below the evening. The fever ceases in the course of the week, the temperature reaching its natural standard, and convalescence commences, as a rule, sometimes in the third week, generally in the fourth week, or at the latest in the fifth week. In severe cases, on the other hand, the temperature sinks but little during the third week, or else it maintains the same height or even rises. In the latter case, we may almost positively expect a severe fourth week. In short, the characteristics mentioned as peculiar to the third week already commence in the second. The temperature in the mornings is high (104° Fahr., and more), and differs but little from that in the evening; or even that high temperature increases in the afternoon and evening to a still higher degree. In this it differs from a remission of the fever in a mild case, inasmuch as in remissions the heat in the mornings sinks below the average degree of temperature in typhoid cases—*i.e.*, below 103.3° to 104° Fahr. In severe cases, on the contrary, the temperature always remains above the average, and rises still higher in the evening. Real remissions in such cases are not met with during the whole of the second and third weeks; but when the case is favourable, although severe, the temperature is about a degree lower than in the second week, and the remissions do not take place till the fourth week; and if the temperature remains as high, or rises higher than it was in the second week, the remissions do not occur till the fifth week, and irregularities in the ranges of temperature always render the prognosis doubtful, and generally is evidence of complications.

In cases that recover, a remarkable fatuity remains behind long after recovery; and there appears to be some diminution of intellectual power for some time after convalescence is restored. Sir William Jenner has seen many cases in which childishness of mind remained for more than a month after apparent restoration to health. The patient generally wakes up, as it were, from the fever a complete imbecile. The whole man is changed. He seems to have renewed his youth. Childhood and infancy return, and the greatest care is necessary to prevent untoward events. Enteric fever is perhaps the most treacherous of all diseases, and from what has been written regarding its pathology and morbid anatomy, it may be confidently stated that NO MAN CAN BE CONSIDERED AS FIT FOR WORK, OR

FOR GENERAL MILITARY SERVICE, FOR THREE OR FOUR MONTHS AFTER AN ATTACK OF SEVERE ENTERIC FEVER.

With regard to the symptoms generally of enteric fever, it is of great practical importance to be constantly alive to the fact that no necessary connection exists between the intensity of the general symptoms of the disease and the extent of the intestinal mischief which may supervene, or the absolute danger of the case. Two cases, out of several related by Dr. Bristowe, show that the patients (men) carried on their daily avocations (*typhus ambulatorius*), so mild seemed the disease to be up to the very moment of fatal perforation of the gut. Indeed, the most suddenly fatal cases seem to be the very cases in which strongly marked febrile phenomena do not occur. In a case related by Dr. Murchison, a man twenty-one years of age died on the twenty-fifth day of the fever. Up till the twenty-third day there were no symptoms to indicate danger. He suffered from very slight diarrhoea; the pulse seldom rose above 90, and the patient could get out of and into bed. About forty-two hours before death the pulse rose to 120, associated with sudden pain in the lower and right side of the abdomen. Profound collapse indicated that perforation had ensued, and death soon followed. The *very slightness of the symptoms* ought, therefore, to rouse suspicion, knowing, as we now do, that, associated with the characteristic eruption, the following *four* sets of phenomena may be all that precede a fatal hæmorrhage or peritonitis, namely:—(1.) An elevation of temperature towards evening of only 1° or 2° above 98° Fahr.; (2.) Moderate increase towards evening in the fulness and quickness of the pulse; (3.) A little headache during the first six days; (4.) Scanty urine.

It must also be kept in view, that relapses of all the symptoms, including the eruption, not unfrequently supervene; and we must not be betrayed into the belief that danger is past, if, towards the eighth or tenth day, the little headache that prevailed may pass away, and the other febrile phenomena just mentioned may subside. It is on record that events such as these have led to the belief that convalescence from a mere “febricula” had been established, leading to the discharge of the unfortunate patient from hospital. His vocation, if a soldier, would then compel him to undertake severe duties during the actual height of a severe disease, made more dangerous and perhaps fatal by such a mistake.

Another symptom, often very painful, is *meteorism*, or the accumulation of air in the large intestine. This is present in a greater or less degree in one-half of the cases, and when considerable it always marks a grave affection, and one generally fatal. On the contrary, the abdominal muscles are, in a few cases, tense and strongly contracted. It is, however, the experience of all physicians that there is no condition of enteric fever so low, and no symptoms so severe, from which the patient may not recover; and, on the other hand, there is no case of this form of fever so slight that it is to be considered free from danger. The prognosis must therefore be cautious, because perforation of the intestine may follow the mildest case, and death from peritonitis ensue.

Generally speaking, the prognosis in enteric fever must always be a very guarded one; and there is always a risk in giving a favourable one, for reasons already stated, and also on account of the variety

of circumstances under which death may ensue in cases of typhoid fever, namely:—

(1.) By poisoning of the blood generally, as indicated by many symptoms which enteric fever has in common with *typhus fever*, *cholera*, *small-pox*, *dysentery*, *scarlet fever*, *diphtheria*, *ichorrhæmia*. The intensity of the fever (measured by the thermometer) is generally great in those cases, and the fatal event occurs either at a very early period of the fever, associated with cerebral congestion, or it may occur later, when it may be supposed that the danger is past. This is sometimes termed the secondary poisoning of the blood (septicæmia), and is most likely due to the ulcerated intestines, with the bowels perhaps on the verge of perforation. The pulse becomes rapid and small; cold, clammy sweats appear; and the body begins, even in life, to exhale a putrid odour. In cases where the blood is so gravely implicated, gas has been observed to become developed during life, and has been detected in the veins at the root of the neck for some minutes before death (CLOSS, FRANK, and JEFFREY MARSTON in *Med. Times*, Feb. 7, 1857).

(2.) By implication of excretory organs at an early period; for example, the kidney, as denoted by persistent albuminuria, or by bloody urine—conditions which tend to aggravate the blood poisoning. (3.) By congestions of important organs; for example, the lungs and the brain, in consequence of poisoned blood, or its high temperature; which congestions are still further brought about by the circulation, in the blood-vessels, of putrid juices, or of the fibrinous debris of clots in a granular condition, having formed as plugs in the varicose veins surrounding the sloughs and ulcers of the intestines. (4.) By hæmorrhage from the bowels during the separation of the gland-sloughs. (5.) By exhaustion from profuse diarrhœa in cases where the catarrh of the mucous membrane has been excessive. (6.) By peritonitis, with or without perforation of the intestines. There are two periods in the course of the fever when perforation is apt to take place. The first period is during the separation of the sloughs, about the end of the second and throughout the third week. The second period is during the protracted convalescence, with atrophy of the intestine already described, and when the ulcers are in a weak atonic state, the result of intense protracted fever and profuse catarrh. (7.) By peritonitis subsequent on suppuration of the large mesenteric glands, and rupture of their inclosing capsule (JENNER); or from the bursting of softened new growth from the spleen into the peritoneum (ROBERTSON, JENNER); or from ulceration of the gall-bladder, or pericœcal abscess. (8.) By a combination of several of these conditions.

On the other hand, with regard to prognosis, it must also be remembered that recovery is by far the most frequent termination of enteric fever—by healing of the lesion, as described at p. 590. It takes place in three-fourths, at least, of all the cases (NIEMEYER). Some epidemics, however, as is always the case with epidemics, are attended with a greater mortality than others; and the cases are of a more malignant type, one form of lesion prevailing rather than any other. In some cases recovery is incomplete, or sequelæ remain, such as *neuralgia*, *anæsthesia*, *mental imbecility*, *partial paralysis*, *marasmus*, and *anæmia*. The tendency to the development of tubercle in the lungs in scrofulous constitutions has been already mentioned, p. 596, *ante*.

Treatment and Management of Enteric Fever.—The management of a case of enteric fever resolves itself therefore into two lines of practice, embracing,—(1.) *Preventive measures.* (2.) *Therapeutic Treatment.*

(1.) *Preventive Measures, or Measures for Checking the Spread of Typhoid Fever.*—Whatever may be the view theoretically adopted regarding either the origin or the propagation of typhoid fever, it is satisfactory to know that, practically, medical officers can employ preventive measures which (to use the words of the late Dr. Lankester) “will cover the issues of both theories.” If these are universally carried into effect, it is not too much to expect that this fever might perhaps soon become extinct. At all events, with the facts before us, it is unwarrantable to permit the great bulk of what escapes from the diseased intestine of typhoid fever patients to be let loose upon society, into the cesspool or sewer, or on the dung-heaps, in full possession of all their deadly power, without being first destroyed in the way to be presently recommended. “The grand fact is clear,” writes Dr. Parkes, “that the occurrence of typhoid fever points unequivocally to defective removal of excreta, and that it is a disease altogether and easily preventible.” Typhoid fever ought therefore soon to disappear from every return of disease, whether in military or in civil life. The measures about to be specified have been made public mainly through the writings of Dr. William Budd; and, *provided they are thoroughly and efficiently carried out*, it is believed that the recurrence of typhoid fever may be entirely prevented. To enable us to judge of the extent of the infection to be destroyed, there are two elements to be taken into account,—*First*, The amount and duration of the intestinal discharge in each case; and, *Second*, The number of cases actually occurring. With regard to the first, Louis has found that the average duration of the alvine flux in cases of typhoid fever is *fifteen* days in mild cases and *twenty-six* days in severe cases. With regard to the second point—namely, the number of cases occurring—the Reports of the Registrar-General show that at least 100,000 to 150,000 cases of typhoid fever occur annually in England alone. In other words “*every year in England more than 100,000 human intestines, diseased in the way already described, continue each, for the space of a fortnight or thereabouts, to discharge upon the ground floods of liquids charged with matters on which the specific poison of a communicable disease has set its most specific mark*” (BUDD).

The measures recommended for preventing the spread of this fever are founded on the power of chemical agents to destroy absolutely the material which contains or carries the specific virus of such communicable diseases. Assuming it, therefore, to be certain that the intestinal discharges in typhoid fever are the media of propagation of the disease, it is no less certain that, by SUBJECTING THE DISCHARGES ON THEIR ISSUE FROM THE BODY TO THE ACTION OF POWERFUL DECOMPOSING CHEMICAL AGENTS, THEY MAY BE ENTIRELY DESTROYED OR DEPRIVED OF THEIR SPECIFIC VIRUS. The following details of procedure are suggested:—(a.) All discharges from the fever patient should be received on their issue from the body into vessels containing a concentrated solution of chloride of zinc. (b.) Two ounces of a caustic solution of chloride of zinc should be put in the night-stool on each occasion before it is used by the fever patient. (c.) All tainted bed or body linen should, immediately on its

removal, be placed in water strongly impregnated with the same agent. (d.) The water-closet should be flooded several times a day with a strong solution of chloride of zinc, or of sulphate of iron or carbolic acid; and some chloride of lime should be also placed there, to serve as a source of chlorine in the gaseous form. (e.) So long as fever lasts, the water-closet should be used exclusively as a receptacle for the discharges from the sick after being thus disinfected. (f.) Pieces of rag should be used for wiping the remains of all discharges from the patient's body, and burned immediately after use. The Privy Council have now made the principle of this method an integral part of their "General Memorandum on proceedings advisable to be taken in places attacked or threatened by epidemic disease," and which is given *in extenso* at p. 402 of this volume.

(2.) *Therapeutic Treatment of Enteric or Typhoid Fever* aims at reducing temperature and subduing vascular excitement, if these be in excess; restraining and moderating, but not actually suppressing or altogether checking the diarrhoea; stimulating and restoring the nervous system; obtaining a free excretion from the kidneys; and bringing about a gradual elimination of the morbid growth from the intestinal glands with as little ulceration as possible. To accomplish the first of these indications the use of *digitalis* has been especially recommended by Wunderlich. He considers that it decidedly mitigates the febrile symptoms which are present in severe cases at the time when the ulcers begin to heal, and which often impede or prevent recovery. He advocates its use in the severe forms of the fever only, especially at a time when most danger is to be apprehended from the violence of the fever in the second week, when the evening temperature is at its highest (105° to 108° Fahr.), and when the remissions in the morning are slight; when the pulse is frequent, 110 to 120 or more. In mild cases it is superfluous. He finds that in the form of *infusion* it is easily absorbed by the intestines of patients suffering from fever; and, if given in a suitable dose, has most marked effects in subduing the rate of pulsation, and in reducing animal heat. Large doses of the infusion should be given without interruption until the full effect has been obtained,—

An infusion of fifteen or twenty grains of digitalis in eight or ten ounces of boiling distilled water may be consumed in twenty-four hours by adult patients.

It acts more rapidly on animal temperature than on the heart. For the first few days after its use the decrease of temperature is rather slight, but may afterwards become considerable; and after the temperature has been much diminished and again rises, it never attains its former excessive height. The full effects of the medicine are known to be brought about if the temperature is reduced 2° or 3° Fahr. in the evening; and the action of the remedy does not continue beyond one day after its use has been discontinued. The diminution of the pulse is slight at first, and occurs in some cases on the second day after the remedy, but mostly on the third day, or even later; and on the fourth or fifth day after the medicine has been commenced, the rate of pulsation may be diminished by thirty to forty or fifty beats within from twelve to thirty-six hours. The pulse

may continue to fall even below its normal velocity, and this reduction of pulsation may last for several weeks in succession. If the velocity of the pulse should decrease rapidly, the use of digitalis must be discontinued at once (*Arch. der Heilk.*, 1862, p. 116). Cold and tepid sponging, or the cold affusion, are remedies deserving of further trial for reducing the pulse and temperature. The oppressive headache may be greatly relieved by cold to the head; and in all severe cases the hair should be shaved off.

To restrain excessive diarrhœa, *lime-water mixed with milk* in equal parts and taken as a drink, is found to be beneficial, agreeable, refreshing, and nourishing. It is clear that the diarrhœa *ought not* to be altogether checked. Professor Gairdner, of Glasgow, is in favour of the French practice of giving saline laxatives rather than astringents; also of the diligent use of enemata to unload the bowels from below, where anything like abdominal distension has occurred. These enemata may be simply of warm water, to which a little aniseed is added; or the asafœtida enema may be given. Of the propriety of restraining *excessive* diarrhœa there can be no question. Dr. Huss, of Stockholm, is of opinion that the diarrhœa during the first stage ought not to be arrested, but abated and mitigated—*only if excessive*. If it is suddenly arrested, meteorism is produced; and pains in the intestines or vomiting may supervene, with cerebral symptoms. The febrile phenomena are then increased. The diarrhœa is too copious or excessive if the evacuations exceed four or five a day, being of considerable quantity, and fluid. Such evacuations weaken the patient rapidly, and should be mitigated by mucilaginous drinks, such as *rice-water*, *infusion of linseed*, decoction of *althœa officinalis* or *ipeacacuanha* in small and repeated doses. This latter remedy retards the peristaltic action of the intestines, and lessens the secretion from the mucous membrane. The dose must be regulated so as to avoid vomiting; and the feeling of nausea which is apt to follow the first dose soon disappears with continued use.

On the other hand, the highest authorities write,—“Restrain diarrhœa and hæmorrhage in typhoid fever, and when you have fairly locked up the bowels, keep them so. Patients will go for four or six days, or even longer, without suffering inconvenience from this state of constipation” (MURCHISON, TODD). The benefits to be derived from the mineral acids—*hydrochloric* and *sulphuric* especially—are highly spoken of. From fifteen to thirty minims of the dilute acids may be given every three or four hours; and with each dose half a grain of *quinine*, as in the following prescription for an adult:—

R. Acid. Sulph. dil., vel Acid. Hydrochlor. dil., ℞xx. ad. xxx.
 Quinæ Sulph., gr. $\frac{1}{4}$ ad gr. i.
 Syrup Aurantii, ʒss.
 Aquæ Carui, ad. ʒi.
 Fiat haustus, 3â vel 4â horâ sumendus.

If there be more than two motions in the twenty-four hours, with marked prostration, astringents should be had recourse to (MURCHISON). A starch enema, containing from ten to twenty drops of laudanum, should be administered towards evening, and recourse may also be had to the following draught:—

R. Acid. Sulph. Aromat., ℥xxx.

Liq. Opii. Sedativ. (Battley), ℥iii.

Aq. Menth. Pip., ʒi.; *misce*.

Fiat haustus, 4tâ vel 6tâ quaque horâ sumendus.

If the mineral acids are not tolerated by the stomach, *acetate of lead* is worthy of trial, in doses of two or three grains in solution every four or six hours, with or without an eighth of a grain of *morphia* (MURCHISON). *Alum* dissolved in gum, to the amount of twenty-four grains in a day, which may be increased to one drachm, is best given in the form of *alum whey*, prepared by adding one drachm of *alum* to a pint of boiling milk, and then straining. Two ounces may be given after each motion of the bowels (FOUQUIER, MURCHISON). A seidlitz powder may sometimes check excessive diarrhœa, by altering the secretions, and is especially beneficial if there be much meteorism (TROUSSEAU, MURCHISON).

The question, therefore, of checking excessive, or encouraging moderate diarrhœa is one which requires the careful study of each particular case; and my experience in the treatment of typhoid fever leads me to recommend rather the restraining of excessive diarrhœa than either encouraging the action of the bowels or locking them up for days. The French practice of giving saline laxatives is not a safe one; and if the action of the bowels is deemed desirable, enemata will be found sufficient. *Nitrate of silver*, in doses of one to three grains, made into a pill, and taken every six or eight hours; or sulphate of copper, in doses of a quarter of a grain similarly given, are remedies most useful in the diarrhœa connected with atonic ulcers, after the fourth week of the disease (BELL, MURCHISON). If meteorism or tympanites prevails from the accumulation of air in the colon, it may sometimes be relieved by the passage of a long flexible tube by the anus, as far up the colon as it can be made to go easily. With regard to stimulation it is not decided how far alcohol is beneficial. The fever eventually is attended with much exhaustion, and is often protracted, yet typhoid fever does not seem to bear stimulation so well as typhus. The tendency to prostration is the only indication for its use; but stimulation must not be persevered in if the pulse is quickened by its use, the hectic flush made more manifest, the tongue made drier, or if delirium supervene. Food and sustenance are the real preventives of delirium, and the best stimulants when necessary. With regard to the maintenance of free action from the kidneys, it is to be observed that so long as the *excretion* of urea and uric acid is abundant, no diuretics are necessary; but whenever the amount of the solids falls greatly (which can be known at once with sufficient accuracy by a comparison of the urine passed in twenty-four hours with the specific gravity), means must be taken at once to increase, if possible, the urinary elimination. The *warm bath*, with repeated small doses of the *alkaline carbonates*, or of the *nitrate* or *bitartrate* of *potash*, will often effect an increased elimination, (PARKES, l. c.)

To accomplish the last indication—namely, to influence the elimination from the intestinal glands, by direct local action on the intestinal mem-

brane—Wunderlich and Parkes especially advocate the use of *calomel*, if it can be given before the ninth or tenth day. The late Dr. Anthony Todd Thomson used to give it; and, from the observation of many cases under the care of this physician, as well as from his own experience, Dr. Parkes considers that *calomel* is a medicine to be strongly recommended in typhoid fever. But it must not be given later than the tenth or eleventh day, and at no time in large doses. In the great majority of cases where it can be given during the first week and before the occurrence of much diarrhœa, the course of the disease is rendered milder and shorter. Such is now the recorded experience of Wunderlich, Niemeyer, Parkes, and Wood. *One or two grains twice a day* is enough, although Wunderlich gives *one to five grains twice daily*; but *five grains* is considered by Dr. Parkes to be too large a dose. Dr. Wood of Philadelphia bears testimony to the benefit to be derived from mercury about the seventh or ninth day of the fever. He believes “it tends in some degree to arrest the progress of the disease in the glands of Peyer, and to promote resolution of the inflamed patches. He prefers minute doses of the *blue pill mass*—a grain every two hours—till the mouth is slightly affected, associated with small doses of *ipêcacuanha*, when the stomach is not irritable. The beneficial effect of this combination is shown by the tongue becoming moist, the skin relaxed, and the symptoms generally being ameliorated. Twelve grains of *blue pill mass* are to be combined with two grains of *ipêcacuanha powder*, and with two grains of *opium powder*. The whole is to be divided into twelve pills, one of which may be taken every hour, or every hour and a half, or every two hours (WOOD, *Practice of Medicine*, Vol. I., p. 345, 4th edition). *Calomel* is, however, contra-indicated if the diarrhœa is excessive, or if there should be excessive pains in the bowels, with early and violent meteorism. It is also not proper to be given if the condition of the patient is anæmic, or if there is a decided hæmorrhagic diathesis. It is most useful as a restorative of the intestinal functions in cases where the tongue is dry and coated, where thirst is absent, and when the urine is cloudy and of low specific gravity. If the first dose is vomited, the administration must be repeated. *Calomel* has no direct effect on the pulsation or respiration, nor on the cerebral functions; but its beneficial influence is very decidedly appreciable by the modifications of temperature which it induces, and which have been already noticed at page 606.

No general rules can be laid down to guide the treatment of the intercurrent phenomena or accidents of the disease. Combinations of remedies must be adapted to correct the several functions which may be simultaneously deranged, so that treatment must be varied according to the functions mainly implicated or suspended, and to the degree of their affection. Abdominal pains and meteorism may be relieved by mustard poultices, or turpentine *stupes* may be applied, followed by simple hot water fomentations. After these remedies have been used, water compresses over the abdomen tend to lessen the tension and the gurgling in the intestines, and to diminish the tenderness on pressure. They counteract the inclination to meteorism, and lessen excessive diarrhœa. Dr. Huss believes also that the ulcerations in the ileum are prevented from spreading, and that perforations of the intestine have been of much

rarer occurrence since he commenced to use these compresses. The compress, after being soaked in warm, but not hot water, is well wrung, and applied so as to cover the whole abdomen; and it must be changed two or three times a day, according to its tendency to dry. The compress is composed of four to eight double folds of coarse linen, and is to be laid over the whole abdomen, and afterwards overlaid with a cover of oiled skin or india-rubber stuff, to prevent too rapid evaporation. The compress should fit as closely as possible, and care must be taken that it be not displaced, otherwise air enters between the skin and the compress, so that cold, instead of a moist heat, is produced. These compresses may remain untouched as long as they are moist and warm; and they may be employed on the chest as well as on the abdomen should capillary catarrh or pneumonia supervene.

Hæmorrhage from the bowels, partly fluid and partly in clots, evacuated with the stools, is a symptom of grave import; and one urgent aim of treatment is to arrest the bleeding. Huss found the *sugar of lead* most serviceable, administered by the mouth, and also by clysters. By the mouth, two grains may be given every half-hour, and even every quarter of an hour; and it is best given dissolved. Twenty-four grains of *crystallised acetate of lead*, being dissolved in one drachm of *dilute acetic acid*, to which six ounces of *distilled water* is added,—a table-spoonful of this solution may be given every half-hour. At the same time a clyster may be administered, composed as follows:—Ten to fifteen grains of *acetate of lead* are to be dissolved in four ounces of *distilled water* (warm), to which twenty or thirty drops of tincture of opium may be added; and the administration of such a clyster may be repeated in four or six hours if required. Pieces of ice may also be swallowed now and then; and even crushed ice may be applied, inclosed in a bladder, over the abdomen. If bleeding from the nose is not arrested by the use of vinegar and cold water injected up the nostrils, nor by the use of cold water compresses applied to the nose, plugging by the posterior nares must be had recourse to. *Turpentine* is a most valuable remedy in hæmorrhages, and in the stage of ulceration. It was originally recommended by Dr. Graves, and is highly spoken of by Drs. Huss, Wood, and Murchison. It may be given in all cases where the tongue is dry, and when “instead of cleaning gradually from the edges and tip, it often parts with its fur quickly, and in large flakes—generally, first, from the middle or back part of the surface, which is smooth and glossy, as if deprived of its papillæ. There is also generally an increase of the tympanites, and the ulceration of the ileum seems to be attended with great dryness of the tongue.” Under these circumstances Dr. Wood gives the *oil of turpentine* in doses of five to twenty drops every hour, or every two hours. It is best administered in an emulsion with gum-arabic, loaf sugar, water (WOOD); or in an emulsion with the yolk of an egg and honey or mucilage (HUSS). Amelioration of the symptoms may be observed in twenty-four or forty-eight hours—the tongue becoming more moist, and covered with a white fur—distension of the abdomen ceases to progress, and after a time diminishes. The use of the oil should be continued under these circumstances; but the dose should be gradually diminished.

Tonics and stimulants may be absolutely essential on account of debility

attending the advanced stage of the disease, generally about the third week. When the pulse is slow and feeble, the skin cool, the tongue and teeth encrusted with dark sordes, at an advanced period of the fever, then stimulants are obviously necessary. But even when the pulse is feeble, but yet frequent, and the skin hot, stimulants are even then known to be of service. It is necessary to administer them in every case with great caution, and to watch the effects constantly and closely. If their use is found to augment the heat of the skin, or to increase the frequency of the pulse, or to aggravate the delirium or stupor, it is then necessary to suspend their use. They are known to be doing good service, however, if they lessen the frequency of the pulse, and increase its fulness and strength; if the skin becomes cool and moist; if the delirium is subdued or moderated; and especially if refreshing sleep be procured. Dr. Wood recommends the use of wine whey, prepared by adding *one quart of good sherry wine to two quarts of boiling milk*, and straining after coagulation. Of this a table-spoonful or more may be given every hour or every two hours. If the strength is greatly reduced, it may be necessary to give pure wine or brandy; or even *sulphuric* or *chloric* ether in cases of great prostration. *Opium* is also a useful stimulant. It may be given when the pulse is not full nor strong, and when cerebral symptoms do not exist. In the later stages it may be given in doses of half a grain, or a grain, every four, six, or eight hours. It is known to be acting beneficially when it promotes sleep, subdues nervous excitement, and induces gentle perspiration. *Carbonate of ammonia* is objectionable, as it may irritate the bowels and increase the diarrhœa.

The diet is of the utmost importance to be attended to in cases of typhoid fever from the very commencement of the disease. It ought then to be both food and drink combined, in the form of a light nutritious liquid, such as meat fluid, soup, eggs, and milk. Barley-water, rice-water, toast and water, thickened more or less with solutions of tapioca, sago, arrow-root, the *juice* of sweet fruits, or the *very soft pulp* of fruits, or the *pure jelly* of ripe fruits; but fruit in its crude state is to be strictly withheld. It is necessary, as a rule, to give food at certain intervals and in certain quantities. A wine-glassful should be given at least every two or three hours, according to the state of digestion and the demands upon the strength of the patient. It may be that the patient is unable to swallow, from the dry and shrivelled state of his tongue. Before offering him food or drink, therefore, the nurse should put a teaspoonful of lemon juice and water into his mouth. She must then wait a minute or so, until the fur upon the tongue and mouth is softened and moist, after which the patient will often drink or take his food with ease. The patient ought to have his mouth regularly washed out with a linen rag, saturated with water, in which a little soda has been dissolved. Milk in small quantities frequently repeated will be found an excellent diet; and animal broths and jellies may ultimately be given. The *extractum carnis*, as prepared by Liebig or Valentine, is a most valuable nutriment for typhoid fever patients. The best drink is pure spring water, or soda-water, or barley-water, or water that has been mixed with oatmeal, but is free from any particles of oats. The patient generally desires and requires to drink freely, in order to replace the loss of fluid by perspiration and diarrhœa;

and, in the advanced stages of the fever, plenty of drink should be given by the nurse.

The utmost caution is necessary as to diet and aperients during convalescence; *first*, as to opening the bowels, castor-oil or simple enemata are the only means which should be resorted to; *secondly*, as to diet, no flesh meat should be allowed till at least seven days after all the febrile phenomena have passed away, and the food should be as free as possible of excrementitious matter. Malt liquors should not be taken *before* food.

RELAPSING FEVER.

LATIN EQ., *Febris Recidiva*; FRENCH EQ., *Fièvre à Rechutes*; GERMAN EQ., *Typhus Recurrens*; ITALIAN EQ., *Tifo Recidivo*.

Definition.—*A continued fever, having a very abrupt invasion and short duration, characterised by an abrupt relapse occurring after an interval of apparent health of about a week. There is no eruption on the skin, nor any specific lesion associated with this fever, which is marked by rigors, chilliness, severe headache, vomiting, and often jaundice; a white moist tongue, epigastric tenderness, confined bowels, enlarged liver and spleen, high-coloured urine, a frequent, full, and often bounding pulse, pains in the back and limbs, restlessness, and occasionally delirium. These symptoms abruptly terminate by an exceedingly copious perspiration between the fifth and the eighth day; and after a complete apyretic interval (during which the patient may be so well as to get up and walk about), an abrupt relapse supervenes on the FOURTEENTH day, counting from the first commencement of the fever. The relapse runs a similar course to that of the primary paroxysm, and terminates between the third and the eighth day. In some cases a second, third, fourth, and even fifth relapse may occur. Death is apt to happen from sudden syncope, especially after the excessive perspiration; or from suppression of urine and coma.*

Pathology.—(a.) *History.*—The name by which this disease is known is derived from one of its most constant and striking peculiarities. It has been also described under the various names of “five” or “seven-day fever,” “seventeen-day fever,” “bilious remittent fever,” and “bilious relapsing fever,” “mild yellow fever,” “synocha,” “short fever,” and “short relapsing fever.” Epidemics of it have prevailed on different occasions since 1739. In Dublin it was epidemic at that time and in several subsequent years. Sometimes it has been described as a variety of a well-known form of fever, and at other times as a new disease. In Scotland in 1817-18 it was clinically recognised and described by Drs. Christison and Welsh; and when it appeared as an epidemic in Edinburgh and Leith in 1843, it was again easily recognised. At this time it also appeared in Glasgow about a month before its outbreak in Edinburgh; and subsequently became prevalent in Dundee and other large towns in Scotland. It was observed with great accuracy, and its phenomena are recorded in the medical journals of the period, by Drs. Craigie, Alison, Arrott, Henderson, Douglas, Jackson, Mackenzie, Cormack, and Wardell. It formed a part of the fever epidemic of Ireland in 1817-18-19, described by Barker and Cheyne. It had been prevalent in Ireland for many years. Epidemics of

it were described by Ratty, in his *Chronological History of the Diseases of Dublin*, as early as 1739 and 1741. In most of the periods of epidemic fever referred to, the commencement was characterised by the greater preponderance of cases of relapsing fever; and as the epidemic advanced, the number of cases of relapsing fever gave place to a preponderance of typhus cases (STEELE, R. PATERSON, ORMEROD, MURCHISON). In 1847, relapsing fever became again epidemic in Glasgow, Edinburgh, and the large manufacturing towns of Scotland, as well as in London, when it was carefully described by Sir William Jenner, who, moreover, shows that its characters have remained constant since they were first described by British physicians. During the same year it prevailed in some parts of the Continent, more especially in the Prussian province of Upper Silesia, and in some other parts of Germany. There it has been described by Virchow, Bärensprung, Dümmler, and Suchanek. These observers, however, did not know or recognise the fever, so well and precisely described by the Scotch physicians; and, indeed, Dr. Parkes was the first to indicate, in his admirable paper on "The Diagnosis of Fevers," that the epidemics these German physicians described were mainly made up of relapsing fever. This fever evidently formed the great bulk of the cases. Yet, although its characters are thus so striking that the most superficial observer could not fail to recognise them, the German systematic writers (except Virchow) make no allusion to relapsing fever as a separate and distinct disease; and even those who observed the fever in Germany failed to draw that obvious inference to which the Scotch physicians unanimously came—namely, that *relapsing fever* is a disease altogether distinct from *typhus* and from *typhoid fever*. If it is not so, "we know not that any medical evidence whatever can be relied upon" (PARKES). In the summer of 1855 it prevailed, after the hardships and privations of the preceding winter, among the British troops in the Crimea, where it was recognised and described by Dr. Lyons. It has not been observed in France, nor in any other part of the Continent of Europe. The observations of Dubois, Austin Flint, and others, leave no doubt that relapsing fever was seen in New York, Buffalo, and other parts of North America, in 1847 and 1848. But all the cases are traceable to Irish immigrants, and there is no good ground for believing that the disease is indigenous in America (MURCHISON). In India, its occurrence has been recently noticed by Dr. Vandyke Carter in the Bombay Presidency during the greater part of 1877, in connection with the prevalence of famine; but no sufficient grounds exist to warrant any distinct causal connection between the famine and the fever (SURGEON-GENERAL HUNTER, T. R. LEWIS). Since the epidemic of 1847 and 1848, relapsing fever has been gradually disappearing; and for the seven or eight years previous to 1863 not one case had been observed in the hospitals of Edinburgh, Glasgow, or London (MURCHISON). Professor W. T. Gairdner has not seen or heard of a single case at Edinburgh since 1855; and, according to Drs. Lyons and M'Ewen, true relapsing fever has of late years been a rare disease in Ireland. In 1869 and 1870 it again became epidemic in London, Edinburgh, and Glasgow.

(b.) *Causation*—Like other continued fevers, its specific cause is unknown; but it selects its victims from the poor and ill-fed, who live miserably,

in crowded, filthy, ill-ventilated apartments, rather than from the wealthy and well-fed, who live in comfort and in well-aired abodes. Its poison appears to be of a specific kind, and its phenomena are very different from those of *typhus* and *enteric fever*. Patients recovering from either *typhus* or *enteric fever* may catch, by contagion, *relapsing fever*, while patients convalescent from *relapsing fever* may also take either of the forms of *continued fever* already described. It has been supposed by some (Dr. CORMACK) to be identical with the malarious form of *yellow fever*; but there is not sufficient evidence to establish the point. It seems more nearly to approach in its nature some forms of remittent fever, on account of the repetition of the rigors, often at regular daily periods, for two or three days. The marked periodicity of its relapses, which "come on like a fit of ague almost to an hour" (Dr. R. PATERSON), and the enlargement of the spleen to a greater extent than in any other form of fever (JENNER), point also to a malarious origin. On the other hand, epidemics of relapsing fever appear to commence, progress, and decline quite irrespectively of the season of the year (MURCHISON). There are causes, circumstances, or conditions which obviously favour the accession of relapsing fever; and no doubt, also, its occurrence in an epidemic form. Chief amongst these predisposing causes must be placed destitution and want of food, while the names applied to the disease by different countries indicate the popular belief as to such predisposing causes being credited with originating the disease in the first instance. Thus it is spoken of as the *famine fever* of the British Isles, and the *hunger pest* of Germany.

(c) *Morbid Anatomy*.—No special anatomical lesion has been pointed out as peculiar to *relapsing fever*. The most constant lesion is enlargement of the spleen, the size attained by that organ being on the whole larger than in either typhus or typhoid fevers. Sir William Jenner has recorded the weight in one case to have been as much as thirty-eight ounces, and of a size in proportion. Its substance is generally softened, sometimes diffuent. It is usually seen at its largest size when death occurs during the final paroxysm; but if death occurs during convalescence, the spleen is of a normal size. Occasionally pale, or red fibrinous infarctions are found in its substance and near its surface. They are easily broken down, have a fine granular fracture, and are considerably firmer than the surrounding tissue, from which they are separated by a distinct line of demarcation. As a rule, there is but little congestion of the lungs, the weights of which contrast singularly with the weights of organs in subjects dead of typhus fever. The blood in a few cases has been found fluid throughout the body after death; but generally, when drawn from the body during the febrile paroxysm, it is buffed; and decolorised coagula are found in the heart and large vessels after death more frequently than in cases of typhus. In several cases urea has been detected in the blood in considerable quantity. The proportion of white corpuscles is increased—a fact of interest in connection with enlargement of the spleen, and the state of anæmia so commonly observed (CORMACK, ALLEN THOMSON, MURCHISON). In the epidemic of relapsing fever which prevailed in Berlin in 1867-68, the late Dr. Obermeier commenced to examine the blood; but without any definite result till 1872, when the fever again appeared in Berlin. He then discovered organisms in the blood associated

with the morbid process so closely, that as soon as the pyrexia disappeared they disappeared with it, reappearing when the patient again fevered in the relapse. The observations of Obermeier have been since confirmed by Engel and Weigert in Berlin and Breslau, by Macnamara and Lewis in India. The organisms were subsequently identified by Cohn and named *Spirillum* (*Spirochaeta*) *Obermeieri* (Fig. 76 E, page 372, *ante*), but which does not seem to differ from other *spirilla* known to be harmless. It does not differ in size or in character of movements from *Spirillum* (*Spirochaeta*) *plicatilis*, found by Eulenberg in water many years ago, and which Cohn himself found in water and also in the mouth—in the mucus surrounding the teeth. The weight of evidence seems in favour of the belief that the *spirillum* found in relapsing fever is non-essential and merely an epiphenomenon; for not only is it absent from the blood in certain cases of the fever examined, but spirilla precisely similar have been found in an abscess opening into the mouth of a fever-free patient, and also in connection with caries of bone (MANASSEIN, BILROTH, HEYDENRICH, T. R. LEWIS). The liver is generally large, and the gall-bladder filled with dark thick bile.

Symptoms.—(a.) *The Primary Paroxysm.*—The period of incubation is not fixed; and is more variable than that of typhus. It is on the whole shorter than that of typhus; and occasionally, as in typhus, there is scarcely any latent period at all, the symptoms commencing almost immediately after the first exposure to the poison. Five, nine, twelve, and sixteen days are periods of incubation which have been observed. (See papers by MUIRHEAD, in the *Ed. Med. Journal*, for 1870). The seizure is generally, indeed almost always, sudden. Sometimes, on waking in the morning, or when employed in business, severe rigors at once come on, with a sense of chilliness and frontal headache. These phenomena are more severe than their expression is in the commencement of typhus. There is slight prostration of strength from the first, but rarely so severe as in typhus. If premonitory symptoms exist, they usually manifest themselves by pains in the limbs, and lassitude, nausea, and perhaps vomiting, with feeling of prostration. Subsequently, and very soon, febrile reaction sets in, sometimes violent, expressed by intense heat of skin, severe headache, throbbing temples, intolerance of light and sound, suffusion of face, sleeplessness, remarkable anxiety of countenance and jactitation, with a very rapid pulse—so rapid as to range from 110, sometimes as high as 140 beats in a minute; the tongue is coated with a white fur, and in a great majority of cases, in some epidemics, there is uncontrollable vomiting of greenish, bitter fluid, with epigastric tenderness, and great thirst. The pains in the muscles and joints are sometimes so severe as to resemble rheumatism; and when the pain in the back is severe, together with the rigors, the vomiting, and the headache, it may not be possible in the first instance to say that the attack may not prove to be one of small-pox. But the pain in the back is not generally so severe, nor is the vomiting so incessant in cases of *relapsing fever* as in cases of *small-pox*. The headache is to be distinguished from what is commonly called a “sick headache,” or “bilious headache,” by the circumstance that the “bilious headache” is in most cases *occipital*; and the heat of skin, combined with the quick pulse, serve to distinguish an attack of relapsing

fever at its outset from one of "bilious headache." From idiopathic head affections the accession of relapsing fever is distinguished by the suddenness of the attack, the rigors, the hot skin, the pain in the joints and limbs, and the white tongue (JENNER). The symptoms generally of *relapsing fever* are so severe that the patient takes to bed at once. He does not feel weak, but he feels so giddy that he is unable to remain out of bed, or off the horizontal position. In some cases there is pleurodynia in a severe degree, but without any stethoscopic indications of pleural inflammation. By the second or third day the pulse almost invariably exceeds 100; as a rule it reaches 120; in not a few cases it is as high as 140 or 160; and it is not rarely 140 on the second day of the disease, being at the same time full and of considerable firmness—symptoms not indicative of commensurate danger—with anxious and oppressed breathing. There may be also sweating, profuse and lasting for several hours, but without relief to the headache and other symptoms. Almost no sleep is obtained, and the little obtained is dreamy and unrefreshing. The skin continues dry after the sweating ceases; or after the primary rigors, if sweating has not taken place.

The heat of skin feels ardent—as much as 102° to 107° Fahr.; and these febrile phenomena are occasionally varied by short rigors or slight sweating. The thermometric phenomena differ from all other fevers. There is a rapid but not a sudden ascent for four or five days, and a sudden critical defervescence. Then the temperature remains normal for an interval extending over a variable number of days, after which there is a repetition, more or less intense, of the fever, followed by a similar subsidence. The temperature will rise to 106° or 108° Fahr., and the highest point will generally precede the critical defervescence. In many cases the rise of temperature will be so continuous as not only to continue ascending from morning to evening, but from evening to morning. The highest point of each day is generally from four to seven o'clock P.M. Delirium does not generally supervene on the first attack, although, by the *fifth* or *sixth* day, just before the crisis, it has been in some cases of a violent kind. In a large proportion of cases there is decided jaundice, and in others the skin exhibits a bronzed hue. The jaundice is not attributable to any obstruction of the *ductus communis choledochus*, as bile passes freely, and even copiously with the stools, and as after death the gall-duct is pervious. There is generally tenderness over the region of the liver in such cases; and it may be enlarged. Thirst is excessive; the appetite absent or voracious, and the bowels constipated. The tongue, at first moist, is covered with a white or yellow fur, which it may retain throughout the illness; and in many cases it may become dry all over, or with a brown dry streak down the centre, after the third or fourth day.

(b.) *The Crisis*.—After the patient has continued in this state for a period varying from five to eight days, a sudden change takes place, immediately preceded, in most cases, by an exacerbation of all the symptoms. "When every symptom appears hourly becoming graver—when the restlessness and general distress have reached their highest point—then ensues a most remarkable series of phenomena, followed by as remarkable an intermission of all the symptoms, and an apparent restoration to health." This period has received the name of "Crisis," and supervenes

DIAGRAM OF TEMPERATURE IN A CASE OF RELAPSING FEVER (Herman).

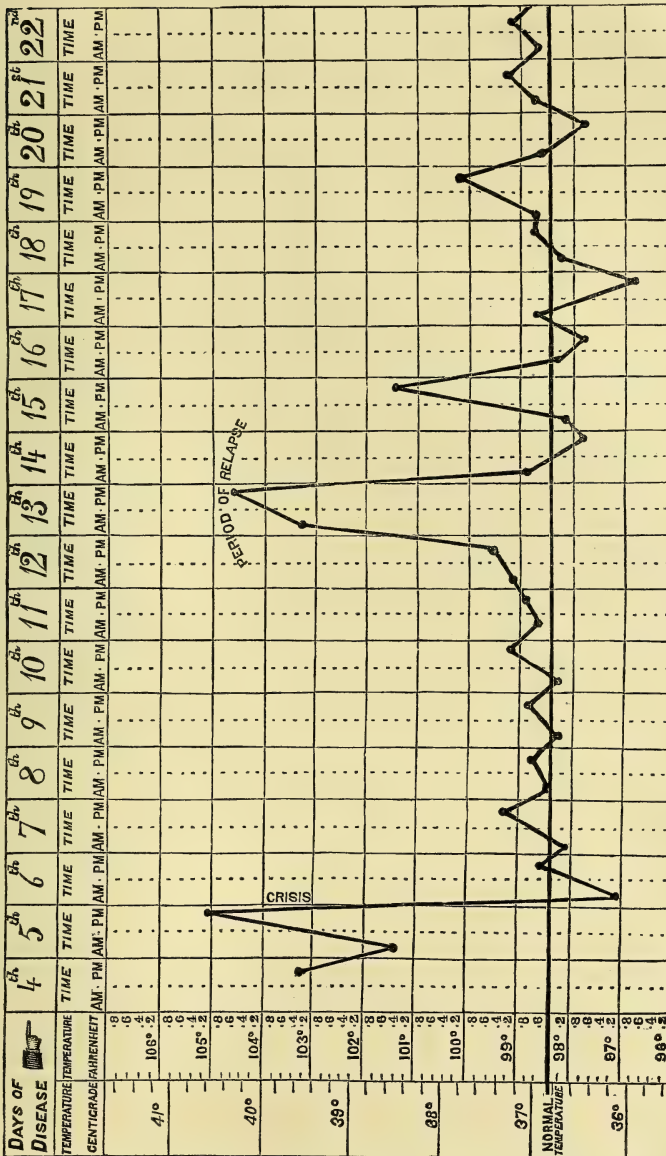


Fig. 86.

generally on or about the *seventh* day, and its advent is rarely prolonged beyond the *eighth*. This change is ushered in by a most profuse perspiration, in some instances with an eruption of miliaria vesicles, which breaks out from the whole surface of the skin, and in the course of a few hours the patient appears nearly well. More rarely the change is indicated by epistaxis as well as by perspiration, or by profuse diarrhœa, catamenial discharge, or hæmorrhage from the bowels; and after either or all of these

apparently critical changes have been established for a few hours, there is a complete and abrupt cessation of all the distressing symptoms. So rapid is the critical change that the temperature may fall 10 or 12 degrees in a few hours. The pulse quickly regains the natural standard, the tongue cleans, the appetite and sleep return, and the countenance resumes its tranquillity. This alteration is very often effected within a few hours, and on the following day the patient generally considers himself in all respects quite well, and may so continue to improve rapidly for *four* or *five* days. During this period, however, there are some patients who suffer from violent muscular pains in the limbs. Symptoms of grave depression may also supervene, so that death may take place by collapse or syncope.

(c.) *The Relapse or Recurrent Paroxysm.*—For about *seven* days after this critical change is a period of perfect health, and between about the *twelfth* to the *twentieth* day from the commencement of the illness, but generally on the *fourteenth* day, a sudden relapse occurs “in ninety-nine cases out of every hundred.” This relapse commences suddenly, and is the characteristic peculiarity of the disease. Like the first seizure, it begins by rigors, headache, loss of appetite, vomiting of green fluid, which is quickly followed by a hot skin, quick pulse, and a coated white tongue, confined bowels, followed by delirium, so that the phenomena may be exactly represented as a repetition of the first attack. In the interval of convalescence between the first and second attacks the pulse often becomes slow to an extreme degree, as slow even as forty-five beats in the minute; but, suddenly on the relapse commencing, it again rises to 120 or more. In ordinary favourable cases perspiration would again occur in *two, three, four,* or *five* days, and the patient would be relieved as before. The chemical qualities of the sweat have never been determined in cases of relapsing fever; but it has a very sour and peculiar smell. In other cases, however, uncontrollable vomiting, great thirst, very rapid pulse, a hectic-looking circumscribed flush of countenance, jaundice, watchfulness, delirium, and death, may terminate the case. The temperature during the relapse may reach a higher point than in the first attack, followed by a similar sudden depression. The pulse generally bears a definite relation to the temperature. It rises with the fever heat and falls with the crisis, and is often rapid in cases otherwise favourable. The urea is diminished during the fever period and in the interval, and is increased during permanent convalescence (HERMAN, FOX).

In some cases the relapse is very slightly marked, and indicated merely by a comparative increase in the rapidity of the pulse and a greater heat of skin than were present on the previous day. The duration of the relapse varies from a few hours to several days; the average being from *three* to *five* days, or less than that of the primary paroxysm. In some cases the relapse lasts less than *twenty-four hours*; in a few it is prolonged to *seven* or *eight days* (MURCHISON); and Dr. Lyons observed in the Crimea that it was occasionally protracted to *twenty-one days*. A second relapse, and a third, a fourth, and even a fifth, are reported to have occurred during epidemics of *relapsing fever*, but the cases are of rare occurrence.

In nearly a fourth of the cases, jaundice is present, and is sometimes intense (JENNER). If present during the first attack, it may disappear

before the relapse, and not recur; or it may occur only on the relapse. It is important to notice, that while the jaundice continues, the stools still retain their natural hue, and may even be darker than common, and at the same time the urine may be frequently loaded with bile. Epigastric tenderness is most marked in the cases where vomiting occurs. When pregnant women are attacked with *relapsing fever* they usually abort, sometimes in the first paroxysm, but often in the relapse, and this event renders the prognosis more doubtful. There is a tendency in *relapsing fever* to the occurrence of sudden death. It may happen by syncope, immediately after the critical periods, when the pulse becomes so very slow. It may also happen in the progress of the case, during either of the severe periods—namely, the primary attack or the relapse. Its approach is indicated by a deep dusky hue of the face, lividity of the hands and feet, and a purple marbling of the whole surface. The trunk feels cool, and the hands feel cold; and without suffering any severe pain, or without sustaining any sudden discharge of fluids, a state of collapse insidiously comes on, from which the patient is unable to be roused. Death may follow in a few hours, generally from *twelve to twenty-four*, even after it was supposed that danger had been escaped. But death is a rare termination to relapsing fever; and when it does occur, the fatal event more commonly happens during the primary fever than during the relapse (JENNER).

(d.) *Duration of the Fever, and Convalescence.*—Under ordinary circumstances, when there are but two paroxysms—*i.e.*, one primary paroxysm and one relapse—the total duration of the fever extends to about *three weeks*; and convalescence is very slow, much slower than in typhus. The *relapsing fever* is very exhausting in its effects upon the constitution; and, dating the period of convalescence from the termination of the last attack, the time taken to recover is in most cases unusually long. To those, indeed, who suffer from more than one relapse, it is almost impossible to have health completely restored for a long time. They become a prey to various sequelæ of fever, or they continue sickly for many months, with pallid countenances, puffed ankles, palpitations, extreme debility, noises in the ears, dimness of vision, diarrhœa, or dysentery. Dysuria is a frequent complication amongst women during the relapse. In many instances during the epidemic of 1847 and 1848 in Ireland, convulsions occurred in cases which otherwise seemed to be progressing favourably, and death invariably followed them. Dr. William Robertson observed in Edinburgh (and the Irish physicians record a similar observation) that delirium of a violent character occurred during convalescence, or after the critical discharge had taken place. It generally came on suddenly, with incessant talking, a rapid weak pulse, followed by perfect unconsciousness, flushed face, and contracted pupil.

Sequelæ of Relapsing Fever.—One of the most common results is the occurrence of excessive pains in the limbs, more especially expressed about the knee and ankle joints; and even the long bones appear to be the seat of these pains in some cases. Combined with those local pains, the joints may swell; and the kidneys are in danger of being implicated. In some respects, therefore, the dangers are similar to those which attend scarlatina. The lymphatic glands are also liable to swell, and so

is the parotid gland. Anasarca and furunculi may likewise supervene. The most important of all the sequelæ, however, is a remarkable affection of the eyes—a form of ophthalmitis—which the late Dr. Mackenzie of Glasgow first described under the name of “*post-febrile ophthalmitis*.” It may occur during the course of the fever, but more often during convalescence, and even some months after convalescence has been established. It was very common in Glasgow after the epidemic of 1843; and assumed two different forms, namely:—(1.) An active inflammation of the shell of the eyeball and of the iris; (2.) An amaurotic state due to congestion of the choroid and the retina (Dr. ANDREW ANDERSON). These two forms of disease may be associated with two characteristics of the fever itself—namely, “the tendencies to visceral congestions, and to rheumatic-like pains;” while the constitutional character of the ophthalmia is in many cases proved by the unhealthy aspect of the blood, which flows dark, in some cases almost tarry, from the vein. Bleeding is found to be the most effectual—the only effectual—mode of cutting short this dangerous ophthalmia; and a very small loss of blood is found to be sufficient. This is especially noticeable, because, during convalescence, tonics and quinine are most likely to be thought of (ANDERSON).

Treatment.—All physicians agree that in the primary attack little medicine is required after opening the bowels by *castor-oil*; or by five grains of the *compound colocynth mass*; or by two grains of *blue-pill*, and three grains of *extract of hyoscyamus* given at night, and followed in the morning by two drachms of the *sulphate of magnesia in compound infusion of roses* (MURCHISON). The symptoms are not readily under the control of remedies; the vomiting is often especially persistent. Five grains of *calomel*, with one grain of *opium*, has been found more efficient in subduing the severity of this symptom than counter-irritation or effervescing draughts. The violence of the headache in well-fed, or otherwise healthy patients, is best subdued by *leeches* or *cupping*; and in the poor, weakly, and ill-fed, by *blisters* to the nape of the neck, or by *dry cupping* there. Till the crisis comes, the symptoms may be mitigated, but not altogether relieved, and cases of ordinary severity are better left to nature without interference on the part of the physician. Active purging is to be avoided; and the action of the kidneys is to be kept up by the frequent use of small doses of *nitre* (ROSS, HENDERSON, CORMACK, WARDELL, MURCHISON). “By keeping up the action of the kidneys from the first” we may “prevent the occurrence of uræmic intoxication, which is one of the main causes of death in uncomplicated cases.” With this end in view *nitre* is to be given as follows:—From one to two drachms of *nitre* are to be dissolved in two pints of *barley-water*, acidulated with a drachm of *dilute nitric acid*, and sweetened with a little syrup. This quantity is to be used up during the twenty-four hours. *Acetate of potash* and *nitric ether* may be used for the same purpose; but the *nitre* has the additional advantage of keeping open the bowels (MURCHISON). Contamination of the blood with urinary products is the great danger in cases of relapsing fever; and therefore particular attention must be paid to the state of the urine, especially towards the period of the first crisis. When the daily amount is much reduced, or if entire suppression should ensue, and particularly if stupor, confusion of thought, or drowsiness should supervene,

the bowels are to be freely moved by *compound jalap powder*, or by a *turpentine enema*. Determination to the skin should be promoted by the hot-air bath; and saline diuretics may be given every two or three hours. No means hitherto discovered will prevent the occurrence of the relapse. The surface of the body should be frequently sponged over with cold or tepid water. Stimulants are not usually necessary, but they may be required in the stage of languor or exhaustion ensuing on the crisis, or in cases where great debility has preceded the attack. If any anæmia exists, or if an anæmic murmur can be detected, stimulants must be given early. When jaundice appears, *nitro-hydrochloric acid* should be given in combination with *nitre*, as in the following formula:—Twenty minims of *hydrochloric acid*, with ten minims of *nitric acid*, every three hours, each dose diluted with the drink of *nitre* and *barley-water* already prescribed (MURCHISON).

SIMPLE CONTINUED FEVER.

LATIN EQ., *Febris Continua Simplex*; FRENCH EQ., *Fèvre Continue*; GERMAN EQ., *Fieber von Unbestimmtem Character*; ITALIAN EQ., *Febbre Continua Semplice*.

Definition.—*Continued fever having no specific character.*

Pathology.—It is stated in the *Medico-Chirurgical Review* for October, 1869, that the College of Physicians have retained this name of a disease in deference to the opinion of a large body of practitioners, who find it impossible to class all their fever cases under the more definite varieties just described. On the other hand, there are not a few physicians who doubt the occurrence of *simple continued fever* as distinct from the continued fevers already mentioned. There can be no doubt, however, that cases of an anomalous or mixed nature do occur, concerning which a decided diagnosis cannot be given from the general symptoms merely; and thus the term *continued fever* “has become a refuge for many cases of an uncertain character.” While the distinct forms of continued fever previously described are capable of being recognised, there can be no doubt that cases of fever do occur in this country which run a continuous course, but have no other specific characters, and which, in many respects, do not seem quite the same as those with which we are now familiar, and which cannot at once be clinically recognised. For instance, in the very interesting investigation into the nature of “*typhus* and *typhoid fever*,” by the late Dr. Murchison, recorded in the forty-first volume of the *Medico-Chirurgical Transactions*, it is related that about 200 cases are left out of consideration altogether, because they were “doubtful cases,” which could not be classed as either *typhus* or *enteric* cases. A similar class of “doubtful cases” are seen to occur in places where *yellow fever* and *remittent* fevers prevail, which cannot be classed as either the one or the other form of fever. Again, in the Mediterranean latitudes, there is a “gastric remittent” fever described, which seems to have many characters in common with some of the forms of continued fever (CRAIGIE, MARSTON). Wunderlich and Murchison both describe febrile phenomena which are of so

anomalous a kind that they refer them to a combination of the poisons of typhus and typhoid fevers, so that the characters of each do not remain distinct. So likewise Dr. W. T. Gairdner, in stating that of late (from 1853 till 1862) the cases of fever in the Edinburgh Royal Infirmary have not been more than seven or eight cases a month under his notice—including numerous anomalous fevers which have prevailed, and which have sometimes quite overborne the numbers of genuine *typhus* and of *enteric* fever together (*Clinical Medicine*, p. 154). A fever termed "*gastric*" is distinctly described by Dr. Andrew Anderson, of Glasgow, which would also come under this head. The "*bilious remittent*" or "*bilious typhoid*," described by Greisinger, of Tübingen, is another form of continued fever which requires investigation. He observed it at Damietta, in Egypt. It is probably a malarious fever of a remittent type, or some form of malarious yellow fever. The sudden fall of the pulse from 120 to 75 was not attended by corresponding improvement of the patient, but was the forerunner of severe typhoid symptoms and jaundice. The mortality was equal to nineteen per cent.; and quinine was found to be of signal service (MURCHISON). The works of Morehead and Sir Ranald Martin may also be referred to for various anomalous forms of continued fever. The "ardent continued fever" of India may be quoted as an example of a very serious disease.

It is these "doubtful or anomalous" cases especially which require careful and special methods of investigation. They are of the utmost importance to science, for more extended information regarding them will either connect them with forms of fever between which they seem to stand; or these "doubtful cases" will eventually separate themselves into distinct forms, whose history is still unknown. In such doubtful cases observations regarding the correlation of temperature, the excretions, the succession of phenomena, and general course of the disease, are imperatively demanded (p. 257, *ante*). The poisons of tropical fevers especially require to be carefully studied, and the phenomena of the febrile state which accompanies them embrace some medical problems of the most abstruse nature. Physiological data of an exact kind are now beginning to rise around us, which will give a standpoint for comparison in the study of the phenomena of fever and air temperature in the tropics. Extremely important observations have been worked out by Dr. A. Rattray, and published in *Proceedings of Royal Society*, June 16, 1870. With all such exact information, and the improved physical aids to investigation, it behoves the physician and pathologist to investigate medical problems with the same logical rigor and severity as a chemical or an astronomical theorem demands. "The power of observation in medicine is a kind of tact which ought to be cultivated with the same assiduity as the chemist practises when he learns how to manage his delicate manipulations, or the astronomer when he wields his wondrous tube. In medicine the observation and recording of phenomena have been held to be an easy and trifling task, which any tyro was competent to do. Hence half the error and uncertainty of medicine. Inaccurate, that is, erroneous and incomplete observation, has been the cause that, till within these few years, the fevers of cold countries have been so absolutely uncomprehended, and that the fevers of hot countries are still shrouded in obscurity. The most

valuable addition any one could at present make to our knowledge of tropical fevers would be a simple record of all the cases in an epidemic. These cases should be observed with the keen tact of a Chomel, and recorded with the fidelity of a Louis. We want no explanation or word of comment added to them; we want merely the cases. Then, when the numbers are sufficient, we should certainly begin to put order into this chaos. And let not any one who may have the opportunities be deterred from the task by that fallacious and, we beg to say, most reprehensible argument, with which some people may favour him,—namely, that his cases will be ‘tedious,’ ‘heavy,’ and ‘unread.’ Unread they will be, certainly, by some of the profession, who consider their routine practice as great an effort as their intellect will bear; but read and analysed, we will venture to say, they will be by those who think no labour too great if they can fix safely the foundations of medicine, and for whom, if *accurately* reported, no cases can be too long, no observations too minute. Only, before the task is commenced, let the observer feel that his powers are equal to it; and let him bear in mind the example of Louis, who recorded most carefully for a long time, that he might train himself to this duty, and then, throwing his probationary cases aside, as too uncertain for use, began to make those remarkable series of observations which have linked his name for ever with the greatest improvements in modern Medicine—the employment of a correct method of studying his science” (Dr. PARKES in *Brit. and For. Med.-Chir. Review*, Oct., 1850, p. 435).

FEBRICULA.

LATIN EQ., *Febricula*; FRENCH EQ., *Fièvre Ephémère*; GERMAN EQ., *Febricula*; ITALIAN EQ., *Febbricola*—Syn., *Febbre Efemera*.

Definition.—*A simple fever in which the expression of the febrile phenomena is of very short duration, lasting, as a rule, for not more than twenty-four, thirty-six, forty-eight, or seventy-two hours, attended with a frequent, full, and often firm pulse, white and coated tongue, pains in the loins and limbs, thirst, constipation, a scanty discharge of high-coloured urine, hot and dry skin, sometimes an eruption of roseola or erythema about the loins or thighs, coming and disappearing with the fever (MOREHEAD); severe headache, sometimes acute delirium, and flushed face. The subsidence of the fever is generally associated with copious perspirations, or herpetic eruptions.*

Pathology.—We do not know of any specific poison as the cause of such phenomena as those detailed in the definition; neither have we any evidence that febricula is a contagious or miasmatic disease. There are many different causes which are known to be capable of exciting expressions of febrile phenomena similar to those mentioned in the definition,—such as exposure to great heat or cold, surfeit, inebriety, mental or bodily fatigue or excitement; and, perhaps, specific poisons, in uncertain or otherwise mild doses, such as the typhus or enteric fever poisons. It is also associated with local and functional disturbances—*e.g.*, catarrhs (bronchial, gastric, intestinal, urethral), milk fever, the fever of alcoholism. Such cases are especially characterised by the apparent

severity of the febrile state, the shortness of its course, and the absence of any local complication or specific eruption.

TYPICAL RANGE OF TEMPERATURE IN A CASE OF FEBRICULA. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wunderlich).

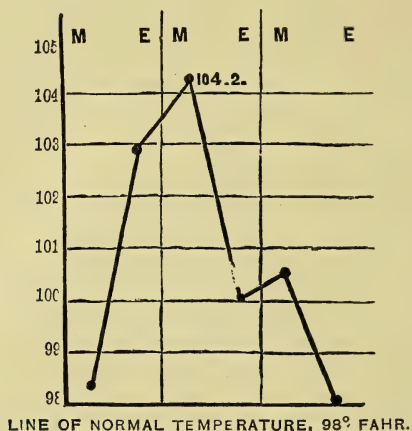


Fig. 87.

The pathology of such apparently simple fevers demands extensive investigation, especially in the tropics, where febricula is a very common disease. The *ardent fever*, the *sun fever*, the *common continued fever* of Burmah and India generally, are all names which indicate severe or protracted cases of febricula—cases of fever which “differ in degree rather than in character” (MOREHEAD). They are common in those parts of India which do not experience much of the influence of the monsoon rains.

Symptoms.—Cases of true febricula commence with chills, followed by reaction, and this by perspiration. They are characterised by a quick and comparatively sudden rise of temperature, as indicated by the preceding diagram—a rise of temperature in a few hours to 4°, or 5°, or 7° above the normal temperature of 98° Fahr. They have thus a sudden beginning and a rapid arrival at a maximum—phenomena which are only shared in by some forms of *malarious fever* (*intermittent*), *variola*, *measles*, and *pneumonia*. The defervescence also is characteristic. In febricula the maximum of temperature may only last for a few hours, or a single day, when the defervescence sets in rapidly; so that in twenty-four or thirty-six hours the body will have returned to its normal heat—an example of *pure crisis*. No other febrile disease gives expression to similar phenomena; and the correlation of temperature to the other phenomena, especially to the excretion of urine, is also peculiar. The urine presents, during this disease, the very type of febrile urine. On the second or third day the amount of urine is extremely small (twelve to twenty ounces), of very high specific gravity (1035–1037), with the solids and sulphuric acid very much over the average, and the amount

of urea large. When the temperature falls, the quantity of urine rapidly augments. The increase of urea and of the solids is not so much, however, as in the height of the more severe and prolonged fevers (PARKES *On the Urine*, p. 243).

TYPICAL RANGE OF TEMPERATURE IN A CASE OF PROTRACTED FEBRICULA (*Ephemera protracta*). THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wunderlich).

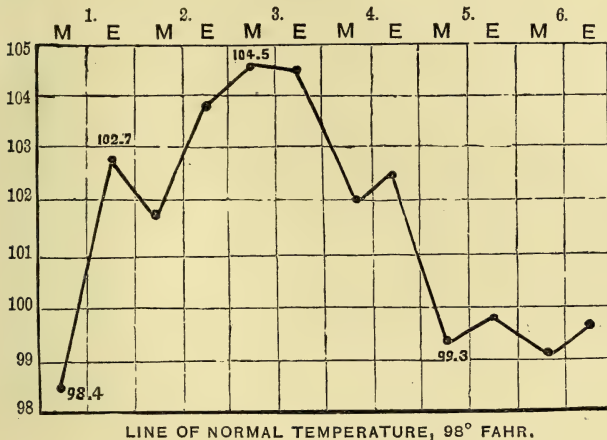


Fig. 88.

In cases of more protracted febricula, the early phenomena are similar to the shorter cases, and the protraction is mainly due to the slowness of the defervescence—an example of *lysis*. The phenomena of such defervescence, as indicated by the range of temperature, are shown in the foregoing diagram. As a rule, these fevers are not serious; but the degree of reaction has always a relation to the state of the constitution, whether sthenic or not (MOREHEAD).

Diagnosis.—Contrasted with the commencement of enteric fever, so far as temperature is concerned, it rises high more early in febricula than in enteric fever. On the first day it will have reached 103° Fahr., or more, while that point is seldom reached in enteric fever before the third or fourth evening. The ascent is continuous in febricula till the highest point is reached; and it continues high for twenty-four or forty-eight hours, with morning remissions. Such a fever may be induced in children by the heat of the sun alone; and it ought to be over within ten days (FOX).

Treatment of Febricula.—Such means as emetics, purgatives, tepid sponging, diaphoretics, and antiphlogistic regimen are to be employed. In plethoric individuals, where there is much headache and flushing of the face, leeches to the temples may be expedient, but such remedies are not often necessary (MOREHEAD).

[SPECIFIC] YELLOW FEVER.

LATIN EQ., *Febris Flava*; FRENCH EQ., *Fièvre Jaune*; GERMAN EQ., *Gelbes Fieber*—Syn., *Gelbfieber*; ITALIAN EQ., *Febbre Gialla*.

Definition.—*A specific fever of a malignant and continuous type; occurring, as a rule, only once during life, and propagated by contagion. It is attended by yellowness of the conjunctive and skin, delirium, suppression of urine, interstitial hæmorrhages and hæmorrhages from the stomach, mouth, nares, and rectum (black vomit, black stools), a slow and, at times, an intermittent pulse. It is limited to very definite geographical limits, never having been known to propagate beyond 48° north latitude, nor without a temperature of 72° Fahr. at least. It has been imported into Lisbon, into St. Nazaire (in the department of the Lower Loire), into Plymouth and Southampton, where the imported cases have run their course and proved fatal, but the disease did not extend to others. It has also been imported and become epidemic as far south as Monte Video. It has occurred as high as 4,000 feet above the sea-level (Newcastle, in Jamaica). But, as a rule, it is endemic in low districts on the sea coast, and rarely occurs over an elevation of 2,500 feet above the level of the sea.*

Pathology.—Yellow fever of a specific kind must now be regarded, from an enlightened consideration of its history, as one *sui generis*, and specifically different from the remittent and intermittent fevers (in which the patient may become yellow), and from any other form of malarious yellow fever (CULLEN, CHISHOLM, BLANE, WOOD). The College of Physicians has given the following definition of yellow fever, namely:—*"A malignant epidemic fever, usually continued, but sometimes assuming a paroxysmal type, characterised by yellowness of the skin, and accompanied, in the severest cases, by hæmorrhages from the stomach (black vomit), nares, and mouth."* There is obviously mixed up in this definition a malarious form of yellow fever and the true specific form, which it is desirable to separate and distinguish. There are those who believe that these fevers are the same in kind, but various in degree; that certain atmospheric conditions, such as great heat or humidity, acting on a predisposed frame, will produce all the symptoms of the most malignant fever; that the intensest form of yellow fever is but the developed degree of the common bilious derangements peculiar to hot and rainy seasons (TOMMASINI, CLEGHORN, LIND, HUNTER, ALISON, CRAIGIE, MARTIN, MUNRO). The opinion regarding the pathology of yellow fever, which holds that it is "*an intense form of the bilious remittent of the tropics,*" has given rise to much discussion. The investigation of this point, however, is attended with extreme difficulties, and is to be carried out with reference to two questions especially, namely:—(1.) The type or mode of progress of the symptoms in mild and severe cases, compared with cases of remittent fever in all grades of severity and stages; (2.) The pathological characters of the morbid processes which take place in severe forms of yellow fever, compared with those of remittent. Three opinions have thus been held regarding the essential nature of yellow fever. These are,—(1.) That there is a malarious form of yellow fever which is an intense and virulent form of remittent, and which becomes more or less a continued fever

(CLEGHORN, LIND, HUNTER, ALISON, CRAIGIE, MARTIN). (2.) That it is a continued fever of a specific kind, different from all other continued fevers (CULLEN, CHISHOLM, BLANE, WOOD, HIRSCH, ARNOLD, MACLEAN). (3.) That it is a mixed fever, of a type variable between the remittent and continued forms (JACKSON, MOSELEY). The grounds upon which the first of these opinions is accepted are—that in the symptoms and effects, progress and pathology, of remittent fevers and ordinary cases of yellow fever, we are unable to discover any essential differences, but merely what is due to intensity of morbid action, degree, and rapidity of progress. Comparisons have been drawn in this way between the summer and autumnal *remittents* of the south of Europe, the *remittent* fever of the Mediterranean, the tropical *remittent* of the East and West Indies and Central Africa, the Bulam fever, or the fever of Sierra Leone and Fernando Po, on the one hand; and between some of the cases of the *yellow fever* of Cadiz, Gibraltar, Malaga, Carthage, Leghorn, Vera Cruz, Havana, Jamaica, St. Domingo, the West Indies generally, and the United States, on the other. There are also cases in which no distinction can be drawn between the symptoms, the effects, or the rapidity of action, if the case (considered to be a remittent) is compared with some cases of so-called yellow fever. In other words, it is not possible to distinguish some cases, and say with certainty that they are cases of remittent rather than of yellow fever, or of yellow fever and not remittent.

But on looking carefully into the history of yellow fever, on which volumes have been written, the conclusions arrived at seem to be—(1.) That there is a specific yellow fever, propagated by a contagious virus or poison which multiplies itself by its passage through the human system, and which reproduces the same specific true yellow fever. The type of this fever is continuous. Pyrexia, delirium, suppression of urine, black vomit, are the leading symptoms of this fever—the *hæmagastric pestilence*, as it has been also called. (2.) That there are other fevers, and especially severe marsh fevers, in certain geographical limits, which have a close resemblance in symptoms to the contagious and specific yellow fever.

Considering true yellow fever, therefore, as one of the specific continued fevers, having a certain limited geographical range, it is necessary at the outset to define what is meant by true or pestilential yellow fever, and what are the diagnostic symptoms which distinguish it from the diseases which resemble it, but which are really dissimilar. It may be asserted unconditionally at the outset, that the significance of the symptoms of yellowness of the skin and black vomit is very small indeed as diagnostic marks. Different shades of yellowness of the skin have been described as forming a prominent symptom, not only in epidemics of yellow fever, but by all writers on the fevers generally of hot countries. Yellowness of the skin in remittent fevers arising from malaria has been noticed in all climates, although it is certainly most common in those of the western hemisphere. Cleghorn observed it at Minorca; Irvine in Sicily, in the autumnal fevers; Burnett in the Mediterranean fevers, of all depths of colours. In the fatal fever of the Mysore country, yellowness during some years has been almost universal; so also in Batavia, and in the fevers of Rangoon in 1824-25. A fever attended with yellowness of the skin ranged

like a pestilence in Rohilcund from 1836 till 1840, at the same time that a fever with symptoms of plague was prevalent in Marwar and Meywar, and common remittents and intermittents prevailed between these districts. A fatal remittent fever attacked Her Majesty's 29th Regiment in 1844 at Ghazepore. In many cases there was "deep jaundice;" and in one case a symptom occurred which has been often witnessed in the West Indies—namely, sloughing of the penis and scrotum (PARKES, "On the Contagion of Yellow Fever," in *Brit. and For. Med.-Chir. Review*, Jan., 1848). So also in some forms of specific yellow fever, as in the *algide* form, so well described by Dr. Lyons, in the Lisbon epidemic of 1857, yellowness was very often wanting, "many cases dying without having ever exhibited a trace of yellowness on any part of the cutaneous surface, or even the conjunctivæ during life" (LYONS, *On Fever*, p. 338).

Black vomit is an event which occurs in fevers of marshy origin, and in the so-called "seasoning fevers," as well as in gastric affections of a purely tropical nature, in *coup de soleil*, and in some injuries of the brain. In the remittent fevers of the African stations, black vomit is not an unusual occurrence. So also in some of the yellow fevers of America, which are of marshy origin, black vomit is a usual symptom (BOOTT). These two events—namely, yellowness of the skin and black vomit—being of themselves insufficient as diagnostic marks of true or specific yellow fever, additional grounds of difference are found—(1.) In the type of the fever, which is continuous and not remittent; (2.) In the fact that it occurs, as a rule, only once during life; (3.) In the fact that it is propagated by specific media from infected persons or places to others. But although in no one of those phenomena, taken singly, except in that of its communicability from person to person, do we find any definite characters to rely upon to prove the existence of a formal and specific yellow fever; yet, in the general assemblage and collocation of symptoms, peculiarities do present themselves which are easily discernible by an experienced eye.

Though the subject of tropical fevers is too little known to warrant decided opinions on many points, yet the true yellow fever, or hæmagastric pestilence, is now so clearly stamped with characters so peculiarly its own, that it takes its place as a specific fever of a continuous and generally rapidly fatal type. Its pathology is best exemplified in the history of such isolated outbreaks of it as are to be found in the cases of the "Hussar" (BLANE), the "Bann," the "Kent," the "Scout," the "Eclair," the "Hankey," the "Icarus," the Lisbon epidemic of 1857, and the importation of the disease from Havanna into the port of St. Nazaire by the "Anne Marie," in 1861; and no description of yellow fever can be complete which does not give an account of some of these remarkable instances of this disease. The period of incubation of the fever at Boa Vista was found to vary from two to eight days; and the facts recorded in the history of the spread of the fever over the island show that certain persons living nearest and most in contact with the sick were first attacked. When the disease appeared with great virulence in the island of Grenada in 1793, its spread by infection first attracted notice by the arrival of the "Hankey" from Bulam, on the west coast of Africa, on the 19th February, 1793, some days before the fever broke out on the island. In this vessel, at Bulam, the fever had prevailed for

five months before, to a great and fatal extent (CHISHOLM, Sir WM. PYM).

A most interesting and consistent account of a yellow fever outbreak has been given by J. D. Macdonald, Esq., R.N., M.D., F.R.S., Professor of Naval Hygiène (Journal of the "Icarus," *Annals of Military and Naval Surgery*, p. 126). Professor Macdonald's account is especially interesting in the following particulars:—(1.) It tends to fix more definitely than has yet been done a period of incubation. (2.) It shows that in certain localities specific yellow fever is always in existence, expressing itself by sporadic cases every now and then, especially in the West India Islands, as a central focus to that geographical district where the disease is endemic, and where it has assumed in some places an epidemic character. (3.) It tends to corroborate the circumstantial evidence already accumulating, that the disease may be propagated by *fomites*, much as in the case with typhus and scarlet fevers. (4.) As with typhus and typhoid fevers, so with yellow fever, "the doctrine of its spontaneous origin" can have no foundation to satisfy the rational mind. Bearing on the *period of latency*, Professor Macdonald shows that yellow fever was rife at St. Domingo when the "Icarus" arrived there, and the professional zeal of Dr. MacLagan, the Assistant-Surgeon of that ship, led him to visit some half-dozen cases on shore at Port-au-Prince. He is believed to have visited these cases on the 10th of June, and the first appearance of the symptoms which ended in his much lamented death betrayed themselves on the 24th of June, *i.e.*, *fourteen* days after having first visited the sick, and after the ship had been at sea four days. Another case is cited—that of a boy, Lambert—which shows that the period of latency could not have been less than *eight* days. The experience of the Lisbon epidemic marks the time of incubation as varying from two to ten days, and in some instances extending to fifteen days. The importation of yellow fever by the ship "Anne Marie" into St. Nazaire—a town in the department of the Lower Loire, about 47°30' north latitude—in the summer of 1861 (*Ann. d'Hygiène*, Oct., 1863, p. 416), confirms the belief in a lengthened period of incubation; and from a careful analysis of the history of specific yellow fever cases, I think it will be found that the period of incubation tends to lengthen with the transportation and propagation of the disease into the cooler latitudes more remote from the equator.

The history of the importation of the disease into St. Nazaire is as follows:—About the 13th of June the "Anne Marie," a wooden sailing vessel, laden with cases of sugar, left Havanna, having been there a month during the prevalence of a severe epidemic of yellow fever. None of the sailors suffered so long as she lay at Havanna, except from a little depression, loss of appetite, and a certain tendency to vomiting. After leaving Havanna for France there was no sickness for seventeen days. On the 1st of July two sailors were attacked (without precursory symptoms) with violent shivering, pallor of the face, injection of the eyes, congested lips, and continued delirium. One died in twenty-three hours, the other in one hundred and ten. On the following seven days other persons were attacked, making in all nine cases out of a crew of sixteen persons. Only two deaths occurred. The ship arrived at St.

Nazaire on the 25th of July with seven men still sick, but all of them convalescent, thirteen days having elapsed from the date of attack of the case last taken ill. Therefore, having had no deaths and no fresh cases for ten days, the "Anne Marie" was not placed in quarantine at the end of her voyage on arrival at St. Nazaire. Near her, as she lay in that port, there were anchored two ships of the Imperial Navy—namely, "Le Chastan" and "Le Cormorant," the former touching her. Three other ships, "L'Orient," "Les Dardanelles," and "L'Arequipa," were also near her. According to the custom of the port, the sailors of the "Anne Marie," being only engaged for the voyage, quitted the vessel on her arrival, and were dispersed throughout the town. The commander, who had been ill, went home; the vessel was left to the second in command; and the places of the men were taken by seventeen fresh men, to discharge the cargo of sugar. These men were strong, very robust, and completed the discharge of the ship in eight days. Of these men twelve or thirteen were attacked with yellow fever, and many of them died. "Le Chastan," having been close alongside the "Anne Marie," left on the 29th July, and sailed to Indret, on the Loire, forty-four kilometres distant. Her crew, five in number, seemed in perfect health when they arrived at Indret; but on the 1st August (*i.e.*, three days after leaving St. Nazaire) a man fell sick of yellow fever; then the remaining four by the 5th of August were all ill; and by the 10th of August all the five men, the crew of "Le Chastan," were dead. These men had all been on board the "Anne Marie" for about a quarter of an hour. "Le Cormorant" quitted St. Nazaire on the 10th; her crew, six in number, were then in perfect health. On the 14th of August two of her men were taken ill, and died on the 26th, of decided yellow fever. Eight vessels in all had been near the "Anne Marie," and sick patients had gone on shore at St. Nazaire and its neighbourhood, communicating yellow fever to two or three, and slighter illness of the same kind to other persons who were about them. Altogether, it is on record that forty-four cases of yellow fever were communicated by the "Anne Marie," resulting in twenty-six deaths. During this period the heat was more like that of a tropical than of a European climate; but neither yellow fever nor anything like it had ever been seen in the district. It cannot now, therefore, be disputed that, by the arrival of the "Anne Marie," yellow fever was imported into St. Nazaire, and by propagating itself occasioned an outbreak of the disease at that port. And when it is remembered that fatal cases of specific yellow fever have already occurred at our own doors,—in Plymouth Sound and Southampton Water,—there is a like possibility of the importation of yellow fever into English ports under climatic conditions favourable to its development, and when we might least expect it, through the rapid and frequent communication which we now have with the West India Islands, the very central focus of yellow fever. "We have no more reason to trust in our own fancied security than the people of Monte Video had before yellow fever made its appearance amongst them" (J. D. MACDONALD). The *Lancet* of Feb. 12, 1853, contains "An Account of Yellow Fever as it occurred on board the Royal Mail Steamship 'La Plata,' in the month of November, 1852," from the pen of Dr. Wiblin, the medical superintendent of quarantine at the port of South-

ampton. He there shows that fourteen cases of yellow fever had occurred on board the "La Plata" during her homeward voyage to Southampton from the West Indies. On the morning of the 18th November she arrived at Southampton, where she was kept for two days in quarantine. On the morning of the 28th the fourth engineer of the "La Plata" (Mr. Napier), who had been lodging in the town for eight days, was seized with symptoms of yellow fever, and, after a week's illness, died of the disease. No other cases occurred in the town. The "La Plata" was a wooden ship; and Mr. Napier had been at work on board of her during the time when he was lodging in Southampton. The ship itself, irrespective of sick persons in it, was thus a focus of yellow fever infection; and in our present uncertainty as to the exact incubation-period of the disease, it cannot be said absolutely that the disease was not latent in the man when he first landed. Also at Southampton in 1852-53, the landing of yellow fever patients from infected ships led on three occasions to ill results; but since these occurrences twenty-six years ago, persons more or less ill with yellow fever have on numerous occasions been landed at Southampton from West India steamers; but in no case has it ever been suspected that their disease has spread to other persons, nor did anything of this kind arise in connection with the case of the engineer (Napier), who ran the whole course of his disease in Southampton. But that England is not insusceptible of this tropical infection, and that (under favouring circumstances) yellow fever can seriously damage a port-side population in this country, was conclusively shown at Swansea in 1865, by the loss of more than sixteen lives. The atmospheric conditions were, however, exceptional to our climate. The heat especially was almost tropical. The history of the occurrence was this:—(1.) The "Hecla" left Cuba on the 26th July with cases of yellow fever on board, and fresh cases arose successively till towards the end of August. She entered Swansea harbour on the 9th September, with one of her seamen dying, and two others convalescing from the fever. She was immediately moored alongside a wharf, where she landed her sick, discharged her cargo, and remained till the 28th, when remonstrances, which at last had become irresistible, led to her being removed from within the dock. (2.) From 15th September (six days after her arrival) to October 4th (six days after her removal from the dock) Swansea witnessed the entirely new phenomenon of yellow fever attacking in succession some twenty inhabitants of the town, besides others who suffered less definitely, or more mildly; and this not indiscriminately over the whole large area of Swansea, but only in definite local relations to this ship; while at Llanelli there also fell sick, in the same way, three of the crew of a small vessel which had been lying for two days alongside the "Hecla" at Swansea.

In connection with the extension or propagation of specific yellow fever beyond its usual geographical limits of constant existence, "it is a remarkable fact," says Professor Macdonald, "that the deaths from yellow fever at St. Thomas's, before it became a coaling dépôt for the Royal Mail steamers, as compared with the deaths afterwards, bear the proportion of 4 to 64. And in connection with this must also be mentioned an equally important fact, that the combustible and other qualities of coal, exposed to the weather as it is at that island, became much deteriorated."

How far this loss may prove to be a *pabulum* to yellow fever poison is unknown, but the covering in, as well as the inclosure of coal in tropical climates, is worthy of the attention of that mysterious assembly—the “proper authorities.”

All the cases, like the “Eclair,” the “Bann,” the “Imaum,” the “Icarus,” the “Anne Marie,” the “La Plata,” the “Hecla,” agreeing as they do in all their main features, it is impossible to doubt the existence of a multiplying infecting virus as the specific cause of the spread of yellow fever; and in cases where it was transported, imported, and propagated, the fever, except in certain cases, seems to have arisen only in persons who had been exposed to whatever deleterious influence was exercised by the atmosphere of ships in which cases of yellow fever had existed. Moreover, the facts connected with the “Imaum,” the “Icarus,” and the “Barracouta,” show that infected places and persons are alike dangerous to those who are at all susceptible; and in the cases of the “Imaum” and the “Anne Marie,” it was shown that a disease taken in a certain locality, and spreading from person to person, may finally affect a second locality through their medium. It appears also to be quite a mistake to suppose that no individual can communicate the disease to another unless he himself is actually under its influence at the time; or, secondly, to consider such an individual as differing in any essential particular from an infected locality. Indeed, a ship itself is only an individual on a grander scale. This is the view Professor Macdonald takes with regard to the “Imaum” receiving the disease from the officers of the “Icarus;” and it is also the view which explains the events which followed the contiguity of the ships to the “Anne Marie” in the harbour of St. Nazaire. To some extent the disease spread through personal intercourse to persons who were not near the ship,—in one very important case, that of M. Chaillon, a physician at Indret, who is said never to have been near the ship nor the town of St. Nazaire, but who contracted infection from four labourers who came infected from the ship, and whom he attended medically at their houses. He contracted yellow fever, and died. In a second case, one of the ship labourers, who himself had yellow fever, is said to have carried the infection certainly to his wife, and perhaps to an old man in whose house he and his wife lodged. All of them were attacked with yellow fever—the old man fatally. Unquestionably with regard to the “Anne Marie,” and doubtless also with regard to the other ships, *the ships themselves*, irrespectively of sick persons in them, were *foci* of yellow fever infection. The men, therefore, no doubt, carried infection passively, as they might have carried an odour from the ship, or as a student carries the smell of the dissecting-room on his clothes, especially felted textures. “I have often looked upon my own monkey-jacket with horror,” writes Dr. Macdonald, “as the possible means of communicating so formidable a disease to others.” Men thus labouring in the hold of infected ships, without themselves contracting yellow fever there, might carry infection to their homes, in climates and places where yellow fever may prevail. And it is a question how far, like cholera and typhoid fever *excreta*, the poison of yellow fever may not increase in places where decomposing animal material abounds.

With regard to the Lisbon epidemic, it has been proved that cases of

the disease remained concentrated in Lisbon, and did not extend to its neighbourhood, nor to any other part of the kingdom, although the communication by land with all the towns and villages continued active and uninterrupted. In many places there appeared cases of yellow fever, evidently brought from the capital, but in no place was the disease transmitted or propagated in an epidemic form. The official report records, that of 182 persons who left Lisbon for different places in Portugal, carrying with them the germ of yellow fever, which broke out or developed itself in them after their arrival at those places, 86 of them died. *In no instance was yellow fever communicated from them to any other person in the places whither they went.* As to the origin of the disease in Lisbon, it appears certain that the first cases were amongst men employed in the Custom House, or with persons in close communication with them. It showed itself in the streets where these people lived. The disease, which commenced in July, did not become epidemic till September; but it remained stationary for a while in the parishes where it first broke out, and then spread gradually and regularly to other parts. It is therefore presumed that the disease did not proceed from any general cause operating on the whole mass of the population, such as would come from meteorological or unhygienic conditions. It was not persons of different classes, and living in different parts of the city, who were attacked at the same time; on the contrary, it commenced with one distinct class, between the individuals of which there was much communication, and went from house to house, from street to street, without invading more than a certain extent of the city. It is presumed, but not proven, that the disease was imported through the Custom House, where luggage and effects are opened to be passed. Many of the ships which arrived at Lisbon during the epidemic and in the previous months had cases of yellow fever and losses during the voyage; but the only case proved was in the "Tamar," which arrived in March from Brazil, the captain of which owned that he had two deaths from yellow fever on board during the passage. This ship arrived again in September, and reported at Southampton two cases which she had had on board. Lisbon and Oporto have been very subject to these importations since 1849, when yellow fever became epidemic in Brazil. In the report it is concluded,—(1.) That Lisbon is not to be considered as liable to *spontaneous* outbreaks of yellow fever. (2.) But, from its latitude and local conditions (especially inasmuch as it abounds in filth in some localities where population is dense and sewage imperfect), it is favourable for the development and spread of specific yellow fever when brought to the port. (3.) It is concluded that the epidemic of 1857 was imported from Brazil.

Symptoms.—Uniformity in the order and character of the symptoms of specific yellow fever must not be looked for. All the best writers on the subject, whether recording their experience in the West Indies, the west coast of Africa, or the south coast of Spain, are unanimous as to this want of uniformity, and consequently, while direct and faithful descriptions may have been given of each epidemic, yet the results are not uniform. Certain symptoms in certain epidemics vary in their nature and in the time of their accession, while others common to former

visitations are wanting in those which follow. Sometimes the full complement of standard symptoms are present, sometimes they are imperfect and deficient, and sometimes displaced. At one time the diagnostic symptom in an epidemic is the supra-orbital headache. At other times the tongue symptoms are alone diagnostic, or their equivalents are expressed in the uvula and fauces. Intense surface heat, albumen early in the urine, and early black vomit, are among the later symptoms; and smoky pale urine, with perfect blood-corpuscles, take the place of the straw-coloured or bilious urine, with its sediment of tube-casts and epithelial matter. The general appearance of the tongue in well-marked cases is redness of the tip and edges, with prominent papillæ and a creamy surface. Subsequently the fur separates from its middle surface, and lies in white wavy flakes; and the next series of changes consists in separation of the epithelium, which begins at the tip, proceeds to the edges and down the raphe, and may continue till the whole surface is denuded, the papillæ obliterated, and the tongue becomes smooth and dryish, of the colour and appearance of raw beef. Such a condition is generally associated with exudation of blood so free as to coat the mouth and tongue, and collect upon the teeth. The tongue is then usually much smaller and more pointed than in the earlier stages.

A rare manifestation of capillary irritation in yellow fever consists in an efflorescence of the skin in the form of a subcutaneous rash on the chest, extending over the abdomen and arms. Rose-coloured spots of a somewhat circular shape have been noticed on fine, delicate, sensitive skins, varying from the size of a fleabite to what might be covered with the point of the finger. They result generally from mosquito wounds, and become hæmorrhagic at the end of the disease, when it terminates fatally. *Bloody furuncles* appear late in the order of symptoms, and are to be regarded rather as sequelæ. Their most common site is on the wrist, over the metacarpal joints, along the front of the legs, below the scapulæ, and over the hip, in the parotid region, and over the forehead and lip. They are generally in close proximity to the smaller arterial branches, such as the ulnar, radial, anterior tibial, gluteal, intercostal, and facial arteries. They become tender, acuminate, and inflamed, and sometimes form large abscesses of purulent matter, with a pale or inflamed surface, and this chiefly when below the scapula or over the hip. Generally on the legs they are flat, presenting no inflamed appearance, but show a flat purplish vesication, about the size of a split pea or a sixpence.

Two, three, or even four forms or types of yellow fever have been described by authors. These have been very clearly defined by Dr. Lyons, of Dublin, in the Lisbon epidemic of 1857, which he investigated with so much care and enthusiasm. The *types, groups, or forms* which he found capable of clinical recognition are,—(1.) The algide; (2.) The sthenic; (3.) The hæmorrhagic; (4.) The purpuric; (5.) The typhous. The first of these—namely, the algide form—is that which presents the most rapid course, and the earliest and greatest amount of prostration of the vital powers. These are the cases which are suddenly killed with the poison. “The patient, while in the enjoyment of his usual health, and in the midst of his usual occupation, feels suddenly the effects, as it

were, of a sudden blow from a heavy bar on the back, falls down while walking (or if standing), and dies within a few hours in profound collapse, and after exhibiting more or less of the other symptoms of this fever." The countenance became sunken, the eye dull and filmy, the surface cold, and the patient felt cold, depressed, and wretched. The face became of a dirty livid hue, and this appearance extended to the trunk and limbs, the surface then presenting innumerable points of minute venous congestion, and sometimes purpuric spots and patches of various sizes. In extreme cases the lips, the breath, and tongue were cold, with a temperature in the axilla not more than 96° , the pulse being small, feeble, and quick; and when the cardiac action became feeble the radial pulse would be obliterated. The *sthenic form* is a marked contrast to the *algide*. It is especially well-marked in both sexes at the prime of life, and in persons with well-developed muscular frames. Such cases are characterised by well-marked febrile symptoms, severe and persistent headache, much rachalgia at the outset, a high, full, and hard pulse, occasionally thrilling and resisting, with flushed face and throbbing temples. A remarkable elevation of temperature prevailed,—an increase of 3° , 4° , or even 5° Fahr., and in some an increase of 7° Fahr. was observed. Death sometimes took place in a very unexpected manner. In the *hæmorrhagic type* the cases are the most characteristic and appalling. In them epigastric anxiety, with or without heat, and pain on pressure in the epigastrium, is well marked; but their great characteristic is a tendency to profuse simultaneous effusions of blood from various parts and organs—the *hæmorrhage never being single, nor from any one source or organ only*. The cases are fatal at an early period; and all the connective tissue of the body is surcharged with blood. There is less considerable elevation of temperature than in the *sthenic form*. In the *purpuric form* the pyrexial state is well marked, with the conjunctivæ and general surface intensely yellow. Purpuric patches commence and spread—sometimes with surrounding oedema. These patches are manifestly caused by subcutaneous effusions of the colouring matter of the blood; and all varieties and shades of colour and tint are observable. In the *typhous form* two orders of phenomena may prevail. In one class of cases stupor and nervous depression exist from an early period of the fever, with all the other well-marked and characteristic *typhoid* symptoms; and to these are superadded the hæmorrhagic phenomena. In another class of these cases the patient, after passing through the *sthenic* or the *algide form*, would insensibly glide into the *typhoid* state, on the cessation of the hæmorrhages (LYONS).

It is of great importance to attend specially to these forms or types; for, as Dr. Lyons justly observes, much of the discrepancy and apparent conflict of medical testimony on the subject of yellow fever is due to the want of discrimination of those leading characteristics and salient features (LYONS, l. c., p. 375). When the black vomit is plentiful, or the urine free, the intelligence remains clear and unclouded, but the skin becomes cold and damp, the pulse small, and finally extinct at the wrist, and the patient dies of gradual *exhaustion* and *syncope*. According to the amount of febrile excitement, the skin is hot and dry; but in many cases there is a turgid fulness of the vessels, and a tingling heat of the surface, which

is imparted in a remarkable manner to the fingers on feeling the pulse. This feverish heat of skin is much more moderate in cases treated in the open air than in those treated in the wards of an hospital, however well ventilated (J. D. MACDONALD). There is a great irregularity in the temperature of the surface. Sometimes the forehead is the hottest part of the body, occasionally it is the chest. The uncovered parts, in the later stages of the disease, are easily reduced in temperature, and thus, while the exposed chest and extremities may feel cool to the touch, the axilla may raise the thermometer to 102° or 103° Fahr. The highest temperature Dr. Blair has observed was 107° in the axilla.

The pulse is at first quick, and of considerable strength, though still compressible, and may vary in these respects within certain limits until it becomes feeble. In those cases which have proved rapidly fatal there has been a marked gradual decrease in its strength; and finally, when the ferrety eye grows clear, and a pallor of countenance shows the mischief of blood exudation going on within, it is scarcely to be felt.

Observations made on the urine in yellow fever, by Dr. Blair, show that it is always acid in the first stage, and continues so generally till convalescence, when it becomes alkaline, or until it becomes heavily charged with bile. During the early stage the urine is normal in colour, clearness, and quantity. About the third day the colour alters and becomes of a sulphur, primrose, straw, or light gamboge hue, perhaps slightly turbid, and with a little floating sediment. The colour deepens till it becomes yellow or orange; and if the case ends in convalescence, the urine is copious, and may appear black. Sometimes the urine has a pale, watery, smoky appearance, with a layer of blood-corpuscles in the sediment, and sometimes it is very bloody. Albumen appears on the second or third day generally; in some cases as early as the first day; and in a few cases not till the day of death, and after black vomit has set in. Albumen appeared in every fatal case of normal duration. It sometimes ceased suddenly in convalescence, and always before the yellow suffusion of skin and eye, or bile in the urine, had disappeared. Between the eleventh and twentieth day of grave cases it generally disappeared. Its colour was never white. When the urine appeared turbid, it was due to the presence of mucous epithelial matter, coagulated albumen, coats of the urinary tubuli, or fine capillaries of the kidney or mucous membrane, passed out with the urine. The tube-casts are generally short, thick, club-shaped, and opaque, attended with large organic cells and epithelial scales. Crystalline deposits are rare. In females the catamenia are sure to appear, whether due or not. No sign is so dooming as a suppression of urine, *black vomit* not excepted. The alvine evacuations may be black towards the close of the disease, or very dark green, and bilious; but after the black stools have ceased, they are succeeded by evacuations which resemble fine, dark, sandy mud, and named the "caddy stool." As the disease still further advanced, and towards its fatal termination, the dejections again changed their character. They became scanty and mucous, of various consistence and colour. These mucous stools almost always appeared *after* black vomit, and were contemporaneous with the scanty urine before described. The alvine evacuations in yellow fever, from the beginning to the end of the attack,

are always alkaline, except in one instance, that of the black vomit stool: in that it is always acid. Its chemical quality is evidently due to the admixture of a portion of the black vomit, which has descended (if not found in the intestines) by peristaltic motion into the intestines, and mixed with the scanty mucous stool, and in such quantity as not only to neutralise it, but to be in excess. The scanty thick mucous stool—almost a jelly—has generally a little thin serum around it in the bottom of the pot. The bulk of all these varieties of the scanty mucous stool consists of mucus, broken-up epithelial matter, and myriads of epithelial granules. Sometimes little wavy flakes, like morsels of cuticle, are also to be found. They also frequently contain the crystalline bodies of the caddy stool, particularly when they are rather thin and serous. By appearance they would be taken for rectal stools, and the results of tenesmus; but such is not the case. A burning sensation is often complained of, but seldom any tenesmus, and no doubt these stools consist of that mucous matter which we find after death lining the intestinal canal generally. In a few cases where there has been total suppression of urine, these stools have become diarrhoeal. The first ejections from the stomach of a yellow fever patient are seldom seen by the physician. Mucus and bile soon appear, occasionally with a streak or speck of blood, and with violent retching. The ejections are alkaline. Generally after the first vomiting the stomach becomes tolerably settled, until the second stage sets in, on the second, third, or fourth, or as late as the fifth day of the disease. Then, without warning or nausea, but on any trifling provocative, the stomach suddenly ejects a quantity of clear, pale, limpid, or slightly opalescent *acid fluid*—the *white vomit*, which indicates the beginning of the stage of acid elimination, and is generally contemporaneous with the first shedding of epithelium from the tongue. Sometimes the evacuation of this vomit has a critical effect, equivalent to the perspiration of intermittent fever. True white vomit consists of serum, more or less acid, which remains clear on the application of heat and nitric acid. The transition of symptoms from *white* to *black vomit* is generally gradual; and is attended with a “suspicious sediment” of “snuff-like specks” before it merges into well-defined *black vomit*. The stage of acid elimination continues to the close of the disease, and is most intensely manifested during the production of the *black vomit*. The presence of ammonia in black vomit is universal, and may be considered as one of its tests; and its specific gravity 1·004 to 1·006, the temperature of the air being 86°. Its sediment consists of coagulated albumen and the *debris* of blood-cells. Another test is acidity, and a third is to be observed in the phenomenon that the sediment is dissolved by liquor potassæ, which disengages ammonia.

Another feature in the pathology of yellow fever may be expressed by the fact that the urea of the suppressed urine is eliminated from the system as a volatile salt, by metamorphosis into a carbonate of ammonia, which as such is frequently found in the breath, in the normal black vomit, in combination with an acid, almost always in the stool, and apparently pervading all the tissues of the body. “It would seem as though the mucous membrane of the stomach were called upon to compensate for the defective secreting and eliminating power of the kidneys;

and in those cases where little or none of the matter of black vomit was found in the stomach after death, the lining membrane presented a thickened appearance, with a muco-sanguineous coating" (J. D. MACDONALD in GRANT'S *Annals of Military and Naval Surgery*, p. 135).

Prognosis is declared by Robert Jackson to be treacherous and difficult in the extreme. The data to judge from may be arranged into symptoms which are favourable, and those which are not so. (1.) *The favourable symptoms* are—A slow pulse and moderate temperature of the body, and quiet stomach. Streaks of blood during the stage of black vomit, or after acid elimination has set in, are favourable, if the corpuscles are found entire. If the urinary secretion continue, and the black vomit be scanty from the first, or is afterwards suppressed, the patient may yet survive. Urine simply albuminous is a less serious sign than when it also contains tube-casts. Free, copious urine, no matter how dark or bilious, is the most favourable of any single sign. Prognostics may be derived from the effects of treatment. (2.) *The unfavourable signs* are—The more fiery crimson the tip and edge of the tongue, the more irritable the stomach, the severer the headache, the worse the prognosis of the first stage, and *vice versa*; but a streak of blood in the early vomit indicates much danger from the attack. In the second stage the earlier or more complete the suppression of urine, and the more copious the ejections of black vomit, the more imminent the danger. If the urine be scanty, and loaded with tube-casts, entangled in epithelial and coagulable matter, the light buff-coloured curdy sediment before mentioned indicates a complex lesion of the secreting structure of the kidney. It is the urine symptom in its maximum of severity, and is as fatal as if the suppression had already occurred. Blood-corpuscles in the urine are not to be looked on with apprehension. A faltering of the articulation is a bad prognostic, and a difficulty of protruding the tongue enhances it. The danger of the case is also enhanced by inflammatory complications, and by hypertrophy of the heart. A recent residence in a temperate climate; the *race* or complexion of the individual; the fact of his previously having suffered from an attack, will enter into an estimate of his chances of recovery.

Treatment.—An early attention to first symptoms among the susceptible is of the greatest value in saving human life. The diagnosis of cases in which the attack has been said to have been "aborted" by remedies may be questioned, and such a belief is opposed to the doctrines of sound pathology. Moreover, the "heroic" doses of calomel which were given in such cases, combined with quinine, cannot be too strongly discountenanced, for "they were first recommended on the strength of a crazy hypothesis" alone. The practice is said to have frequently proved successful in Jamaica; but, according to Dr. Davy, it was not attended with beneficial results at Barbadoes, and the American physicians at New Orleans have not found it to answer their expectations in stopping the fever. The large and frequently repeated doses of quinine were often also highly injurious (LAWSON). This discrepancy may, in some measure, be explained by what has been stated at the outset in explaining the pathology of this peculiar fever. It is in cases where the fever is of the periodic, paroxysmal, or paludal form, and not the continuous or true yellow fever, that quinine

may be of use, if the system can be brought under its influence. The ill effects of quinine in specific yellow fever consist in its checking secretion and deranging the circulation within the head. It is an object to keep the bowels freely open, and to get the skin to act freely. The main object of the physician should be to moderate excessive action in any organ, and to endeavour to bring about as complete a crisis as possible about the fifth day, which seems to be the natural period of resolution of the disease. For this purpose nothing is of more importance than to re-establish the secreting function of the colon, and to obtain fœculent evacuations—not mere bilious discharges, but proper dark-brown fœculent stools (LAWSON). Gentle excitement of an extensive portion of the lining membrane of the colon, with frequent copious enemata of a pint and a half of warm water, in which a tablespoonful of common salt has been dissolved, and to which has been added a tablespoonful of olive-oil, or more stimulating enemata, such as of turpentine, deserve a full and careful trial. All the depurative functions must be kept in activity. Turpentine is recommended by Dr. Copland, by Dr. Archibald Smith, and Mr. Laird, of H.M.S. “Medea”—one drachm doses by the mouth, or half-ounce doses as a lavement, several times daily, using it also as an epithem on the abdomen. “When the mucous surfaces,” writes Dr. Blair, “as indicated by the tongue, were denuded of epithelium, the use of *gum water* was decidedly beneficial. It lubricated, defended, and soothed the raw surfaces. The strength was generally three drachms of the purest powdered gum arabic, dissolved in six ounces of cold water, and a tablespoonful of this given every one or two hours. The patient at last gets tired of it; but for thirty-six or forty-eight hours of the most critical period of the disease it is used without dissatisfaction, and then can be substituted by, or alternated with, smoothly and thin-made arrow-root. When the heat of surface was ardent, a *wet sheet* or *blanket* was used for the reduction of temperature by evaporation, with frequently very good effect (and if ice could be obtained, its internal use is well worthy of a trial). But in the later stages of the disease, when the skin was cool or cold, the patient seemed to have an instinctive craving for its reapplication, and frequently asked to be put into it. There would appear to be two causes for this feeling. We find it to exist in cases in which black vomit has been copious, and the associating thirst distressing; also in cases where there has been no black vomit of any consequence, and the breath is highly ammoniacal. In the former class of cases the stomach ceases to be an *absorbing* viscus in anything like the proportion of its secretions and transudations. The skin is therefore employed in reducing the crisis of the blood by the absorption of water, as shipwrecked mariners are said to quench their thirst. But not only does the skin afford an inlet for the imbibition of diluting fluids, but the softening of the cuticle would seem to afford an additional outlet for the noxious elements of the circulation; and it is probably in this direction we must in future look for auxiliary means of relieving the blood of its poisonous, metamorphosed, and effete constituents, the onus of which is now thrown on such vital organs as the stomach and lungs. At one time the heat of the surface was so ardent and persistent that the wet sheet failed to reduce it effectually. The most distressing symptom in yellow fever, both to the patient and the medical attendant, is irritability of the stomach; it is so constantly pre-

sent, and so often uncontrollable, that the knowledge of every available means of checking it is of the utmost importance. The *food* during the course of yellow fever should be of the blandest description—chicken tea, arrow-root, sago, and barley-water constituting the chief articles; and these should be taken in minute quantities at a time when the stomach is at all irritable. This rule applies to drinks of all kinds. The patient is greedy for a large draught of fluids; but by sucking them through a glass tube, or a straw, or a hollow reed of small bore, or by the tea or tablespoonful, they are much more likely to be retained. A cold infusion of oatmeal was found an agreeable drink for Scotch seamen, of which they did not seem to tire. A dislike of sweets was observed among the patients; and when lemonade was asked for, the usual quantity of sugar was objected to, probably from its rendering the liquid too dense for ready absorption by the stomach, and therefore less quenching. *Tea* was found so uniformly to disagree with the patients, and cause vomiting, particularly in the advanced stages, that at length it had to be expunged from the yellow fever dietary. Dilute alcoholic drinks were given freely, and with good effect. When brandy could be obtained pure (tolerably free from acidity and fusel oil), and was well diluted with water, that spirit answered every indication. Sometimes the effervescing wines were relished and retained, but they are very liable to the objections of containing foreign matters and the products of mismanaged fermentation (BLAIR). Four or five minims of chloroform prepares the stomach for the reception and retention of food, by lessening its irritability; and the dose should be repeated a short time before food is again taken, as the effect of the chloroform is transitory (J. D. MACDONALD). On the same principle the administration of *chlorodyne* may be advocated, the composition of which is given by Mr. Squire in his *Companion to the Pharmacopœia*. Lime-water has been also found to have a most beneficial effect in allaying vomiting, and thus enabling the patient to partake of food; and the essence of beef is well adapted for such cases, or beef-tea and arrow-root in equal quantities mixed.

During the course of the disease, *auxiliary treatment* may be required to meet contingent symptoms. This is embraced chiefly in the use of sinapisms and stimulating liniments. Tenderness over the liver may be benefited by them. Dr. Blair writes that when the primary reaction was violent, and the face turgid, and the head symptoms severe, arteriotomy was performed with benefit. In a few such cases, and when the patient was young, strong, and full-blooded, and where the dynamic congestions were so violent that the vessels yielded to the turgescence and impulse, and blood-corpuscles without tube-casts, or even but a haze of albumen, was present in the urine, a vein in the arm was opened, and free bleeding relieved the tension of the vascular system. In such cases convalescence was slow and unsatisfactory, but the immediate results were beneficial. The severe pain in the loins, which is a constant symptom in yellow fever, is generally associated with renal congestion; and sinapisms or stimulating liniments afford great relief to the lumbar pain (MACDONALD). With regard to the administration of opium in any form the rule is, *not to give it when there is suppression, or tendency to suppression, of urine*. Restlessness and sleeplessness are best met by *chlorodyne*,

remembering that the effect of yellow fever on the system is to make it sensitive to narcotics. The congestion of the kidneys about the fourth or fifth day requires watching, so as to diminish the chances of suppression, by reducing congestion and preventing the closure of uriniferous tubes by accumulated epithelium. For this purpose Dr. Lawson recommends cupping, either dry or with the abstraction of blood, and the use of frictions, with stimulating liniments over the loins. These, with warm baths or hot-air baths, deserve a full trial; and small doses of *acetate of ammonia*, with *potash* or *soda*, or their salts in common use, with diaphoretics (so as to act gently on both kidneys and skin), may prove beneficial.

PLAGUE—*Syn.* LEVANTINE, BUBONIC, OR ORIENTAL PLAGUE.

LATIN EQ., *Pestilentia*; FRENCH EQ., *Peste*; GERMAN EQ., *Pest*; ITALIAN EQ., *Peste*.

Definition.—*A specific malignant fever which has prevailed at different times and places epidemically, attended with bubo or swellings of the inguinal and other lymphatic glands; or occasionally with carbuncles, pustules, spots, petechiæ of various colours, distributed in different parts of the body; and severe manifestations of implication of the nervous, sanguineous, and bilious systems.*

Pathology.—(1.) *History.*—Modern medicine restricts the term “plague” to a disease of dreadful severity, and of a peculiar character, which appears to have been first recognised in Egypt and in the neighbouring countries. The remotest period to which we can distinctly trace it is when spreading into other countries, as the plague of Constantinople, which broke out in 544, when Justinian was emperor. This is the first time that the disease, from its course and symptoms, can with certainty be recognised as the plague of more modern times. From that period it has prevailed, at short intervals, in various parts of Europe up till now (1879), and the following account of the disease has been compiled from “*Papers relating to the Modern History and Recent Progress of the Levantine Plague*” presented to Parliament in 1879. Sir Gilbert Blane has calculated there were no less than forty-five epidemics of plague in the seventeenth century. Fourteen of these occurred in Holland, imported, it is supposed, by the Dutch engaged in the Levant trade, about the year 1612; and twelve in England, imported, as has been supposed, from Holland. For an undetermined period it had prevailed in England either as a continuously present disease, or as a series of frequently recurring epidemic invasions. The last epidemic of plague which prevailed in both of these countries was in 1665, the year before the memorable fire of London, after which the plague quickly subsided and died out. This epidemic was termed the “Great Plague,” and spread “with such intolerable infection” that 7,165 persons are said to have died in one week, while in one year no less than 68,596 died in the city of London and its suburbs alone—an immense mortality, considering the then comparatively small amount of population, about half a million. In the year following (1666) the deaths were 1,998; and in the subsequent thirteen years (1666 to 1679) 81 deaths only,

after which no death from plague was recorded in the bills of mortality and in the year 1704 the name of the disease was omitted in the bills, and it has not since had a place in them. For centuries Egypt and the countries lying east of the Mediterranean had been regarded as the birth-place of several diffusions of plague which had spread over Europe. In these countries the disease lingered longest, and was latest to disappear during that gradual decrease of the area of prevalence, which, observed in this country after the great outbreak of 1665, came to an end in Egypt, with the apparent extinction of the disease in 1844.

The phenomena of the diffusion of plague in Europe since the end of the eighteenth century indicate a gradual diminution of the area within which the malady could retain its activity; nevertheless the disease apparently at no time has presented any amelioration of its accustomed virulence. In 1812-14, plague became widely diffused in the Levant, in the districts bordering on the lower Danube; in Transylvania, Asia Minor, and America; and also in Northern Africa, west of Egypt. This diffusion continued in one district or another within the area of dispersion throughout the next twenty years; but without extension into Central, Northern, or Western Europe. The diffusion of the disease, which again commenced in 1834-35, was chiefly confined to the Turkish dominions in Europe and Asia, and to Egypt, to which district in Africa it was limited, extending also to the towns and districts of Wallachia bordering the Danube, and also into Greece; but no other countries in Europe contiguous to Turkey were affected. Five years after the commencement of this diffusion (1839) plague disappeared from Europe, but again became actively diffusive in Egypt, Syria, and Palestine and parts of Asia Minor (1840-41). With the termination of this diffusion plague vanished from the East in 1843-44, to reappear nine years afterwards (1853) in the highlands of Western Arabia; and fourteen years afterwards (1858) in the basin of the Mediterranean, within the regency of Tripoli, in the province of Bengazi, North Africa. There it first appeared in the middle of April, in an Arab encampment consisting of thirty huts pitched at the time eight hours distant from the town of Bengazi; and by the beginning of May the disease appeared in the town itself; and afterwards spread throughout four of the five districts into which the province is divided, affecting both the nomadic and the fixed populations. The disease had disappeared by June, 1859; the mortality among the sick was about 40 per cent., and the total deaths about 4,000. Four years afterwards (1863) it appeared in the district of Maku, Persian Kurdistan, in the extreme north-west of Persia; and after another four years (1867) the malady appeared in the Lower Euphrates among the Arabs inhabiting the Hindieh Marshes, on the west bank of the river, in close proximity to the ruins of ancient Babylon, covering a district about 60 miles in length (N. to S.) and 20 miles in breadth (E. to W.), on the eastern border of the great Syrian desert. Utter desolation characterises the district. The marshes are inhabited by many communities of Arabs, who dwell in villages erected upon the islets within the marshes or their outskirts. These villages, for the most part, are formed of huts, or rather of "dug-outs," hollowed in the wet soil, roofed with reeds, covered with matting, or thickly plastered with mud. The wretchedness of these

dwelling is augmented by the indescribably filthy habits of the occupiers in persons and surroundings. The Hindieh Canal opens into the northern extremity of these marshes.

Here in 1867, after an excessive flood of the Euphrates and inundation of the marshes, plague, which had been absent from Mesopotamia for thirty-three years, reappeared. The inhabitants of five villages only were affected by the disease—a population not exceeding 1,000, of whom 300 died. To the north-west of these villages is Kerbela, and to the south is Medjef, the burial-place of Ali, while Kerbela is the burial-place of his two sons, sacred to the Ghiah Mohammedans. They form a centre of pilgrimage second only in importance to Mecca. To both cities annually resort many thousands of pilgrims from Persia, the Mohammedan districts of Caucasia, and from Hindostan, carrying with them great numbers of corpses in all stages of decomposition, for burial in the sacred soil. After this occurrence of plague on the Lower Euphrates, nothing more was heard of the disease till 1871, when it appeared in Persian Kurdistan, and was investigated by Drs. Cartaldi, Paduan, Wortabet, and Schlimmer—Ottoman sanitary physicians resident at Teheran, Bagdad, and Suleimanieh. Again, in December of 1873 and spring of 1874, the disease broke out in three localities widely separated from each other, among the Affij Arabs, occupying the “mighty marsh” on the east bank of the River Euphrates, extending to its junction with the Tigris, nearly opposite the scene of the outbreak of 1867 in the Hindieh Marshes. The habitations, means, and food, as already described, equally apply to the Affij Arabs. About the same time the disease appeared in North Africa among certain tribes of nomadic Arabs then encamped on the Cyrenaic plateau, in the province of Bengazi (Tripoli), within the area of the outbreak of 1858; and a little later it broke out in the Assyr country—Western Arabia. Plague was first observed at the end of December, 1873, in the village of Dagarra, and the corpses of those who died earliest were carried to Medjef for burial. The disease extended to others of the Affij villages, and afterwards spread irregularly in the district, on the west bank of the river intervening between Hillah on the north, Kerbela and Medjef on the west, overlapping the scene of the outbreak of 1867. The population of this infected district is said to have been 80,700, and the deaths from plague during April, May, and June of 1874, to have been 4,000. Of these deaths 2,000 took place in Dagarra and the surrounding villages of the Affij tribe, of which the population is estimated at 35,000. At the beginning of 1876 (third week of February), plague again became active in Mesopotamia, but 50 deaths had already taken place in January. The malady appears to have shown itself in various parts of the tract of country along that part of the course of the River Euphrates where it had been chiefly active the previous year. The disease also appeared at Hillah and Bagdad, and in the villages of the district lying between these towns. The deaths from plague recorded at Hillah from January 1 till June 22 inclusive, numbered 1,007 out of a population of 15,000; and those at Bagdad, from February till June, numbered 3,639 out of a population of 60,000; while the minimum mortality from plague throughout the province, including the nomadic tribes, is estimated at 20,000 during the outbreak of 1875-76. In July of 1876 all active manifestations of plague in Mesopotamia had

ceased, and continued to remain dormant till October, when it again broke out at a point higher than it had been before observed on the left bank of the Euphrates—namely, fifty miles or so above Musseib. On the 15th of January, 1877, it appeared among an Arab tribe in the district of Azzie, south of Bagdad, on the right bank of the Tigris; and on the 17th of January two cases occurred in Bagdad, one of them fatal; and by the middle of March plague was prevalent in the city, and steadily extending—the deaths up to the 16th of June being 1,672—a number less by 939 than during the like period of 1876. In March 1877, plague broke out in Resht (population 20,000), the capital of the province of Ghilan, near the N.W. corner of the Caspian Sea. It had been free from disease since 1830, when half the population was carried off by the plague, and the prosperity of the place received a check from which it never recovered.* The malady on this occasion showed itself in March, 1877, and cases were recorded up to January, 1878; but after 4th and 5th May, 1878, no information has been received of the existence of plague in any part of Persia.

With regard to India, a rapidly fatal disease, known as *Māhāmari* (locally *gola* or *phutkia*—e.g., *bubo*), became prevalent in the mountainous district of Kumaun, on the southern slopes of the Himalayas. Forty-one of the hill villages in this district suffered from the malady in 1876-77, and in forty of these villages 291 persons were attacked, of whom 277 died—the malady being identical with true Levantine or bubonic plague; and the same disease as the Pali plague of Rajputana and contiguous provinces to the westward. It was first noted in Cutch in 1815, prevailing for six years (1815 to 1820), and described as the Bombay Plague of 1818 (Dr. JAMES RANKEN, Calcutta, 1838). In 1823 it again appeared in the district of Garhwāl, lying to the north of and contiguous to Kumaun; where, in 1834-35, it was again prevalent, and more or less so up till 1877; so that the disease seems to be endemic among the hill villages of the district, and in other parts of the same vast mountain system.

Dr. Russel states that at Aleppo he met with twenty-eight cases of re-infection, or 1 in 157; and Clot Bey states that he and his colleagues saw many individuals perish of plague in 1834-35 who had formerly survived an attack of the disease.

(2.) *Conditions under which Plague has been developed and propagated.*—The plague, and the specific poison which it generates, as shown from the foregoing history, seem to have a very limited geographical range; and it

*“Very few persons in Resht are old enough to recollect the days when this town was attacked by real plague, towards the year 1832. In a few weeks half the population of a very flourishing town, then numbering upwards of 40,000 inhabitants, had been laid low, and the remainder had fled in all directions. Resht for a while became a charnel house, a city of the dead. No living creature was to be seen in it, and those who had been abandoned by their friends when stricken by the disease died from sheer want. When the people returned to their homes the disease had spent itself, and the population was reduced to 8,000 inhabitants. The putrefying remains of the dead were buried in the homes in which they were decaying. Property changed hands in all manner of ways. Some who had enjoyed wealth were reduced to the threshold of poverty, their goods and chattels having been appropriated by their neighbours; while others had suddenly merged into unexpected opulence by the extinction of their richer relatives. Such were the effects of plague when it last appeared at Resht.”—Mr. CONSUL H. A. CHURCHILL, C.B., Resht, May 24, 1877.

is not less certain that the disease is often circumscribed within a very small space of country. All that we can affirm of the poison of the plague is, that it is at all times endemic in Egypt, along with the cognate diseases of "carbuncle," "anthrax," or "boil," already referred to, and that about every five or six years it becomes epidemic. It also appears to be, to a certain extent, influenced by season, not spreading in any very sensible degree till December, and attaining its greatest height in June, when it rapidly declines, till winter and spring. The period of the year, however, at which the plague prevails differs in some degree in different countries; but the total duration of the disease in any country to which it is not native appears to be inconsiderable, unless kept up by a fresh importation.

The appearance of the plague in the province of Bengazi had been preceded by four years of drought and consequent failure of crops; and each successive year the miseries arising therefrom had increased. The greater part of the flocks and herds were destroyed from want of food; and the mortality among horned cattle was augmented by a fatal epizootic which attacked them. Plague appeared when the sufferings of the population from famine was greatest. Moreover, the lanes and alleys of the Kurdish villages are full of filth—the usual characteristic of them—although the houses are not crowded together, nor do the walls of one abut on those of another. Whatever is most afflicting in poverty, and most revolting in filthiness, is accumulated, as if designedly, round these infected dens (foul hovels, miserable burrows), in the interior of which live (or rather vegetate) fifty to sixty individuals—men, women, and children. The cultivation of the ground furnishes insufficient nourishment. In other instances the malady has appeared during the winter; after a fall of snow so deep that the villages, being shut in, were "snowed up" in the midst of indescribable filth, plague became active to the extinction of many villages. The outbreak in the Bengazi province in 1874 had been preceded by three years' privation, arising from failure of crops in successive years consequent on persistent drought. In the winter months of 1873-74 the famine had become so severe that persons might be seen hunting for undigested grain in the dung voided by camels. The winter months of 1873-74 and spring of 1874 had been unusually wet, and had converted some of the favourite camping grounds of the nomadic races into swamps, adding to the misery of the starving population, but securing the summer crops. H. M. Vice-Consul, Mr. F. Henderson, at Bengazi, states that in April of that year he found the greatest misery, the wretched inhabitants subsisting on wild artichokes, and the villages almost unapproachable, owing to the continuous rains having converted the plains into a vast marsh. The famine also, which had followed on the long continued drought, had decimated the population of the province and the Bedouins of the plain; and it was no uncommon thing to find in the morning three or four corpses in the streets of Bengazi of persons who had perished miserably over night (Letter to *Times*, 19th August, 1875). At such a crisis of misery plague appeared in a diffusive form, spreading from encampment to encampment, and to certain of the villages and monasteries in the highlands, but not to the towns on the littoral, nor to the tribes on the low-lying plains.

Two or three months previous to the full manifestation of plague in

Mesopotamia, *glandular swellings, free from fever*, prevailed. They showed themselves in the groin, arm-pit, or neck, and were not accompanied by other symptoms. They began to appear amongst the inhabitants about the end of autumn, and continued through the winter; but towards the end of it plague broke out, and reached its acme of intensity in the spring, and died out suddenly in the summer season, when great heat declared itself (113° to 122° Fahr.). During the prevalence of plague the thermometer varied from 14° to 86° Fahr., when it reached its maximum intensity; and as temperature increased from 86° to 113° Fahr. the epidemic began to diminish; and at 113° Fahr. it ceased abruptly. On the cessation of plague the apyretic glandular swellings reappeared, and continued to manifest themselves about two months longer. A *period of latency* after exposure to infection varied from five to six days, with an ultimate limit of eight days (Dr. ARNAUD). As to contagion, no great risk is incurred in touching infected persons, provided exposure for any lengthened period to the atmosphere of the apartment occupied by him is avoided. Those persons who lived in the same house with a plague-smitten patient, but who avoided touching him or his clothes, generally did get the plague; while those who lived in houses exempt from the malady, but who visited plague patients and handled them freely, without remaining long in the same room with them, hardly ever caught the disease. When plague entered a house it seldom spared its inmates, and often carried them all off within a week. And, while the Mohammedans, disregarding all sanitary precautions, lived with and handled plague patients, the Jews and Christians, on the contrary, firmly believing in its contagious properties, very seldom *touched* them; yet all of them equally caught the disease, if they *lived in the same house with the plague patient*, but were safe if they *quitted the house the moment* a case of plague occurred in it. *With one exception*, none of the physicians, surgeons, or assistants who daily attended on plague patients *caught* that malady; and Dr. Cabiadis himself handled nearly 2,000 plague-smitten patients without any inconvenience. The exception was that of an assistant who actually did die of the plague. It is still a problem whether the *specific contagium* of plague is exhaled from the patient's respiratory organs, or his cutaneous surface, or some other emunctory. The disease seems capable of giving off its contagia before any swellings or boils have suppurated. The atmosphere that surrounds a patient is the true medium of transmission; and numerous instances show that the clothes and bedding of plague-smitten patients communicate the disease to sound persons living beyond the sphere of the epidemic outbreak. Wool, cotton, flax, and hemp thus become the bearers of *fomites*. Beyond what has been stated, the origin of plague, and the causes which bring it about, are utterly unknown; but the most palpable predisposing condition for its propagation is undoubtedly *poverty*, which implies *privation*. No other malady shows the influence of this factor in so striking a degree. There are also especially three facts which subvert the hypothesis that plague is engendered by "*malaria poison*." These are—(1.) No intermission has ever been observed in the symptoms of plague—not even in those intercurrent phenomena which arise from the complications of the nervous, circulating, and assimilative organs. (2.) There is no instance in which the plague

has been "cut short," or been benefited by the administration of the *sulphate of quinine*. (3.) The expression of the countenance and the general aspect of a patient suffering from an attack of *pernicious fever* and those of one suffering from the plague are very striking. The pulse and temperature in plague may be normal, while the patient may have but a few hours to live. On the other hand, great danger seems to threaten a patient suffering from pernicious fever.

(3.) *Morbid Anatomy*.—On removing the calvarium the sinuses were found filled with black blood, the arachnoid veins hyperæmic, and the arachnoid cavity infiltrated with serum, and occasionally with a trifling effusion of black blood. The substance of the brain was generally less consistent than in health, and sprinkled with more bloody spots than usual. The bronchial membrane appeared red, although during life the patient had presented no catarrhal symptoms. The pericardium frequently contained a reddish serosity. The serous membrane covering the heart was often extensively affected with petechiæ. The heart, distended with blood, was almost always enlarged from a third to a half greater than its natural size, its tissue pale and sometimes softened. In acute cases the stomach and small intestines were softened, and presented similar petechial appearances. The liver was almost always larger than natural, and loaded with blood, while petechial spots were seen at its surface. The gall-bladder was the seat of petechiæ. The spleen was always twice its natural size, or even more, but was rarely the seat of hæmorrhagic effusion. It was softened, and deep in colour. The kidneys were often found immersed in a hæmorrhagic effusion into the surrounding tissue. They were loaded with blood, and the pelvis filled with clots. The ureters occasionally contained blood, and sometimes the lumbar glands were so enlarged as to press upon them, and to account for the suppression of urine. The bladder occasionally presented petechiæ, and occasionally the urine was mixed with blood. Every dissection showed that buboes, wherever seated, always resulted from enlarged lymphatic ganglia, varying in size from an almond to a goose's egg. The least altered were hard and injected. In a more advanced stage some of these glands were without any change of colour, others as richly coloured as lees of wine, and either wholly or partially softened or putrescent. Sometimes these glands became agglomerated, forming masses which weighed two pounds or more, and around these agglomerations a hæmorrhagic effusion extended into the areolar tissue. The cervical glands often became so enlarged as to form a sort of chaplet, uniting with those of the axilla and of the mediastinum. The axillary glands, again, communicated with the cervical, and with those which surrounded the bronchi. Those in the groin connected themselves in the same manner with those of the abdomen, and these might be traced without interruption through the crural arch into the pelvis and along the vertebral column. It was especially among these latter that sanguineous effusion was found in the subperitoneal tissue. The mesenteric glands were often so numerous that the whole of the mesentery seemed covered with them, but they seldom exceeded an almond in size.

The Symptoms that characterise plague are of two orders, local and general. In severe cases the general symptoms precede the local manifestations. The patient is suddenly seized with fever, which, in some

instances, is ushered in by a prolonged shivering fit, the pulse beats 130, or even more, and the temperature of the body rises to above 107.6° Fahr. The fever lasts from twenty-four to thirty hours. The eyes become red and turbid. The tongue appears swollen and fissured, sometimes black, or white, as if cotton were upon it. He may have to be spoken to twice or three time before he can understand the question. He will then probably sit up before answering, when his head will roll from side to side, and his face is flushed like that of a drunken man. Sometimes no answer is to be got to a question however often it may be repeated, or only a moan. Sordes are invariably about the teeth and gums. Thirst is intense, and if the patient can speak he calls for water. Pain of a stabbing or burning character may be complained of in the epigastric region. Vomiting is generally bilious, sometimes bloody, and blood may at the same time pass by vagina or rectum. During the fever stage the breathing is hurried, the pulse very rapid, and the body temperature about 100° while that of the air is 93° Fahr. in the shade. The urine is generally natural, abundant, and pale coloured. Usually there is obstinate constipation; and diarrhoea, when it occurs, is considered a favourable sign. In some cases the febrile condition is hardly appreciable, while, on the other hand, alarming complications indicate the severity of the attack by perturbations connected with the nervous centres, such as convulsive tremor, drowsiness, coma, delirium; or with the circulating system, such as epistaxis, hæmatemesis, hæmoptysis, sanguineous diarrhoea, menorrhagia; or with the assimilative organs, such as painful tumefaction of the liver, bilious vomiting, bilious diarrhoea, jaundice, and the like. These phenomena appeared to be dependent on some *periodic impulse*, because at times the bilio-gastric manifestations prevailed; at others the hæmorrhagic, and at others the nervous. The nervous agitation which sometimes ushers in an attack of plague is a very remarkable symptom, and one that Dr. Cabiadis had never met with before. He describes it as a *prolonged regular shake*, lasting from six hours to three days. While the patient is suffering from it he does not complain of cold, nor does the thermometer indicate a lowered temperature. The pulse is small, short, and quick, and the temperature of the body remains in nearly its normal state. This tremor is always followed by profound coma, during which the patient rapidly sinks. In some instances of plague Dr. Cabiadis has seen death supervene within a few hours of the attack, and before any of the characteristic indications of the disease, such as buboes and carbuncles, had shown themselves. These severe cases were mostly met with at the *commencement* and at the *termination* of an epidemic outbreak, in this respect resembling cholera epidemics. They were observed in the plague of 1876 at Hillah, as well as in that of 1877 at Bagdad; and in these instances the glandular swellings either did not appear at all, or were very small and never suppurated; the patient, moreover, seldom lived more than three days. The cutaneous surface, in some cases, was so filled with *petechiæ* that, when death supervened the skin assumed a dark livid hue, giving the corpse a *blackened appearance*, and so characteristic of the malady that it might even to this day be called the *black death*. In the usual course of plague, however, the local manifestations precede the

general symptoms. The patient first complains of pain in the groin, the armpit, or the neck, and on examining the part a swelling is found there, which rapidly increases and usually suppurates by the seventh or eighth day; at the same time carbuncles may show themselves, and in bad cases petechiæ. In these instances the fever runs high and in accordance with the gravity of the case. In slight attacks of plague the local manifestations are seldom accompanied with *fever*, and you often meet such patients walking about the streets with two or three suppurating buboes upon them. These cases are hardly ever fatal.

Several symptoms are usually met with which are common to all febrile complaints. Those have been noticed which may be considered as pathognomonic of the plague. Headache, thirst, constipated bowels, lassitude, and aches in the limbs exist more or less in all fevers. The appearance of the tongue in cases of plague is not in general much altered; usually, it is red at the tip and edges, and sometimes dry. A comatose state is much more frequently met with than a delirious one. Diarrhœa is always either sanguineous or bilious, but never *serous*. The buboes of plague seldom mature till the fever is on the decline, which rarely happens till the eighth or ninth day; nor are they generally ripe for opening till between the fifteenth and twenty-seventh day. The glands which chiefly enlarge are those of the groin, next those of the axilla, after that those of the neck, and sometimes the gland above the bend of the arm. The largest gland may be the size of a small orange. The carbuncle is by no means of constant occurrence, Dr. Russel having found it only in 490 cases out of 2,700, and Dr. Cabiadis 36 in 1,826 cases of plague at Hillah. It appears more commonly in the middle or towards the decline of the disease. Hardly any external part is free from them, not even the penis; and in one instance a carbuncle formed in the throat, which was fatal. They occur more particularly on the limbs, and more especially on the legs. In some cases they form on the cheek or lips, and, by the tumefaction they cause, give to the face a hideous aspect; in others the whole of one side of the jaw has been laid bare; while in others they have formed on the eyebrow and on the eyelid, and partly destroyed the eye. Clot Bey, however, observed that they never formed on the scalp, the palms of the hands, or on the soles of the feet. There are three different varieties of carbuncle all commencing the same way, by a small red pimple, which increases, and in the centre of which a vesicle forms, containing first a yellow and afterwards a blackish serum. In the more benign the vesicle bursts, and dries up in three or four days from its first formation, the epidermis alone having been infected. The second variety involves the whole thickness of the skin, as well as portions of the cellular tissue, which is moderately tumefied, and surrounded by a dark-red areola. The gangrene in this form is circumscribed, and there results an eschar from one to two inches in diameter, which is detached by suppuration, leaving an ulcer with a sharp perpendicular edge. In the severe forms the redness and tumefaction cover a large space, and the gangrene rapidly involves the skin, the cellular tissue, and sometimes even the bones. It has been observed that the malignity of the carbuncle is in the direct ratio of the severity of the disease, but the mere existence of carbuncle is not of

unfavourable augury. Their number is various, sometimes only one, at others ten or twelve. When there are several, they often form in succession. These tumors are very painful; and Aubert mentions one, seated on the back of an Arab soldier, four inches in diameter. Petechiæ are observed in some seasons and not in others. They present different shades of colour, according to the intensity of the disease—rose colour, violet colour, or black. Aubert considered their appearance an almost certain sign of death.

Diagnosis.—No other malady presents an assemblage of symptoms so well-marked and so significant as plague. No other idiopathic fever, attacking a multitude of persons at the same time, is characterised by glandular swellings, by carbuncles, and by those severe manifestations of the nervous, sanguineous, and bilious systems, which declare themselves in an attack of plague. At the commencement, however, it is often not to be distinguished from ague. In both the attack is sudden; but in plague before fever sets in, and before pains are complained of as in ague, the plague patient becomes distracted, tosses about in evident fear of something he cannot describe, and if he is absent from his house runs home like a maniac and throws himself on his bed (SURGEON-MAJOR COLVILL).

Prognosis.—As a rule, an attack of plague lasts from a few hours to four weeks. Out of 534 deaths 311 occurred within the first three days. After this time the risk of death gradually diminished as the days went on; so that the natives were pretty correct when they said a patient may consider himself safe after the seventh day; but Surgeon-Major Colvill thinks 60 per cent. of deaths to attacks in the first three days is much under the mark. About one-third of the attacks end fatally. At Hillah, in 1876, the proportion of deaths to attacks was 52·6 per cent. (CABIADIS); at Bagdad the proportion was 55·7 (COLVILL). With regard to age, the greatest number of attacks occurred between the ages of 10 and 19 years; next between 20 and 29; then 30 to 39, and next from 2 months to 9 years. The fœtus has been in some cases attacked; and in one case of premature birth, which Surgeon-Major Colvill saw at Bagdad, where there were twins—one child was dead, with the glands in the left side of the neck swollen. The other child is alive and well. Hæmorrhagic phenomena are unfavourable. Petechiæ are almost without exception to be considered as fatal signs; they appear generally only a few hours before death. Severe stabbing pains in the glands, which begin to enlarge before fever sets in, are also cases quickly fatal. Carbuncles are an unfavourable sign.

Treatment.—It is to be regretted that recent experience has not in any degree advanced the successful treatment of the plague. In the beginning of the epidemy, when the morbid cause acts with a rapidity so great that some hours are sufficient to compromise the life of the patient, any treatment is powerless to arrest the course of the disease. When, however, the malignancy of the disease abates we may hope for the recovery of patients (CLOT BEY). Looking, however, to the pathology of the disease, and regarding it as bearing some resemblance to malignant *typhus fever*, the principles of the general treatment of plague ought to be similar to those laid down in the account of that disease. We have

no sufficient evidence to prove that plague may be carried beyond those geographical limits where it is epidemic. Quarantine establishments to prevent the transmission of such epidemic diseases are now not only unwarrantable, but they are vexatious interruptions to mercantile enterprise. It is now more than 154 years since any part of Western Europe has suffered from plague; but till a comparatively recent period, more or less of the disease had been habituate or frequent in the Levant, and though occasions have been very numerous when arrivals from the Levant in this country must have been more full of danger than those now represented, the experiments thus performed on the public health of England have in no case led to any injurious result. I am informed by a medical friend who visited Malta in 1861, that a curious instance of the wavering nature of opinion regarding the efficacy of quarantine was afforded during the last Bengazi plague. The Maltese—the most sensitive people formerly on the subject—absolutely declined to put any quarantine on arrivals from Bengazi, and trade went on as usual, until the Austrians intimated that, unless Malta put Bengazi in quarantine, Trieste would put Malta in quarantine; and the poor merchants were obliged to submit. There is little risk of plague being imported now, because of the great sanitary improvements which have taken place; and it is to be hoped that as sanitary measures are developed the barbarism of quarantine will entirely disappear; except, perhaps, where the intelligence of the people does not go beyond that of the Russian Governor of Eupatoria, who requested that the allied armies of France and England might go into quarantine when they landed in the Crimea in 1854! (KINGLAKE.)

AGUE—*Syn.*, INTERMITTENT FEVER.

LATIN EQ., *Febris Intermittens*; FRENCH EQ., *Fèvre Intermittente*; GERMAN EQ., *Kaltes Fieber*—*Syn.*, *Intermittens*; ITALIAN EQ., *Febbre Intermittente*.

Definition.—*Febrile phenomena occurring in paroxysms, which observe a certain regular succession, characterised by unnatural coolness, unnatural heat, and unnatural cutaneous discharge, which prove a temporary crisis, ushering in a remission. These phenomena continue to be developed in an uninterrupted series or succession of paroxysms, more or less regular, which pass into each other by insensible steps, so long as the ague lasts.*

The Pathology of malaria and of malarious fevers has been already fully considered at page 419, Chap. VI.

Symptoms.—Ague may be sudden in its attack, and without previous illness. More commonly it is preceded by general indisposition, headache, weariness, pain in the limbs, thirst, loss of appetite, white tongue, frequent pulse, high-coloured urine, and dark-coloured discharge from the bowels. These *prodromes* are accompanied with well-marked exacerbations and remissions of fever, displaying a periodic tendency. After this feverish state has lasted from four days to a fortnight, a severe rigor occurs and ague is manifested—the attack being commonly called a “fit of the ague,” the phenomena of which are as follow:—The paroxysm like the disease may

be of sudden invasion; but in whichever way the cold stage begins, the patient experiences first a sensation of coldness of the extremities, then of the back, and lastly, of the whole body; at the same time the nails turn blue and the features shrink, becoming pale and sharp. If the case be severe the whole body shrivels up, turns purple, and the surface of the skin assumes that rough condition popularly named "goose-skinned." The coldness increasing, the motor nerves of the fifth pair are affected, and the teeth begin to chatter. This tremor extends to every muscle, till the whole body shakes with the rigors. Cough, dyspnoea, and oppression of the præcordia now occur, with a painful sensation round the temples and down the back. The patient suffers from nausea and vomiting, the latter symptom being speedily followed by the hot stage. When the cold stage has lasted a period varying perhaps from half an hour to two hours and a half, a reaction takes place, accompanied by partial warmth or flushings. These extend, and at length the whole body rapidly acquires a heat of from 105° to 107° Fahr. As the heat returns, so also does the colour, and the body, especially the face, becomes preternaturally swollen and red. The hot stage being formed, the heart and arteries beat with unusual violence. There is headache, with a frequent full pulse, and all the distressing symptoms of continued fever are present. The mean duration of this stage is from three to eight hours. At its close a gentle moisture breaks out, first on the forehead, and thence extends till the patient lies in a general sweat, sometimes so profuse as to soak the bed and linen as completely as if they had been dipped in water. After the sweat has continued to flow for some time, the fever gradually abates, a state of apyrexia ensues, the paroxysm is terminated, and (a sense of exhaustion excepted) the patient feels restored to health. Sometimes, however, he continues pale, debilitated, and incapable of exertion till, on the recurrence of the paroxysm, the symptoms just described are repeated. Upon the approach of the attack the pulse is slow and feeble, but as the sense of coldness increases it becomes small, rapid, and irregular. When the hot stage forms, the pulse becomes full and strong, and on the sweat breaking out it again becomes soft, less rapid, and at length natural. The tongue in mild forms of the disease is clean in the cold stage, white in the hot stage, and again cleans after the sweat has flowed. In severe cases the tongue is white during all the stages, and also during the apyrexia; while in the worst cases the tongue is brown in all the stages. Excepting some unusual instances, attended throughout with diarrhoea, the patient seldom passes a stool till towards the close of the paroxysm, when it is generally a loose one. The febrile paroxysm, or "fit of the ague," has three stages—a cold stage, a hot stage, and a sweating stage. These three stages are not necessarily of an equal duration, but vary greatly in different cases. The duration of the cold stage is from a few minutes to five or six hours; and in general, if the case be severe, the shorter the cold stage the longer the hot stage. The hot stage may last from half an hour to any period less than twenty-four hours. The sweating stage is generally shorter than either of the former, and sometimes does not exist at all. The rule, however, is, that the *quotidian* has the shortest cold stage and the longest hot stage; the *tertian* a longer cold stage and a shorter hot stage than the *quotidian*; while the

quartan has the longest cold stage and the shortest hot stage of all the varieties. An “*irregular*” variety is also recognised, which takes the form of “*Brow ague*.”

The paroxysm of intermittent fever, of whatever description, is conventionally considered to terminate within twenty-four hours. The duration, however, varies in different types. These types or varieties have been named *quotidian*, *tertian*, and *quartan*; and of these there are sub-varieties—namely, *double tertian* and *double quartan*. The mean length of a *quotidian* paroxysm is about sixteen hours, that of a *tertian* ten hours, and that of a *quartan* six hours.

The varieties of intermittent fever are also distinguished from each other by the interval of time which elapses between each *paroxysm*. For instance, when the paroxysm returns every twenty-four hours it is termed a *quotidian*, when every forty-eight hours a *tertian*, and when every seventy-two hours a *quartan*; and these primary types have been extended by early writers to every period comprised within a mensual or bimensual period. Of these primary types it is believed that in Europe the *tertian* is by far the most common type, then the *quartan*, and lastly the *quotidian* (WATSON, COPLAND, CHRISTISON). In the French army in occupation of a portion of the northern shores of Africa, *quotidian* was most common, next *tertian*, and last *quartan*. In the Peninsular War the *quotidian* was likewise the prevailing type; at one time in the proportion of 16 to 1 of any other type. In the West Indies the *tertian* and the *quartan* are only about one-twelfth of the whole number of intermittents treated, the rest being *quotidians*. At Prome, in Burmah, 83·5 per cent. were *quotidian*, and 1·6 per cent. were *tertian* (MURCHISON). The results given concerning the Madras Medical Service are very similar (WARING).

Most authors have stated that the accession of the *quotidian* paroxysm occurs early in the morning, that of the *tertian* about noon, and that of the *quartan* in the afternoon, between three and five o'clock. But to this law there are many exceptions. The greater number of *quotidians* occurred from midnight to mid-day, and fewest from mid-day to midnight in the French army in Africa. This result is corroborated by Dr. Murchison's observations at Prome, in Burmah. In 76 per cent. the paroxysm commenced between midnight and noon; and in 24 per cent. between noon and midnight. The most frequent hours of attack were 9, 10, and 11 A.M.; and in 65 per cent. of the cases the paroxysms commenced between 8 A.M. and noon. Of 730 *tertians*, 550 occurred from midnight to mid-day, and 180 from mid-day to midnight; out of 26 *quartans*, 13 were seized from mid-day to midnight, and 13 from midnight to mid-day. As the most general conclusion, the paroxysm returned in a great majority of the *quotidian* cases from ten to twelve o'clock, and in the *tertian* from nine to twelve o'clock.

The Temperature in Cases of Intermittent Fever.—The paroxysm of fever, notwithstanding the subjective sensation of chilliness, is invariably indicated by a decided, sudden, and rapid rise of temperature. In this respect it resembles the accession of *febricula*; but while the latter requires only from eighteen to twenty-four hours from the commencement of the rise of temperature to the end of the defervescence, in perfectly normal cases of *intermittent fever* there is a whole day free of fever between every two days

of the paroxysm. All the types of the fever present this characteristic peculiarity of a sudden and speedy rise of temperature to a high degree (mostly up to 105° or 106.3° Fahr.); and of an equally rapid and complete defervescence, till the period of another fever paroxysm comes about. This comportment as to temperature secures correctness of diagnosis in cases which may be obscure or ambiguous. The annexed diagrams represent variations of temperature in cases of malarious fever.

- (1.) TYPICAL RANGE OF TEMPERATURE IN A CASE OF INTERMITTENT FEVER OF QUOTIDIAN TYPE. THE RECORDS INDICATE THE HIGHEST AND LOWEST TEMPERATURES DAILY (Wunderlich).

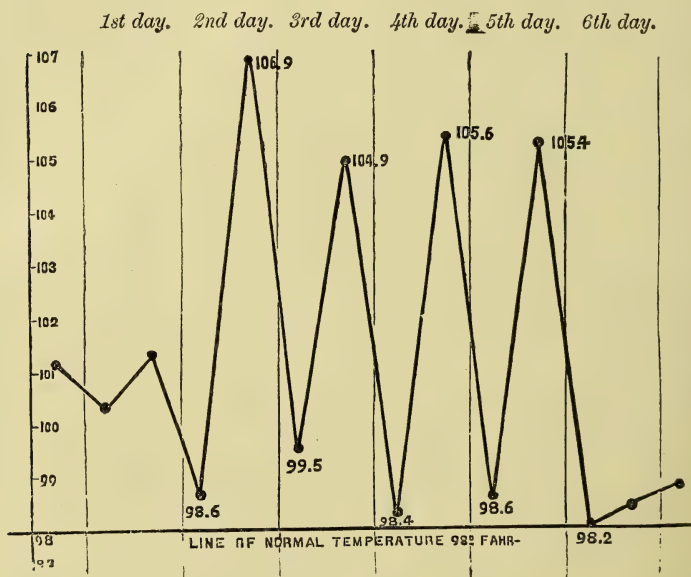


Fig. 89.

In a paroxysm of intermittent fever much may be learned as to the relations of the excretions to temperature, and especially those of the urine, by observing the changes of temperature in very short spaces of time; for example, every fifteen, or even every five minutes (MICHAEL, JONES, RINGER). The rise of temperature is found to begin with, or even to precede, the sensation of chilliness. It takes place at first slowly; and gradually, by about the middle of the period of chilliness, the rise becomes greatly accelerated, lasts through the period of the sensation of great heat, and may even extend into the sweating stage. At the commencement of the sweating stage small vacillations occur, and continue for a short time; and when the sweating has fairly set in, the decrease of temperature begins, and progresses steadily, without any temporary rise, and with great regularity, decreasing at the rate of $.2^{\circ}$ Fahr. (or more) every five to fifteen minutes, till it has arrived, after several hours, at the normal heat.

Condition of the Urine.—The observations made on the condition of the urine are divisible into two series:—(1.) The condition of the urine during the fit, as compared with the urine of a non-febrile period; (2.) The condition of the urine of twenty-four hours during a fever day, as compared with the twenty-four hours' urine of a non-fever day (PARKES, l. c., p. 235). During the fit and the apyretic period the water of the urine is increased in amount during the cold and hot stages: it is most abundant at the ter-

(2.) RANGE OF TEMPERATURE IN A CASE OF INTERMITTENT FEVER OF TERTIAN TYPE.
THE RECORDS INDICATE THE HIGHEST AND LOWEST TEMPERATURES EACH DAY
(Wunderlich).

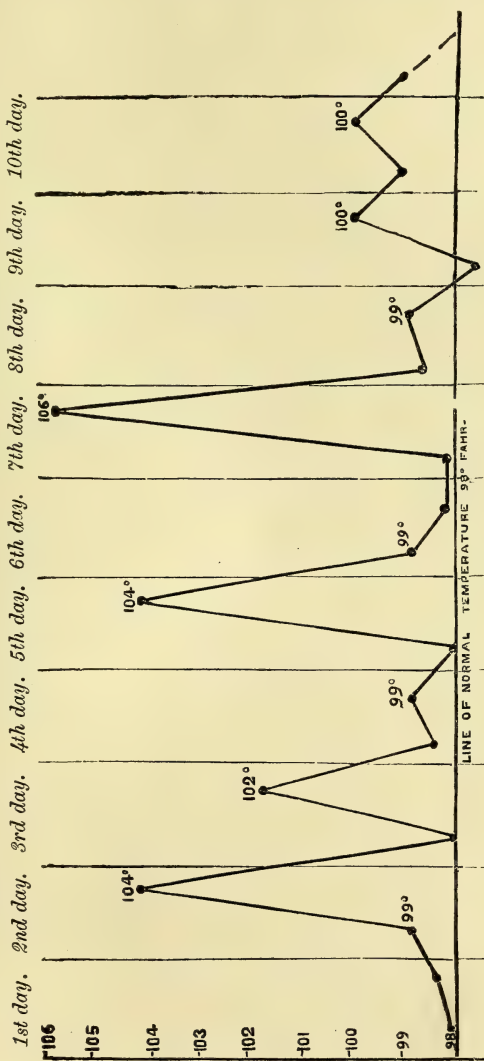


Fig. 90.

mination of the cold or commencement of the hot stage. It decreases during the latter part of the hot stage slowly; rapidly during the sweating stage. The amount of increase is variable; it stands in no relation to the

quantity of fluid drank, and may be great when this is small (RINGER). The amount of urea excreted by a person with ague, not actually suffering from a fit, is less than in health; but directly the fit commences—that is, at the very first moment of elevation of temperature, or even for some time before this, the urea suddenly increases—an increase which lasts during the cold and hot stages, and then sinks, sometimes gradually, sometimes suddenly, through the sweating stage, or into the commencement of the intermission. It then falls below the healthy average. The amount of the increase is variable, and the type of the fever has no influence upon it; but there seems to be a very close connection between the temperature and the amount of urea. The amount corresponding to a degree of Fahrenheit was greater at a high than a low temperature; and in the fit of each day the same amount was excreted for each degree of temperature. This increase in the urea must be regarded, in some measure at least, as an indication of increased metamorphosis; and the close relation to the febrile heat certainly implies that it owns only this source, and is not caused by elimination following previous retention (PARKES). The uric acid is greatly increased during the fit; and after the fit there are often deposits of urates, either spontaneously or on the addition of a drop of acid, and it seems probable that the increase in the excretion of uric acid continues for some time after the paroxysm; and the enlargement of the spleen in connection with this great increase of uric acid is probably not fortuitous (RANKE). The influence of quinine in diminishing the amount of uric acid in health is of interest in connection with its effect upon malaria, and with the condition of the spleen in malarious fever (RANKE, BOSSE). The chloride of sodium is increased during the cold and hot stages to a great degree (TRAUBE, RINGER), to five times the normal amount; and phosphoric acid is diminished to *one-eighth* (NICHOLSON). The results are contradictory regarding the urine of a fever day, compared with the urine of a fever-free day. This may be explained to some extent by the relative duration of the fit, compared with the fever-free period; and great differences may arise from the comparative length of the apyretic period on the fever days; also from the severity or the reverse of the fit, and from the amount of food and drink able to be taken. With respect to abnormal constituents, albumen is found during the fit in a considerable number of cases. Blood in some quantity, and renal cylinders, are seen about as frequently as albumen; and occasionally chronic Bright's disease is a consequence of ague (PARKES, l. c.)

Prognosis.—Uncomplicated cases of ague are never fatal. The danger to life arises from lengthened residence in malarious places, and from the persistent pernicious influence of malaria on the constitution, bringing about the malarious cachexia, with all its organic effects. These are especially splenic and hepatic congestions. During each paroxysm, and subsequent to it, the condition of these two important organs—the spleen and the liver—ought to be carefully examined into. It ought to be observed, also, whether any symptoms exist of effusion into the abdominal cavity. When the type of the fever is malignant, or of a severe and complex kind, or when the complications are locally severe, it is difficult to cure an ague, which otherwise is a very manageable disease. During the cold stage, especially if it is of long duration, the liver, and especially the spleen, become turgid,

the symptoms of which generally disappear with the sweating stage of the fever. It is when the endemic influences are severe, or when the attacks are prolonged over months and years, that these organs begin to suffer permanently from organic disease.

Treatment.—It is useless to attempt the cure of intermittent fever if the sufferer is permitted to remain within the sphere of malarial influences. The old empirical treatment in the English fens was first an emetic, then a sharp purge, followed by quinine and arsenic. But an emetic must not be given for ague, without regard to the special circumstances of the case. Emetics are only of use when the tongue is very foul or the stomach overloaded; or when there is nausea and headache with epigastric oppression. An emetic will then give great relief, and hasten the stage of reaction. In the simple cases, when removed from the sphere of malarial influence, active purgatives are always beneficial in relieving the full and congested state of the abdomen generally; and they prepare the way for the action of *quinine*, especially combined with diuretics, such as *cream of tartar* (WOOD). There are also cases of intermittent fever complicated with hepatic and other engorgements, which continue to recur despite of all means, until a few doses of *calomel*, followed by purgatives, are administered; then the *quinine*, which before failed, will speedily cure the disease. The *compound jalap powder*, combined with *calomel*, is found very beneficial in such cases. If much urinary irritation exist a mixture of ʒvi of *bicarbonate of soda*, dissolved in ʒviiss. of water, to which ʒss. of *Sp. Ætheris nitrosi* is added, given in half-ounce doses every two or three hours, with or without *tincture of opium*, will relieve it. Certain morbid conditions, both of liver and spleen, may produce and maintain the tendency to recurrences of ague. Ramazini relates the case of a patient harassed by an obstinate ague, and who was cured by mercurial frictions administered for syphilis. The influence of splenic disease in keeping up the morbid train of actions of the original fever, and in producing relapses, has been recorded by M. Piorry. In more than 500 cases of ague in which he observed the state of the spleen, he comes to the following conclusions, namely:—that the organ is invariably enlarged during the progress of the fever, and that by the use of quinine the spleen diminishes in size; that its reduction in size bears some relation to the quantity of quinine taken; that the effect it produces upon the fever is in proportion to the reduction of the spleen; that the disease is cured simultaneously with the subsidence of the splenic enlargement; and that the fever is apt to recur so long as the spleen exceeds its normal size. The treatment of the disease as to stages is to be managed as follows:—During the cold stage the patient generally desires to be let alone, care being taken that he has abundance of bed-clothes. If the stage continues unusually long without signs of reaction the application of external warmth becomes necessary with warm drinks; and even *ammonia*, *ether*, *camphor*, and other diffusible stimulants may be required if the vital powers are unequal to the development of reaction. During the hot stage, diluent drinks may be indulged in, if the patient desires them, with cooling diuretics; but the danger consists in the approach of collapse when the sweating stage commences; and when stimulants and support may be required. The first dose of *quinine* should, then, be given towards the close of the sweating stage. “I always assume,”

writes Professor Maclean, "that in first attacks the type of the disease will prove to be *quotidian*, and take my measures accordingly, making sure that the patient shall have *thirty grains of quinine* between the termination of one paroxysm and the hour when we may look for another; and looking to the fact that we may look for the setting in of the cold stage, perhaps two hours earlier than on the first day, the last *ten-grain dose of quinine* should be given so as to anticipate that time by at least an hour." It is to be administered in solution, dissolved by a small quantity of *dilute sulphuric acid*, and the dose mixed with syrup of orange peel. It may also be administered in starch or beef-tea *enemata*, or by *subcutaneous* injection. The object and action of quinine are to break the system off "the habit of going through the phenomena of an 'ague fit.' It counteracts the influence of the malarial poison, and prevents the repetition of the attacks. A moderate degree of *cinchonism* must still be maintained for some days, to the extent of 'ringing in the ears,' by giving three or four grains of *quinine* in solution every three or four hours, and a day or two before the interval of a month the patient must be again brought under the influence of this remedy. Within this period (a lunar month) there is always a tendency to the repetition of the 'ague fit,' the habit of which must be thus broken; otherwise it will be strengthened by every successive attack" (MACLEAN). Next to *quinine* in the cure of ague comes *arsenic*. From six to eight drops of *Fowler's solution* may be given during the interval of freedom from fever. It should be given in small doses, and not persevered in for more than eight or ten days; and like *quinine* its use must be continued in diminished doses for some time after cessation of the fever. The earliest signs of its physiological action must be looked for to determine to what extent it must be pushed—these being redness of the eyes, watering of the mouth, and a silvery state of the tongue. The dose should always be given after food. In malarial brow-ache (brow-ague) and malarial hemi-crania it is more efficacious than *quinine*. Due attention must be paid to feeding during the intermission, especially by beef-tea and farinaceous food.

In all complications of ague, be they cerebral, pulmonic, hepatic, or gastric, *quinine* must still be the therapeutic agent to be relied upon. Antiphlogistic measures are not to be substituted in place of it on any account whatever. As to the malarious cachexia, removal from the malarious locality is the first essential condition to restoration of health; the next is a combination of nutritious diet, abundance of exercise, pure air and pure water; and clothing should be warm on first coming into cold latitudes. The prophylactic use of *quinine* must still be persevered in; and if appetite fail, a course of *syrup of the phosphate of iron, quinine*, and *strychnia* in ten-minim doses, in a large quantity of water (a claret glassful), will be of service. In the experience amongst the invalided soldiers at the R.V. Hospital at Netley Dr. Maclean has found this medicine of much use. The chronic malaria cachexia is also much benefited by *arsenic*, especially when there is diarrhoea. In such cases two minims of *Fowler's solution* with five minims of *laudanum* should be given before meals. So also the use of *coti bark* may be of service in diarrhoea. In the treatment of the *anaemic chlorosis* of malaria *arsenic* is especially adapted to those cases in which *iron* does not agree; while the efficacy of *iron* is often also increased by its combination with *arsenic*.

Should the spleen continue of large size ointment of the bin-iodide of mercury is to be rubbed in over the gland. A portion about the size of a nutmeg is to be applied with a smooth spatula, the patient sitting before the heat of the fire, as long as he can bear the smarting which follows. The remedy has a not less beneficial influence on enlarged livers (PROF. W. C. MACLEAN).

REMITTENT FEVER.

LATIN EQ., *Febris Remittens*—Idem valet, *Febres Pestiferæ Singularum Regionum*; FRENCH EQ., *Fièvre Rémittente*—Syn., *Fièvres Pernicieuses*; GERMAN EQ., *Bösartiges Endmisches Fieber*—Syn., *Bösartiges Locales Fieber*; ITALIAN EQ., *Febbre Remittente*; *Febbri Perniciose dé Climi Caldi*.

Definition.—*Febrile phenomena of malarious origin, characterised by irregular repeated exacerbations and remissions, the remissions being less distinct in proportion to the intensity of the fever, so that in malarious yellow fever the exacerbations and remissions are so connected that the fever resembles a continued fever. There is great intensity of headache, the pain darting with a sense of tension across the forehead. It is accompanied by functional disturbance of the liver, and frequently by yellowness of the skin, as in malarious yellow fever, in which, however, the urine is not suppressed, and continues free from blood and albumen. The malignant local fevers of warm climates are usually of this class.*

Pathology.—See p. 419, *ante*, Chap. VI. It may be here noted that there are great varieties in the degree of severity and type of this fever, more especially as it occurs in England, France, Holland, and Germany, compared with its occurrence in Spain, Italy, the Mediterranean islands—or still more so in Africa and the East and West Indies. Accordingly some authors (CRAIGIE) distinguish three varieties,—*e.g.*, (1.) The autumnal remittents of temperate countries, as England, France, Germany, Holland, Hungary. (2.) The summer and autumn remittents of warm countries, as Spain, Italy, Greece, the Mediterranean coasts and islands generally, the Levant, the north of Africa and Asia, and the United States. (3.) The endemic remittents of hot and tropical climates, as in the south of Asia, Central and Western Africa, Equinoctial America, and the West India Islands. Accordingly, remittent fever has received different names from the localities where it prevails. Thus we have the *gall-sickness* of the Netherlands, the *Walcheren fever*, *fever of the Levant* (IRVINE), *Mediterranean fever* (BURNETT), *Hungarian sickness*, *puka fever* of the East Indies, *jungle fever*, *hill fever* of the East Indies, *bilious remittent* of the West Indies and Mediterranean, *Bulam fever*, *Sierra Leone fever*, *fever of Fernando Po* and *Bight of Benin*, *African fever*, and *Bengal fever*. Prevailing on the borders of inland lakes, as in America, it is sometimes called the *lake fever*. (See page 269 on such nomenclature.) Malarious yellow fever seems to prevail, for the most part, in towns situated on the sea or river coasts of alluvial countries in warm climates; and, that while the banks of these rivers or seas are liable to occasional

alternate periods of inundation and drying up, the fluctuations of the tides, co-operating with these, contribute powerfully, under intense solar heat and a windless atmosphere, to render the towns along the shores of such districts the seat of malarious yellow fever. "It appears to be the product of that state of the atmosphere which takes place after a long continuance of solar heat, with little or no wind, in those points chiefly where the atmosphere of the sea and that of the land are in constant communication and interchange. It is, indeed, a remarkable fact that the intense form of remittent fever which has been distinguished as malarious yellow fever, and sometimes as 'bilious remittent of malignant type,' is rather rare in the interior of countries, and is seldom found in towns situated on rivers higher than the influx of the tide. The fevers which appear in these situations are more of the usual remittent character; and in the interior of the American continent there is little doubt that the *lake fever* represents the malarious *yellow fever* of the coasts. Even in Europe, while the towns on the sea-coast and on rivers were labouring under the malarious yellow fever, the sickliness in the interior approached more to that of the remittent or remittent continuous type" (CRAIGIE). For this reason the term *littoral*, as well as *paludal*, is used to designate this class of fevers. (See Chap. VI., p. 419.) The endemic conditions under which malaria gives rise to this form of yellow fever may be referred,—(1.) To thermometric temperature of the air; (2.) To the state of the atmosphere as to currents of winds and electricity; (3.) Local peculiarities of surface already referred to; (4.) Constitutional susceptibility and crowding together of masses of people. When an observer has seen only the milder form of marsh *remittent fever*, and is then suddenly called upon to witness an attack of *malarious yellow fever*, he may well believe that the affections are entirely distinct. But after a time, when the intermediate forms have been more closely scrutinised, it is found that at no point can any valid line of demarcation be drawn between the several forms of these malarious fevers, so numerous are the connecting links which bind them to each other (ALISON, PARKES).

Symptoms.—There are so many grades of intensity in remittent fever (varying as it does from a severe intermittent to malarious yellow fever), and so many different modifications are impressed on it, from the great variety of country and climate in which the poison is generated, that it is extremely difficult to generalise the phenomena. The severer forms of remittent fever may be preceded by certain premonitory languor, listlessness, restlessness, or chilliness; or there may be a want of appetite, anxiety, lassitude, pain at the epigastrium, pains in the loins and limbs, headache; slow, small, and irregular pulse; coldness of the skin, and chilliness for one or several days before the commencement of the attack: these are symptoms which usher in a short cold stage. But in other cases the attack is sudden, and the patient, immediately after a hearty meal, may be seized most unexpectedly with faintness, vertigo, nausea, confusion of thought; and these almost without a rigor, or a very short one, not exceeding half an hour: a hot stage follows, usually of much greater intensity than that which accompanies the worst forms of intermittent fever. The hot stage, or period of exacerbation, generally commences at noon, or early in the afternoon, subsiding towards evening, or

in the early part of the night, the remissions being generally most complete early in the morning. Sometimes, however, the exacerbations come on towards evening, and continue all night, the remissions being then most complete in the forenoon; while, in a few cases, there may be two exacerbations in the twenty-four hours; and these cases are generally most severe. The exacerbation is usually marked by much cerebral affection, as severe headache, a painfully acute state of every sense, an injected state of the conjunctiva, and great action of the carotid arteries. The pulse, varying from 90 to 120, is generally at first full, but is sometimes from the first small, and generally soft and easily compressible. The tongue is dry, with a white and sometimes yellowish fur, and a bad taste in the mouth. There is generally unquenchable thirst, parched lips, tenderness at the epigastrium, and sometimes pain, with increased dulness on percussion, in the region of the liver. These symptoms are frequently accompanied by delirium, sometimes of a violent character. When giddiness is distressing, and proceeds to high delirium at an early period, a severe form of fever may be expected. In other cases the patient is oppressed with great drowsiness, lethargy, or coma. The stomach is often the seat of great pain and uneasiness, followed by vomiting, and the matters vomited are either colourless, bilious, or bloody. The duration of this paroxysm varies considerably. When the disease is mild it may terminate in six or seven hours; but if severe it may last fifteen, twenty-four, thirty-six, or even forty-eight hours; and Dr. John Hunter once saw a case in which there was no remission for seventy-two hours. Inability to sleep is almost constant. The urine is scanty, high-coloured, and of high specific gravity (1024 to 1030), acid, not coagulable by heat (MURCHISON). Albumen was tested for by Dr. Murchison in numerous instances, but never detected; and according to Jones's experience in America, it is very rarely present,—a point of difference, if verified, of great importance as a distinction between severe remittents and specific yellow fever (PARKES). In severe remittent fever Jones found the urea increased, and the uric acid lessened till convalescence, when it again increased, and the pigment was also lessened (PARKES, l. c., p. 242). The fever, however, at length remits, sometimes with sweating, but at other times without any sensible increase of perspiration. The first exacerbation is generally the longest, lasting, in some cases, for twenty or twenty-eight hours; but generally after twelve or sixteen hours the symptoms remit. The duration of the remission which follows is as various as that of the hot stage. Sometimes it does not last longer than two or three hours; more commonly it extends to six, eight, ten, fifteen, thirty, or even thirty-six hours. The fever then returns, and in some cases assumes a quotidian type, and has an exacerbation every day, and perhaps nearly at the same time, yet more frequently there is no regularity in the times either of its accession or remission.

The second paroxysm is always more severe than the first, if the progress of the fever has not been checked during the remission, and usually neither any cold stage, rigor, nor even chilliness precedes it. On the other hand, all the febrile symptoms run much higher, the skin is hotter, the pulse more frequent, the headache greater, the senses more confused, and the delirium or coma, when that exists, more violent in degree and more

sudden in its accession. Delirium, with more or less loss of consciousness, may not supervene till the third or fourth paroxysm; and is of a low wandering character in the asthenic form of the fever. The tongue becomes dry, hard, and brown, or almost black; the teeth covered with brownish scales; and the pulse becomes small and weak. These symptoms sometimes persevere, with or without black vomit, till they terminate perhaps in coma more or less profound, great prostration, *subsultus tendinum*, fetid breath, resembling the odour of a dead body, convulsions, and at length in death. The severe forms of the fever are sometimes accompanied with a yellowish hue of the skin and white of the eyes. The yellowness is said to be less where there is a copious bilious diarrhoea, and where the urine is of a dark yellow-brown colour. When the disease does not terminate fatally, amendment is generally observed after the fifth exacerbation, which may subside in very copious perspiration, with the following symptoms of progressive amendment—the tongue begins to clean and grow moist at the edges, the sordes disappear from the teeth, the thirst diminishes, and the appetite gradually returns. The pulse remains slow and soft, but begins to acquire strength; and the skin continues cool and moist, sleep returns, and the strength is gradually but very slowly recovered. Headache may continue for some days, till relieved by epistaxis. Young and robust men, particularly recruits who recently arrive in India from Europe, suffer a considerable amount of vascular excitement, with marked symptoms of determination of blood, either to the head or to the abdominal viscera, at a very early stage of the fever. In such cases the pulse is at first full, and of tolerable strength, the skin burning, and the delirium raging and acute. Again, in other men more advanced in years, or those debilitated by long service in India, by previous disease, or by habits of intemperance, there is very little vascular excitement, even during the exacerbations; and the pulse, though quick, is small and weak. There is no great heat in such cases—there is even coldness of the skin, with a yellow tinge, often severe hiccup and vomiting, with great prostration of all the vital powers. In such cases the chief indications of the exacerbations are increased restlessness, vomiting, headache, or wandering delirium; and the remissions are not well marked, even from the commencement of the attack (MURCHISON, l. c.)

Diagnosis.—See Specific Yellow Fever.

Prognosis is generally favourable when uncomplicated. Early subsidence of gastric irritability, of headache and frequency of pulse are favourable signs; so are distinct well-marked remissions with action of skin and postponement of exacerbation. But if the fever tends to be continued rather than paroxysmal, with a pulse failing in strength while it gains in frequency; if there be tendency to collapse at the close of the exacerbation; hæmorrhages from stomach or bowels, cold sweats, delirium, coma—each and all of these signs point to danger (Professor W. A. MACLEAN).

Treatment.—With fever so various in its degrees of severity, it is not possible to do more than indicate the nature of the treatment which may be followed, as every special case must be prescribed for and treated by its own special indications, and with a due regard to the nature of the prevailing epidemic. The best possible hygienic arrangements should be made, especially to secure efficient ventilation, with a plentiful supply of

fresh air. In every form and variety of the fever one of the most important guides in the treatment is to be derived from the nature of the prevailing disease, whether endemic or epidemic, whether *sthenic* or *asthenic*, and to study each individual case in relation to the prevailing type. First, *the duration of the stage of the fever* must be ascertained—i.e., whether it be of some hours' or of some days' duration, and whether, when the practitioner sees the patient for the first time, the actually existing paroxysm is at its accession or its decline.

Regarding the method of treatment of remittent fever, my friend and colleague, Professor W. A. Maclean, whose extensive experience in India gives authority to his conclusions, writes in the following terms:—

"I have been led to take a view of the treatment of malarial fevers generally, and remittent fever in particular, differing from that laid down by many authors. It appears to me that the so-called antiphlogistic treatment, so much insisted on by many writers, is based on the belief that the phenomena observed in a case of remittent fever are consequent on a process of inflammation. It is only on such a belief that antiphlogistic treatment can be justified. During the exacerbation of a remittent fever there is violent disturbance both of the vascular and nervous systems. Almost every organ, almost every function suffers,—the gastric intestinal membrane is affected, the liver and spleen suffer, the brain is involved, for rending headache and delirium are often present. Is it rational to suppose that an inflammatory process can be going on at one and the same time in all these various organs? Do the appearances observed *post-mortem* give any support to such a doctrine? If not, on what principle can spoliative treatment be justified? Is it not rather the case that this terrible disturbance of so many organs is due to the presence in the blood of a subtle poison acting on them all? If so, surely the guiding principle of the physician in his treatment should be to counteract this poison, to neutralise it, or to expel it from the system, and so to prevent a recurrence of the exacerbation. This is the principle on which I have long acted, and I am satisfied that it is at once a safe and successful one. In *quinine* we have such an antidote—a therapeutic agent of unrivalled efficacy, which, if skilfully used, will rarely disappoint the expectations of the practitioner.

"It is always, of course, advisable to have the bowels thoroughly evacuated; and if the patient is seen when his stomach is loaded, it is well to evacuate its contents by an emetic. In ardent remittents, however, there is generally little call for this, as obstinate vomiting is almost always a troublesome symptom, sometimes requiring to be checked. This done, the period of remission must be watched for, and the moment it arrives quinine in a *full dose* should be given—not less than *fifteen grains in the case of an adult*. If the irritability of stomach be so urgent that the remedy is rejected, while measures must be adopted to allay it—such, for example, as *alkaline remedies* in combination with *dilute hydrocyanic acid, turpentine stupes*, or even a *blister* to the epigastrium—time—precious time—should not be lost. Quinine should be given *by the rectum in a full and efficient dose*. By mouth or by rectum, or by both, quinine in quantity sufficient to induce some of the symptoms of cinchonism, should be given before the time of expected exacerbation. According to my judgment and experience, it is bad practice to withhold quinine until an impression has been made on the force and frequency of the heart's action, from fear

of increasing headache, causing congestion of organs, or the like. An impression on the force and frequency of the heart's action is best attained by arresting the paroxysm; and this is done most quickly, simply, and effectively by the early administration of *quinine*. I have over and over again had patients brought to me from the malarial quarters of the city of Hyderabad, in whom it was impossible to distinguish any period of remission—the tongue black and dry, sordes on the teeth, the skin hot and parched, the pulse enormously quick, the intelligence feeble or gone—all pointing to a system so charged with malarial poison as to be well-nigh overwhelmed. In such cases *quinine*, with concentrated *beef-tea* and *brandy*, are urgently called for, and should be administered freely; and it is astonishing how men, by such measures, are often snatched from impending death. I have seen in a few hours consciousness return, a striking reduction in temperature, in the *frequency* of the pulse, with a remarkable accession of force and volume, follow the treatment indicated above. I do not advise, and never used *quinine* in the heroic doses advised by some. I have never exceeded *twenty grains*; but within such reasonable limits I have never seen it aggravate headache. On the contrary, I believe that in remittent fever—in fact, in all forms of malarial fever with which I am acquainted—I believe *quinine* to be a powerful remedy in quieting the tumultuous action of the circulation disturbed by the presence of this terrestrial poison. For some years past Warburgh's tincture has been much used in the treatment of malarial fevers in Southern India. It is a secret remedy, and therefore open to the objections very properly urged against all such remedies. It is now known that *quinine* enters largely into this remedy, the composition of which is published in the *Lancet*, Vol. II., 1875, p. 716. I have given this 'tincture' a fair trial in some of the gravest forms of malarial fever, and it has also been extensively used by some of the most experienced officers of the Madras army; and I do not hesitate to say that I think it a valuable remedy. I have known it arrest at once some of the severest cases of remittent fever, no exacerbation appearing after the second dose. It almost invariably acts as a powerful diaphoretic—the most powerful with which I am acquainted. I have seen patients, under the influence of this remedy, saturate not only the bed-clothes, but the very mattress, the patient's room and his person for days after giving out a strong odour of the medicine. For this reason it requires to be used with extreme caution, if at all, in the adynamic form of the disease.

"In urgent cases I follow the practice of the American physicians. I do not wait for a remission, but give *quinine* at once; and in all I am conservative of the patient's strength. I have seen violent delirium follow free leeching of the temples, and over and over again seen extreme and dangerous prostration follow depletive treatment, and that in cases where the violence of the disturbance indicated *power*; but these signs of power in the system are often most delusive, and, if combated by depressing measures, we must be prepared for sudden signs of collapse. Against the system of treating this fever by saturating the system with mercury, I enter my strenuous protest. I know nothing more deplorable than the condition of a patient whose constitution, already depressed by the presence of malaria, is further saturated by another poison which acts as a powerful ally of the first."

In the asthenic form of remittent fever, such as that so well described by the late Dr. Murchison as prevailing in Burmah, it is necessary to exer-

cise great caution in depletion. All the cases he relates which had been freely bled exhibited the most aggravated typhoid symptoms, and most of them died. Even in the instance of young and robust recruits, low adynamic typhoid symptoms were sure to supervene in a short time after blood-letting; and, even although it gave temporary relief, it was certain to aggravate, if not to induce, the subsequent typhoid condition. If the headache is very severe and the pulse full; if the hair be cut short, or shaved off the scalp, cold lotions applied to the head, or the cold douche kept up for ten minutes at a time, gives great relief, and is the preferable remedy (MURCHISON). As soon as possible after the commencement of the paroxysm the bowels should be cleared out with a purgative of *calomel* and *compound jalap powder*; or by *colocynth*, *antimonial powder*, and *calomel*. If typhoid symptoms betray themselves, stimulants, such as wine and brandy, must be given; but, as in intermittent fever, "*quinine* is undoubtedly the sheet-anchor," and it is best given, as in the former fever, in one large dose of *twenty grains* in solution at the very commencement of a remission. *Carbo-azotic* or *picric acid* has been lately introduced as an active remedy in the treatment of malarious fever. Prepared by Calvert, of Manchester, it is of a light yellow colour; and in doses of two grains, cautiously repeated, it is to be pushed till the patient gets yellow-skinned.

The period of convalescence demands no less careful attention on the part of the medical attendant, especially as to diet and a timely removal from all malarious influences, by a sea voyage, or a change of climate. It is to the mismanagement of convalescence, and a too early discharge from hospital principally, that we must refer the numerous and fatal relapses in the fevers and dysenteries of our seamen and soldiers (MARTIN).

SIMPLE CHOLERA.

LATIN EQ., *Cholera Simplex*; FRENCH EQ., *Choléra Simple*; GERMAN EQ., *Sporadisch Cholera*; ITALIAN EQ., *Coléra Sporadico*.

Definition.—*An acute catarrhal inflammation of the mucous membrane of the stomach, which extends into the intestines, attended with nausea, retching, and vomiting, followed by severe watery diarrhoea, consisting of profuse transudation of fluid containing little albumen; the whole system generally being affected, as evidenced by fever.*

Pathology.—(a.) *History.*—A form of diarrhoea which often attacks a number of persons simultaneously, so as to prevail almost epidemically, and mostly during the hot weather towards the close of summer, or the beginning of autumn. Sydenham regarded its epidemic occurrence then as certain as swallows in the spring, and that it seldom continues longer than the month in which it appears. Now it seems to occur more frequently—for example, rather in September than in August; and cases occur earlier than in August, namely, in May and June (BATEMAN). It has therefore also received the name of *summer cholera*. If it occurs during an epidemic of *malignant cholera*, it is most difficult (except by the result) to distinguish

it from that severe disease; and while *summer cholera* itself cannot be regarded as a miasmatic disease, it is something different from a mere bowel-flux, and must be regarded "as a disease affecting the individual in his entirety, and not limited merely to the mucous membrane of his intestinal canal. It is a general and not a local disease," therefore it finds a place here as distinct from *diarrhœa*. The term *cholera* was used by Hippocrates, and is now given to any complaint in which the prominent characters are simultaneous and repeated purging and vomiting, with painful spasm of the stomach and bowels, and occasional cramps of the external muscles. Celsus derives the word from $\chiολή$ and $ῥέω$, literally meaning *bile-flux*. Others derive it from $\chiολάς$ and $ῥέω$, literally, *intestinal flux*. The term *morbus*, since added to the original Greek name, is a superfluity. The name *cholera* stands alone in Celsus, Galen, and Hippocrates; and as the additional term was unnecessary in their day, it is wholly superfluous in ours. It has not been adopted in the new Nomenclature of the College of Physicians, either as an equivalent or a synonym, and it is to be hoped it may finally disappear. The term *sporadic cholera* is intended to distinguish the disease from *malignant Asiatic cholera*, which spreads as an epidemic; while sometimes by French writers *sporadic* or *simple cholera* has been called *European cholera*, and by British physicians *English cholera*, but it is common in all countries where *malignant Asiatic cholera* is not. In America it is most common in July and August; and generally in hot countries, and during the hot season in temperate latitudes. A complex series of morbid conditions characterise this disease *per se*. It is more than simple indigestion. The increased flow of fluid, greatly altered in quality, from the stomach and bowels; the general irritation and the spasmodic contractions of the stomach and intestines; the enteralgia,—are features which, taken collectively, mark the general affection of the system.

(b.) *Cause*.—An elevation of atmospheric temperature seems to have some special influence in bringing about this form of cholera, either by contributing to the development of other more immediate causes, or by rendering the system more susceptible to the disease. It occurs more frequently in childhood, youth, and middle age, than in advanced life. Of the more immediate causes may be mentioned—(a.) Overloading the stomach with food. This is especially apt to be the case with children; and mainly among such as have their diet restricted as a rule, and who, having a chance of eating without control, are certain to overload the stomach. (b.) The use of food difficult of digestion, resulting in products of decomposition, which act like a poison; (c.) The use of food which has begun to decompose before entering the stomach,—e.g., spoiled meat, new beer, sour or acid food, such as milk; (d.) Exposure to cold and a chill when the body is heated and perspiring, and the stomach engaged in the midst of digestion.

Symptoms.—The attack of simple cholera is rarely preceded by any premonitory symptoms; on the contrary, it is often abrupt, by a sudden seizure during the night. If it occurs during the day there may be a period, usually very short, rarely longer than a few hours, of considerable discomfort from a sensation of pressure at the pit of the stomach, of weight or uneasiness referred to the epigastrium, and extending more or

less over the abdomen, with nausea and the occasional pains of colic. The attack then begins with vomiting and increase of nausea. The food last eaten is vomited, often but little changed; and, as the vomiting is frequently repeated (the entire contents of the stomach being thus got rid of) liquid ejecta follow, sometimes in large quantities; first of a pale yellow or green colour, and of a bitter taste like bile; sometimes very acid, and so acrid as to produce a scalding sensation in the throat. A great noise is made by the movement of air (*borborygmi*) in the intestines, followed by stools, at first pulpy, then thin and liquid, which, like the first liquid vomited, frequently occasions a burning sensation at the anus. Vomiting and purging thus occur in rapid succession, and recur again and again either simultaneously or in close alternation, constituting the prominent and distinctive features of simple cholera. Enormous quantities of fluid are thus evacuated, of such a character that the greater the amount the less colour it has, as the bile, even if it were of normal quantity, is not sufficient to give colour to the amount of liquid transudation from the stomach and intestines. Intense thirst then begins to be excited from so large a loss of fluid; and drinks, taken in however large quantities, do not seem to quench inordinate thirst. The acts of vomiting and purging are sudden, and without or with but little warning, are often also so violent in robust patients that the contents of the stomach and intestines are ejected with considerable force. Fluid taken as drink is thus rapidly evacuated alike upwards and downwards. Before the earliest acts of purging, and generally preceding and accompanying the subsequent acts, colic pains prevail over the abdomen. At first they may be extremely severe, but are generally relieved by free evacuation. Spasm or cramp also often attends the acts of vomiting, and there are apt to be painful contractions of the muscles, especially of the calf of the leg and feet, and of the abdominal muscles, causing hard lumps or knots, as in colic and cramp. These irregular muscular movements amount sometimes to general convulsions; and add greatly to the sufferings of the patient, who becomes anxious and restless, with a feeling of depression and exhaustion proportionate to the violence and continuance of the vomiting, the purging, and the cramp. The pulse becomes rapid, small, and feeble. The voice is weak, altered in tone, and occasionally disappears altogether. The countenance is pale and shrunk, the skin cool and damp; the urine scanty or suppressed. After these symptoms continue for several hours, the vomiting and purging become less severe, and longer intervals of rest and quiet intervene, so that the symptoms progressively diminish, the vomiting and purging subside, the skin becomes warm, and acquires its natural feel. The patient now exhausted to the utmost, falls asleep, and awakes to experience an amount of depression proportionate to the continuance of the evacuation, and to the violence and duration of the attack.

Diagnosis.—The points regarding which there is a chance of error are as regards,—(1.) *Malignant epidemic cholera*; (2.) *peritonitis*; (3.) *poisoning from acrid or corrosive substances*. During an epidemic of *malignant Asiatic cholera*, it is not always possible to distinguish cases of *simple cholera* from those caused by the *malignant cholera poison*. The symptoms are not only similar, but are absolutely the same as those of the milder cases of Asiatic

cholera. The chief difference is in the result—almost all recover from simple cholera; more than half the patients die of malignant cholera. *Acute peritonitis*, accompanied by vomiting and purging, has been mistaken for simple cholera; but it is rare for vomiting and purging to occur so early in peritonitis as to lead to the belief that the patient suffers from cholera and not from *peritonitis*. Pain on pressure also is absent in cholera; and other distinctive marks of peritonitis ought to be sought for in all cases of apparent cholera. With regard to *poisoning*, the acrid and corrosive poisons occasion vomiting and gastric irritation, which continue some time before the commencement of purging; whereas in *cholera* the diarrhoea is simultaneous with, or quickly follows, the vomiting. In *poisoning* the vomiting is more severe, and is usually severe out of all proportion to the diarrhoea. There is also, in cases where the poison has been in a sufficient dose, decided pains or tenderness over the stomach, and a constant excruciating pain between the acts; whereas in *cholera* a period of ease usually follows the acts of vomiting and purging. If the disease lasts unusually long, every circumstance that can indicate the presence of poison ought to be examined into with care.

Prognosis.—No matter how threatening the symptoms may be, however great the collapse and depression of the patient may seem, a previously healthy adult very seldom dies of *simple cholera*. Although the disease so much resembles *Asiatic cholera*, “it rarely goes on to the disappearance of the heart’s beat and beat of the pulse, to the cyanotic hue and reptile temperature of the skin, which is seen on the approach of collapse in that disease.” The patient usually recovers rapidly, and the mucous membrane of the stomach and intestine speedily resumes the normal exercise of its functions. But *simple cholera* is very apt to kill children (*cholera infantum*), and to cut off aged, sickly, and weakly people. In them the bowels are early paralysed, vomiting and purging may seem to cease, but transudation continues, the pulse disappears, the movements of the heart become weaker, the intellect cloudy, and the patient dies of exhaustion.

Treatment.—If the stomach retains undigested food believed to be unwholesome, a mild emetic may be given; but this is rarely necessary, as the stomach usually empties itself completely. When the solid matters have been ejected, the subsequent treatment will depend on the acidity or acrid nature of the matter ejected. Simple alkaline remedies, like *carbonate of soda* in solution *ad libitum*, will sometimes relieve the nausea and sickness; and when the vomiting becomes liquid, *opium* is the remedy most to be relied upon. It must be, however, given in solution, such as *laudanum*, *Battley’s solution*, or with *chloroform* and other *stimulants*, as in the form of *chlorodyne*. A full dose of either of these, or a full dose of *muriate of morphia*, laid on the tongue, should be administered immediately after the act of vomiting. If the first dose should be immediately rejected, a second is to be repeated; if that too is rejected, a third may be so given, but no more. An *enema of laudanum* (one to two drachms) with *starch* must be given instead, directly after an evacuation of the bowels. If this is rejected after being again repeated, then *hypodermic* injection of *solution of morphia* may be adopted; and in any form when retained, the remedy will require repetition every hour or half hour

till the vomiting and purging are arrested. The effects of each dose must be carefully watched, so that narcotism may not be induced. It is important that so much fluid may not be taken as the greatness of the thirst desires. A table-spoonful of ice-cold water, given at short intervals, or a bit of ice to dissolve in the mouth, is all that ought to be permitted. Gruel made from the boiled fluid in which oatmeal has been soaked, and allowed to cool, and given with ice, is a grateful and soothing drink.

MALIGNANT CHOLERA—*Syn.*, SEROUS CHOLERA; SPASMODIC CHOLERA; ASIATIC CHOLERA.

LATIN EQ., *Cholera Pestifera*—Idem valent, *Cholera Serosa*, *Cholera Spastica*, *Cholera Asiatica*; FRENCH EQ., *Choléra Asiatique*; GERMAN EQ., *Cholera Asiatische*; ITALIAN EQ., *Colèra Asiatico*.

Definition.—*A disease essentially specific, existing at all seasons of the year in certain parts of India (its perennial endemic area), capable of being propagated or disseminated over the surface of the earth, through the atmosphere, or in other ways, but chiefly by means of human intercourse between the healthy and the sick. The seizure is characterised in many (but not in all) cases by premonitory diarrhœa, sudden muscular debility, tremors, vertigo, occasional nausea, and spasmodic griping pains in the bowels, depression of the functions of respiration and circulation, and a sense of faintness and oppression in the præcordial region. A copious purging of serous fluid (alkaline when passed, and in appearance resembling water in which rice has been washed, sometimes containing blood) is succeeded or accompanied by vomiting and burning heat at the stomach, coldness and dampness of the whole surface of the body, coldness and lividness of the lips and tongue, cold breath, an unquenchable thirst, a feeble rapid pulse, difficult and oppressed respiration, with extreme restlessness (a state expressed in physiological language by the term “anxietas”), suppressed urinary secretion, blueness of the entire surface of the body, a sunken and appalling countenance, a peculiarly suppressed voice, a peculiar odour from the body, partial heats of the præcordia and forehead—fatal collapse, a reaction and secondary fever. Under conditions favourable to its propagation malignant cholera often becomes epidemic.*

The College of Physicians defines cholera to be “*an epidemic disease, characterised by vomiting and purging, with evacuations like rice-water, accompanied by cramps, and resulting in suppression of urine and collapse.*”

Pathology.—(a.) *History.*—Cholera has been and is endemic or common to the inhabitants of a certain part of the world only. It is a disease native to and endemic within a certain area of India—“*the endemic area of cholera.*” It is not endemic either in Arabia, or Persia, or the Straits Settlements, nor in China, nor in any part of Europe, Africa, nor America. The chronological and geographical history of cholera has commonly been considered as affording *primâ facie* evidence that, as regards this country, it is originally an exotic disease, the product of another climate, which yet has met with conditions favourable for its development and propagation in this country. It is now quite clear that exactly the same disease as Indian cholera was known in India when the Portuguese went there first, about the year 1500 (Dr. MACPHERSON). Cholera in

India has had periods of increase and of decrease; but it was never diffused in that country so widely as it has been since 1817; nor has it ever wandered so extensively from India before. Whatever cholera we may have had in Europe in former times, few doubt that, since 1817, it has always been carried out of India to other places. It is endemic in Calcutta, Madras, and Bombay, and probably in all large towns along the sea-board of British India, including Chittagong, and parts of Pegu. It frequently makes its appearance in the ceded districts of Madras, of which Bellary is the capital (which includes the table-land between the Eastern and Western Ghauts), having an elevation of some 1,600 feet above the level of the sea. It breaks out year after year during the annual festival at Humpi. From these districts, passing northward to Saugur and Nagode, in the Gangetic basin, it appears there (to a greater or less extent) twice a year. To identify the cholera endemic area within a general but definite boundary, Dr. Macnamara draws "an imaginary line to the north-east, through Saugur, Allahabad, and Goruckpoor, to the foot of the Himalayas. Throughout the whole of the plains to the east of this line cholera is endemic, the intensity of the disease increasing as we approach the sea-board of the Bay of Bengal, the cities of Dacca and Calcutta being pre-eminently the stronghold of this terrible malady. . . . Cholera is less frequently met with as we advance to the north-west and west from the line above indicated, until the disease may, with certainty, be said not to be endemic in the Punjaub, Rajpootana, and Sindh. But along the valley of the Nerbudda and Tafti rivers, and throughout a very considerable part of the Bombay Presidency, cholera is endemic." Dr. Bryden, on the other hand, roughly describes the endemic area as the region bounded on the east by the 91° or 92° of east longitude; on the west, by about the 85° , passing a little to the west of Patna; on the north, by the 27th parallel of latitude; and on the south, by the coast of the Bay of Bengal, including the deltas of the Ganges and of the Mahanuddy rivers, or from about Chittagong round to Porree. It forms a great basin, having the hill country east of the Bramahpootra river for its margin in that direction, and the Rajmahal and Cuttack hills for its western margin; while its northern limit is the Terai of the Himalayas, from Lower Assam, on the east, to the Terai of the Purneah district on the west. The details of the geographical distribution of cholera, with reference to the routes it has followed in its progress from its endemic area in India over the face of the earth, are of extreme interest; and it is of the greatest practical advantage to be familiar with them, so as to recognise them. "It is one of the most important subjects that can be studied in connection with cholera; for, by defining accurately the various outlets from India which have from time to time been taken advantage of by this insidious enemy of mankind, we may hope to bar its exit from its breeding grounds on future occasions." But beyond stating general results, the topic is much too long for a text-book; and therefore the student is referred to the most interesting accounts given by Drs. Macnamara and Bryden in their great works on cholera. From such works they will learn to form a sound judgment as to the true nature and habits of cholera. "One of the first things that strikes us in the 'interesting history given by Dr. Macnamara' is, that in every instance in which

cholera has extended beyond the confines of its endemic area, it has originated in an outburst of the disease in Lower Bengal or in Madras, as in 1833-34." Excluding the latter epidemic, it may be stated as a general rule, "that cholera has two well-defined routes from Bengal,—the one its westward course, along the basin of the Ganges into Nagode and Saugur, and from thence into the Bombay Presidency; the other, its north-western route, from Cawnpore (or up the Jumna) to Agra and Muttra, to Delhi, and so over the North-Western Provinces, into the Punjab. Cholera first appeared or was recognised in Central India, and thence extended to Bombay, in 1818, 1834, 1845, 1850, and 1864; and in 1820, 1835, 1846, 1850, and 1865, it had burst out either in the south of Persia, or along the sea-board of Arabia and the Red Sea, usually in both localities at the same time. From Mecca it travelled with the returning pilgrims into Egypt; and thence over Europe, in 1831, 1848, and 1865. In connection with outbreaks of cholera in Europe, Dr. Macnamara has traced the disease up the Persian Gulf, and the rivers Tigris and Euphrates, in 1823, 1846, 1852, and 1865. "It therefore appears very certain," he concludes, "that the course usually followed by cholera in its advance westward into Europe from India, has been either along the Red Sea (notably from Mecca) into Egypt, or else along the shores of the Persian Gulf, and up the Tigris or Euphrates into the north of Persia, or into Turkey in Asia, passing into Europe *via* Astrachan; or gaining Orfa and Aleppo, reaching the Turkish dominions in Europe. Its passage in this direction is impeded on account of the difficulty presented in the navigation of the Tigris and Euphrates, the country to the west of the former river being protected by the desert separating Bagdad from Syria. It is evident, also, that cholera has on several occasions travelled from the Punjab into Cabul, appearing at Herat; and thence extending to Mushed, Teheran, and Tabreez; or along the shores of the Caspian Sea. It followed this course in 1829 to 1845 (from Cabul), and also in 1853. Each of these years was followed by an outburst of cholera over the north of Persia, and by its extension into Europe over the Caucasus Mountains, or more commonly by the sea-route from Reshd to Astrachan." All the routes thus indicated by Dr. Macnamara are those *by which the natives of these various countries travel*. He mentions especially the *natives*, because it is quite certain that cholera has never yet reached Suez by means of the Peninsular and Oriental Company's steamers; although it has, without doubt, been imported by pilgrims passing up the Red Sea from Mecca in several instances. Mecca and Kerbela are extensively resorted to every year by pilgrims from India. (See "The Plague," p. 657, *ante*.) Besides this, there is a very considerable trade kept up between the various parts of India and Muscat, Makalla, Mocha, and Jedda; and still more extensive commercial relations are kept up between India and the various ports (particularly Bassora) on the Persian Gulf. And so, again, in the passage of cholera from the west and north-west of India, it evidently travels with human beings through Peshawer and the Kyber Pass; or from Dera Ismael Khan to the Kattawaz Plain, south of Ghazni—this being the great caravan route from the Indus valley to the Candahar Steppes. It also passes from Sinde, *via* the Bolan Pass, into Afghanistan. From this latter country the disease has on

several occasions travelled *via* Herat into Persia; and this is the only possible route it can take westward by land into Persia—the deserts of Rajpootana, and those again of Beloochistan, protecting Persia (as deserts always do) from the disease, except by way of Herat, or by sea from Bombay and the Persian Gulf (MACNAMARA).

But the history of cholera within the endemic area requires more careful study; and although there may be differences of statement as to the exact boundaries of the district of India where cholera is endemic, this much seems now to be accepted, namely,—that cholera comes into existence among the hot, moist vapours of the wet, undrained, uncultivated deltas of the Ganges and Brahmapootra. That it intensifies itself. That at first it scarcely attracts attention. That after a time it covers more ground, and becomes more fatal. That then it overflows its accustomed limits, and spares neither race, class, age, nor sex. That it becomes the deadly connecting link between the filthy inhabitants of the lowest classes in Bengal and the highest civilisation of the Western Nations (FLORENCE NIGHTINGALE). In the endemic area of cholera the army of Sanitary Reformers must therefore find their work in doing away with those conditions of unhealthiness which abound in India. Land drainage and cultivation must go hand in hand with the improvement of Indian villages, whose unhealthy conditions exist in the greatest intensity, as described by Drs. Macnamara, Smith, De Renzy, and Townsend. To the Health Officers of India, and to the Local Sanitary Commissioners, we must look for the “breeding-ground” of cholera being rendered a soil less productive of that disease. The education of the people, the cleansing out of their villages, the cultivation of the soil, the abundant and systematic supply of pure water, are the main directions for sanitary improvement.

More detailed information is required under the following heads, namely:—*Names of cities or Pergunnah towns of the district*, (1.) *In which cholera prevails epidemically*; (2.) *In which isolated cases of cholera occasionally occur*; (3.) *In which cholera never breaks out*. The only countries which, up to the present time, have remained free from cholera are Australia, New Zealand, and the other islands of the Pacific; the Cape of Good Hope and its adjoining settlements; the West Coast of Africa from the Cape as far northward as Gambia, including the islands of St. Helena and Ascension; the Azores, Bermuda, Iceland, and the Faroe islands; the Orkney and Shetland islands; and, lastly, the Western Coast of South America. The characteristic features of the countries exempt from cholera are, that they are at a great distance from India, separated from her by a wide expanse of ocean, and having very slight intercommunication with India. The exemption of these countries is strong presumptive evidence against the spontaneous generation of the disease in these places (Dr. GAVIN MILROY).

All the theories regarding the nature of cholera agree in two main points (or at least they are not at variance with them), namely,—that cholera is induced by a special poison, and that this poison is of foreign extraction, and not indigenous to this country.* The principal differences

* Our early knowledge of the progress of cholera in Great Britain was originally contained in an official report presented by the Commissioners to His Majesty King

between the recent epidemic (1866) of cholera in Europe and those of former years are,—(1.) For the first time in the history of the disease, Europe was invaded from the south. In 1829-32 and 1845-48 the disease spread from Persia to Russia, and thence along the Danube into Central Europe; and in Britain the towns first attacked on all previous occasions were seaports on the east coast. (2.) A remarkable feature in the epidemic of 1866 was its rapid and great extension along the coast line as compared with its slight and sluggish penetration inland. The central districts of Europe escaped altogether during 1865, except that there was an isolated outbreak in Saxony. (3.) The progress of the disease was much more rapid than in former epidemics. In 1829 cholera took fifteen months from the time of its entrance into Europe to reach Great Britain; two years, less one month, to arrive on the North American coast. In 1848 its diffusion occupied nearly the same period of time. In 1866 the disease had in less than five months spread from Alexandria to the coasts of the Euxine, and even to the Western hemisphere. A strong point in favour of the view that in 1866 cholera was introduced from the Mediterranean by ships coming thence is furnished by the fact that Southampton was the only port at which ships arrived having had cholera deaths on board shortly before reaching England. Moreover, it had been predicted that the disease would enter the country by Southampton, and not, as before, by towns on the east coast; and the fulfilment of the prophecy will seem to many, as Dr. Parkes observes, sufficient evidence that the outbreak at Southampton arose in this way (*Sydenham Society Biennial Retrospect*, 1867, p. 494). But England seems to have been infected in 1866 at many different points of invasion, and from many different directions, almost at the same time (J. SIMON, F.R.S.) For example:—

“On the 28th of April, 1866, a first case was reported from Bristol—that of a trader who had arrived there sick from Rotterdam. On 15th May telegrams from Liverpool and Birkenhead reported that the disease was prevailing on board certain vessels in the Mersey, among German and Dutch emigrants, who, with a view to crossing the Atlantic for New York, had come in flocks, travelling rapidly from the Continent, often from infected parts, by way of Hull, Grimsby, and other of our north-eastern ports, and had now fallen ill at their port of embarkation. Much alarm was occasioned by this outbreak; the more as new arrivals of the same sort were occurring from day to day. The outbreak, so far as England was concerned, soon came to an end; but the subsequent progress of the emigrants was unfortunately not unattended by cholera. Indeed, in several cases, vessels such as the above, leaving in apparent health, suffered during their voyage cholera deaths among their passengers and crew, and were, of course, very dangerous arrivals for their port of destination. Within the next few days after the 15th, my Lords of the Privy Council were apprised of the first two cases of what afterwards became a serious epidemic at Swansea; and single cases in various other parts of the country were also notified to them. Anxiety became general in the William IV., of which only one copy existed. This was rescued from oblivion by the late Sir James Clark, aided by the Royal Librarian, who found it in a drawer, buried among a heterogeneous mass of papers. It is now appended to Dr. Graves's *Report on the Progress of Cholera*.

country; and there was much correspondence with local authorities, often on precautions to be taken against the disease, or provisions to be made for treating it, and often on questions of jurisdiction and responsibility. On 15th June the Peninsular and Oriental Company's steamship 'Poonah' arrived at Southampton with a case of cholera on board, and several other cases had appeared in the town. On the 29th two deaths were reported by telegram to have happened at Goole, whither clearly the disease had been imported from Antwerp. On the 30th three deaths were reported to have occurred at Northwich, in Cheshire, and on the same day a case of cholera occurred at Shields, on board the 'Clio,' from Hamburg. On 3rd July a case was reported to have happened at Harwich, on board the 'Redstart,' from Brussels; and on the same day, from Brixham, the death of the captain of a coasting vessel was reported. A serious extension of the disease was imminent. Reports of new centres of infection became more and more frequent; and on 14th July, 1866, the time had come for putting the Diseases Prevention Act in force throughout the whole of England and Wales, by which ample powers of medical relief (not restricted to paupers) were exercisable by local authorities throughout the country; and on the 18th July, the first cholera death in the metropolis was reported from Poplar. Two days afterwards there was already an alarming proportion of cholera cases in parts of East London; and on the 21st the Secretary of the London Hospital reported that the resources of that most useful institution were being overtasked by such claims for admission as attested a very terrible epidemic of cholera."

(4.) This swift propagation of the epidemic does not appear to have been dependent on any peculiar virulence of the disease (N. RADCLIFFE). At the same time diarrhoea of so severe a form as to be called "choleraic," preceded the epidemic outbreak in 1865 and 1866; and if it is found impossible to disentangle, at the beginning of an outbreak, cases of *simple cholera* (p. 795, *ante*) from those of a *true* epidemic character, and to shut out absolutely a theory of the development of epidemic cholera by gradation out of *simple cholera* or severe diarrhoea, it is equally impossible to set aside the fact of exposure of the metropolis and busy ports of embarkation and debarkation (like Southampton and Hull and Liverpool) to continuous transmission of the epidemic malady from the early autumn of 1865 to the early summer of 1866 (RADCLIFFE).

These apparently distinct outbreaks in several towns and localities, however isolated, must all be regarded as forming parts of one general epidemic; and the histories of them, so ably set forth in the various reports collected together by Mr. Simon, Dr. Parkes, and others, compel the conclusion that—the *chief agents in the dissemination of the epidemic of 1866 have been the sick from the malady in its slighter, as well as more marked and characteristic forms*—a conclusion which has been adopted absolutely of epidemic cholera by the International Sanitary Conference, which met at Constantinople to consider the question of the preservation of Europe from this pestilence. The history of the epidemic in its entirety in this country points to the transmission of the disease to the metropolis and other ports from localities previously visited by it in Western Europe. Mr. Simon's dictum, that "*contagious currents on the Continent of Europe must be deemed virtually contagious currents in England,*"

is to be accepted as an axiom in State Medicine, notwithstanding that links of transmission may fail to be discovered (*Ninth Report*, p. 288). The testimony of Dr. Macpherson also is to the effect that, whatever cholera we have had in Europe in former times, since 1817 at least has been always carried out of India, or Persia, or Arabia, to other places.

"The outbreak of cholera in the metropolis, in 1866, cannot well be considered apart from the wide diffusion of the disease on the Continent of Europe during 1865 and 1866. It is inextricably linked, both chronologically and etiologically, with that rapid dissemination of the malady which, in May of 1865, commencing at the most sacred city of Mohammedanism, Mecca, extended to Egypt, and thence, before the close of the summer, to many places on the eastern and southern coasts of Europe, and in the basin of the Mediterranean. During the autumn the epidemic spread largely in the south of France and in Spain. It appeared at Altenburg in Saxony (where it was introduced from Odessa), and extending to several neighbouring towns, broke out with severity in Paris, and infected slightly our own coast at Southampton (RUDOLF, GÜNTHER). From the 24th September to the 4th November, thirty-five individuals succumbed to the disease in the last-named seaport town; and from the 28th September to the 31st October, nine deaths occurred from cholera (an offshoot of the Southampton outbreak) at Theydon Bois, in Essex, a hamlet lying about eleven miles in a direct line N.N.E. from Bowbridge. As the winter of 1865-66 advanced, the epidemic extended to north-western France, chiefly affecting the departments of Finisterre, Morbihan, and Côtes du Nord; and throughout the cold season it manifested more or less activity along the opposite coast of the channel. In the north-east, the department of the Vosges received the infection. With the increasing spring the disease became more rapidly disseminated. In several localities of Belgium and Holland it early showed itself. As the summer grew, and its mid-season approached, the diffusiveness of the epidemic augmented largely. The malady re-appeared in several cities and towns of Eastern, Southern, and Western Europe which had suffered from it the previous year; it spread generally throughout the provinces of Belgium and Holland; and extended widely in Prussia, Central Europe, and European Russia. The epidemic broke out in Rotterdam prior to the 21st April; in the port of Antwerp on the 19th May; in Stettin before the 2nd June; in St. Petersburg on the 26th June, or somewhat earlier; in Memel before the 10th July; and in Dantzic before the 12th of the same month" (*Ninth Report to Privy Council*, by Mr. SIMON).

(b.) *Conditions which hinder, and conditions which favour the Propagation of Cholera.*—It is stated by Macnamara that a desert is a district in which cholera is unable to establish itself. It never spreads beyond three stages into a desert. Those of Arabia and Syria have been the most effectual barriers to the propagation of cholera from companies of pilgrims coming there. A great desert is the best of all obstacles to the propagation of cholera (Constantinople Cholera Conference). So, also, the suppression of the usual rains is known to have brought the progress of cholera to a stand-still. There are also several characteristic features of Asiatic cholera, especially considered at length by Dr. Macnamara, which require to be enumerated here—the student being again referred to his excel-

lent work for more extended information. These features are as follow:—(1.) *The unequal and very partial distribution of cholera out of its endemic area.* In this respect it is allied to influenza and to the plague. Its history in Indian jails is an example. Prisoners may be free from the disease while it is raging outside the walls of the prison. This characteristic may be an argument against any general atmospherical or epidemical influences being a cause. (2.) *The inhabitants of certain districts being specially liable to be visited by cholera, these localities have features in common with one another, differing from other places which have usually escaped its influence.* It has been most destructive in large seaport towns built on low-lying alluvial soils, at the mouth of rivers; and, as Dr. Farr has shown, there is an inverse ratio between the mortality and the elevation of the dwellings above high-water mark. The lower the land the greater the mortality from cholera. (3.) *It by no means, even in the majority of instances, attacks the inhabitants of the most insalubrious localities even in our large towns.* While many habitual seats of fever in such towns are visited by cholera, many of the worst fever-nests in the whole metropolis are unaffected by it. (4.) *No amount of overcrowding, no special conditions of the soil, nor any circumstance with which we are acquainted, has ever been known to originate Asiatic cholera de novo among men removed from its endemic influence, or, unless the disease has been epidemic at the time, beyond the confines of India.* The disease has been traced to the Mauritius on several occasions, but always after the arrival of vessels from India with persons on board who had been among those suffering from cholera. The same thing has occurred in America, Guadeloupe, the islands of the Grand Canary group, and so on. A direct chain of cases always connects an outbreak of the disease in this country with cholera in British India. But it is not always possible to trace cholera immediately from man to man in its progress over the world, and this for two reasons, namely:—(a.) Because our evidence must frequently be wanting in precise data; (b.) Because articles of clothing, or goods soiled with the dejecta of cholera patients, are capable of propagating the disease. (5.) *The intensity of cholera varies during its continuance in a county or a large town, so that it has periods of little and great activity—in fact, usually well-marked periods of increase, culmination, and decline.* As a general rule, the first outburst of the disease is the most malignant; so much so, that ninety-six out of ninety-eight may die. It declines more gradually than it commences, and often exhibits periods of renewed activity. These becoming fewer and less virulent in the course of time, the malady finally disappears from the infected locality. (6.) *After having been a certain time epidemic in a locality, it ENTIRELY disappears, unless in its endemic area.* Passing out of India, it reaches Europe or America within the usual period of time (according to the route by which it comes), and after exercising its baneful influence for two or three years, it gradually dies out and disappears, until again rekindled by a fresh importation of the disease from India. It is capable, however, of a long-continued existence if fostered by a suitable climate. It may live and bear fruit even in the cold of a European winter, if fostered in carefully warmed conservatories, such as the overheated houses of the Russians. The imported disease is terribly prolific and fatal in its first year's growth on a new soil; but from that time it deteriorates

in its power of destroying life, and gradually declines. The counterpart of this characteristic is seen in the case of newcomers into a locality under the influence of cholera. (7.) *Every outbreak of cholera beyond the confines of British India, may be traced back to Hindostan, through a continuous chain of human beings affected with the disease, or through articles stained with their dejecta.* "In this characteristic, cholera has been seen invariably to follow the routes by which man travels; and if it has thus spread from country to country by his agency, then we may fairly assume that it has extended continuously from man to man. Cholera has never been known to extend from one place to another faster than man can travel. Whatever the specific poison is, it never appears to spring up in a new locality, unless introduced by men or articles of clothing from an infected place." This is the first elementary fact to be accepted and inculcated in the pathology of Asiatic cholera. (8.) *The more explicit the examination the clearer the fact appears;—that, the disease, in the majority of cases, spreads from one human being to another by means of the cholera fæces contaminating the drinking water, and thus getting into the intestines of other people.* "So surely as water contaminated with cholera dejecta is capable of reproducing the disease when consumed during the vibronic stage of decomposition, so certainly it may be drunk with impunity after this stage is over, and when various forms of ciliated infusoria have replaced the vibriones." Not that the vibriones have any influence in inducing cholera in the human body, but they indicate that the organic matter "in the water is passing through a certain stage of decomposition, during which process it seems capable of imparting a similar action to the epithelium of the intestinal canal." Positive evidence is given by Dr. Macnamara of water contaminated with cholera evacuations being a medium of the communication of cholera from one person to many others, during the epidemic of 1861, in the Punjaub. The most positive evidence exists in this case as to the fact of fresh cholera dejecta having found their way into a vessel of drinking water, the mixture being exposed to the heat of the sun during the day. Early the following morning, a small quantity of this water was swallowed by nineteen persons (when partaken of, the liquid attracted no attention, either by its appearance, taste, or smell). They all remained perfectly well during the day—ate, drank, went to bed, and slept as usual. One of them, on waking next morning, was seized with cholera; the remainder of the party passed through the second day perfectly well, but two more of them were attacked with cholera the next morning; all the others continued in good health till sunrise of the third day, when two more cases of cholera occurred. This was the last of the disease: the other fourteen men escaped absolutely free from cholera, diarrhoea, or the slightest malaise. Cholera was not prevalent in the place. It had not visited the locality for several years, nor has it appeared there since. Examined microscopically, the surface of the fluid was found covered with numerous large vibriones, and showed that the organic matter in the water was in the vibronic stage of decomposition.

But there are other and occasional circumstances which facilitate and give energy to the spread of the cholera poison, although they may not be able to generate or reproduce the disease independent of the existence of the specific poison. The observations made by Drs. T. R. Lewis and

D. D. Cunningham on cholera in relation to certain physical phenomena are the most systematic, extensive, and conclusive that have yet been made in India. They relate to the influence of conditions of the soil on the prevalence of cholera, and embrace the physical conditions of localities associated with the seasonal prevalence of cholera in them. Their results are embodied in the following statements:—(1.) With regard to the seasonal prevalence of cholera in an endemic area (Calcutta), April appears to be the month of maximum prevalence; and November most nearly represents the average prevalence of the disease, and is therefore taken as a good starting period for comparison, from which successive diminutions in prevalence are noted during December and January—a rapid rise in February, continuing to the maximum in March and April; a marked diminution in May, continued through June to a minimum in July, August, and September; and, finally, a rise in October to reach the average in November. Stating the facts of prevalence generally, it may be said that there are four months in which the prevalence of cholera greatly exceeds the average, three months in which it falls far short of it, and five months in which it ranges round it; the prevalence in November approaching it more nearly than that of any other month. (2.) An inquiry is then made into the meteorological and other physical conditions coincident with them, in order to determine whether any connection be traceable between the two series of phenomena;—whether, in fact, there be *any series of meteorological and physical constants characterising the various seasons of prevalence*. The conditions selected for consideration are—(a.) Atmospheric pressure; (b.) air-temperature; (c.) atmospheric humidity; (d.) rainfall; (e.) water-level; (f.) soil-temperature; and, (g.) amount of carbonic acid in the soil-air, mainly regarded as an index of soil-ventilation. Of these in their order:—(a.) As regards *atmospheric pressure*, it is shown that the season of minimum prevalence is characterised by low atmospheric pressure; but further than this the coincidence ceases. There is no indication of the existence of any definite relation between the degree of atmospheric pressure and the prevalence of cholera, on which it cannot be regarded as exercising any direct influence. The coincidence of low atmospheric pressure with minimum prevalence must be regarded as a coincidence merely; or, if any influence be exerted, it must act through some intermediate agency. (b.) As to *atmospheric or air-temperature*, it is shown that if it exerts any influence on the variations in the prevalence of cholera in Calcutta, it does so only in a very subordinate way; for maximum, minimum, and medium prevalence occur with an almost unaltered temperature. Taking November as the starting point, there are two months of diminishing temperature and prevalence, followed by three months of increasing temperature and prevalence. But in considering data regarding other conditions, it is found that some disturbing influence manifests itself during December and January, breaking in on the coincidence between the prevalence of cholera and those conditions which otherwise correspond in their fluctuations most accurately with those of the disease, and there is hardly anything else to which this can be ascribed save temperature. Taking this into consideration, together with the well established fact of the general tendency to subsidence, or even disappearance of the disease during the winter months of periods of its

endemic manifestation in Europe, there appear to be grounds for ascribing some influence to atmospheric temperature on the prevalence of cholera in Calcutta. The precise method in which it acts, however, remains undetermined. That it acts directly is extremely improbable; but there are many indirect means by which it may produce an effect; and whatever the latter may be, they must, at all events, be entirely independent of peculiar habits of life of one section of the community as compared with another; for we find as marked a decrease in prevalence among European troops as among the native community. (c.) As to *atmospheric humidity*, it is shown that there is a certain amount of coincidence between diminished humidity and increased cholera prevalence in Calcutta. From data extending over eight years, it appears [that, starting from November, we find a considerable decrease of humidity in December, continuing until February, and followed by a slight fall to the minimum in March. A considerable increase occurs in April, followed by a smaller one in May; next follows a rapid increase through June and July to the maximum in August. This is followed by a slight fall in September, succeeded by a considerable fall in October, and an even greater one in November. These data are confirmed by others extending over another period of twelve years; and, on comparing those data with the periods of cholera-prevalence, we find the periods of maximum prevalence and of minimum humidity, and of minimum prevalence and of maximum humidity—coinciding very closely. We have maximum prevalence and minimum humidity in March; and minimum prevalence and maximum humidity in August. There is thus a general coincidence of the phenomena of seasonal prevalence in Calcutta with those of the seasonal fluctuations in atmospheric humidity. The maximum and minimum periods hold a reverse relation; there is a rapid rise in cholera coincident with an equally rapid fall in humidity during October and November, and a similar phenomenon of coincident fall in prevalence and rise in humidity occurs in May and June. The greatest want of coincidence occurs in December, January, and April. In December and January, as compared with November, there is diminished prevalence coincident with diminished humidity; and, in April, compared with March, there is increased humidity coincident with a cholera prevalence, which the data of Lewis and Cunningham teach them to regard as increased in place of diminished. (d.) As to *rainfall* (extending over a period of forty-seven years, compared with cholera prevalence), it is shown that the correspondence between special phenomena of prevalence with special periods of rainfall is much less distinct than that between the phenomena of atmospheric humidity and of prevalence. Beyond the fact that the three months of minimum prevalence correspond with three of the period of maximum rainfall, there is nothing indicating any special relation, either direct or inverse, between the two phenomena. There is, in fact, nothing to justify the belief that rainfall in Calcutta exerts any direct action either in producing or diffusing the essential cause of cholera; but, on the other hand, there is some evidence that excessive rainfall exerts a directly opposite action. (e.) As to *level of soil-water*—the average monthly water-level in the soil for six years is compared with the cholera prevalence, with the result—that a maximum cholera prevalence is found to be associated with a low level of the soil-water;

but that the absolute water-level is, in itself, of no importance; and mere depression of water-level, mere diminution of the bulk of the water in the soil, is insufficient in itself to secure prevalence. (f.) As to *soil-temperature*, a comparison is made of average monthly soil-temperature for three years, at a depth of six feet below the surface, cholera prevalence, and air-temperature, in order to allow of ready comparison of the relations of the air above and within the soil in this respect. The data regarding the soil-temperature represent the conditions of a stratum towards the lower portion of the layer of soil, which lies permanently above the soil-water. The mean temperature for the year is 78.5° Fahr. The minimum soil-temperature occurs in January; the maximum in June and July. The soil-temperature exceeds that of the atmospheric air in the months of November, December, and January; falls far short of it during March, April, and May; and is almost equal to it during the remaining months; and in so far as conditions of temperature are concerned, soil-ventilation is favoured during November, December, and January; obstructed during March, April, and May; and almost in equilibrium during the remaining months. Comparing the data of soil-temperature and cholera prevalence, the maximum of prevalence in April and the minimum elevation in November both occur when the soil-temperature is between 78° and 79°; and here the coincidence ceases. The only other condition in which they tend to agree is the atmospheric humidity. The period of minimum prevalence occurs along with that of maximum elevation of temperature; but the same elevation extends beyond it in both directions, commencing and terminating in June and October—two months of medium prevalence. If temperature really exerts any influence, that of the soil must be considered as well as that of the air above it. (g.) As to the *carbonic acid of the soil-air*, as a *measure of soil-ventilation*, the observations show that it is excessive during the rains. During November, December, and January the amount is high. In February a considerable decrease occurs; and the minimum for the year is reached in March and April. During May a slight increase occurs, continued through June and July, followed by a rapid rise in August to the maximum in September, after which a decrease occurs, reducing the average for October to an equality with that for August, on the one hand, and for November, December, and January, on the other. These fluctuations are to be regarded as an index to the varying degrees of soil-ventilation present at different times of the year—an index to the varying degree in which emanations escape from the soil into the atmosphere. These fluctuations in the amount of carbonic acid in the soil-air are due to one or other of two causes—(1st.) Variation in the amount produced at different times; (2nd.) Variation in the degree of accumulation of what is formed—*i.e.*, variation of the amount retained in the soil. This latter agency mainly determines the phenomena of fluctuation in Calcutta. The maximum prevalence of cholera is found to coincide with maximum of soil-ventilation; and the minimum of prevalence with obstructed soil ventilation. In estimating the influence of the rainy season on condition of health, its action in effecting soil-ventilation cannot be left out of consideration; and the prevalence of any influences obstructing or facilitating the escape of emanations deserves careful consideration in any attempts at the expla-

nation of the phenomena of disease as related to season. Hitherto rain has only been regarded as affecting health through the agency of the water supply, or of its action in washing the surface of the soil; but, its relation to soil-ventilation has almost entirely escaped notice. (3.) *The physical characteristics* of the different seasons of cholera prevalence in Calcutta may be summed up as follow:—The season of minimum prevalence is characterised by low atmospheric pressure, high atmospheric and soil-temperatures, extreme atmospheric humidity and rainfall, elevation of the water-level, and obstructed ventilation of the soil, as indicated by the amount of carbonic acid in the soil-air. The season of medium prevalence is characterised by high atmospheric pressure, low atmospheric and soil-temperature, minimum humidity and rainfall, depression of the water-level, and obstructed ventilation of the soil. The season of maximum prevalence shows characters occupying an intermediate position in regard to those of the other two seasons, except in so far as water-level and soil-ventilation are concerned—the former being at a maximum of depression, the latter at a maximum of activity. The depression of the water-level and the increase of soil-ventilation, are the only phenomena which reach a climax during the season of maximum prevalence of cholera. Then, as to London, the initial activity and rapid development of the outbreak of 1866 was preceded and accompanied by an excessive range of temperature. The mean temperature of the week ending the 30th June, in which the earliest cases of cholera occurred, was $4\cdot8^{\circ}$ above the mean of the same week on an average of fifty years. During the next week the mean temperature fell $5\cdot1^{\circ}$ below this average; but in the following week the mean was in excess $6\cdot3^{\circ}$. In the first third of these weeks the range of temperature was $24\cdot0^{\circ}$; and in the second $17\cdot5^{\circ}$. During the three months of principal prevalence of the outbreak of 1866 in the metropolis (July, August, and September), the atmospheric pressure was remarkably low. From the 26th of July to the end of the quarter the barometer, reading at the height of 160 feet, never reached the point of 30 inches—"a most rare occurrence," as Mr. Glaisher writes.

But in order to give character and energy to the development of cholera, there are other conditions required besides meteorological. These other conditions are described by Dr. Barton as the "*terrene element*," and correspond with what have been termed the "*localising causes*" of cholera, especially as developed in the theory of Pettenkofer, and in some of the results just noticed regarding soil-ventilation and soil-moisture. That some local circumstances play a very important part in the evolution of cholera is evident from the following additional facts:—

(1.) An analysis of the history of cholera epidemics shows that they are most frequently made up of a succession of partial local outbreaks, not only in different districts, but even in the same place. (2.) The pestilence has also been observed to linger in some few favourite haunts throughout the entire course of an epidemic; and that, now and then, after visiting a place at the commencement of an epidemic, it has returned to it again, after an interval of complete immunity, before its close. (3.) That some places escape an epidemic visitation at the very period when others in the immediate vicinity are suffering severely from its

presence, the meteorological influences being the same. Even in the same town, whilst the inhabitants of some streets or courts are being decimated, those dwelling in others not far distant altogether escape, or (as frequently happens) the inmates of certain houses suffer severely, whilst their neighbours are entirely spared. (4.) That the limits of a tainted district are sometimes clearly marked out. In illustration of this, "*one wing of a cavalry regiment, just arrived from England, and in high health, ascended the Ganges from Calcutta in boats, there being no cholera at the time in Calcutta. At a certain period of the voyage the troops arrived at a part of the country where cholera prevailed in the villages on the banks of the river, but with which they did not communicate. Here cases of cholera occurred in the boats; the men were advised to push on rapidly, and after a few days, when they had passed the limits of the existence of the disease on the banks, it ceased to show itself in the boats. What makes the case peculiarly conclusive is, that the other wing of the regiment followed afterwards by the same mode of conveyance, became affected with the disease at the same point, and lost it again at the same point.*" (PROFESSOR W. P. ALISON on "The Exciting Causes of Epidemics," in *The Medico-Chirurgical Review* for 1854.)

The geological feature of the surface-soil characteristic of the endemic area of cholera—the Gangetic plains—is alluvial, consisting chiefly of a mixture of firm sand and clay with decayed animal and vegetable matter—loam, very much like the silt that settles from muddy river water. Below this, at a distance of from 6 to 10 feet, comes a bed of stiff clay; and below this again, a layer of peat, resting on alternating layers of sand and clay (H. F. BLANDFORD, quoted by LEWIS and CUNNINGHAM).* The nearer the soil of a district, in India, approaches in character that now stated as constituting the lower portion of the Gangetic plains, the greater is the likelihood that cholera will be an endemic disease in it; and this general statement regarding endemic cholera is not merely applicable to the districts which occupy the Gangetic plains of Lower Bengal; but also to certain parts of Oudh and the North-Western Provinces, as well as to similar plains which have been formerly the silt of the Godavery, the Mahanuddy, the Brahmapootra, and the Cauvery—in all of which areas cholera is more or less distinctly endemic. Lewis and Cunningham have also shown that the seasonal manifestation of cholera changes gradually as we proceed up the river, the disease manifesting a tendency to be deferred later and later in the year the farther the affluents of the Ganges are followed towards their sources, and the drier the climate and the soil become; so that in the upper part of India and in the Central Provinces, the maximum prevalence of the disease occurs just at those periods when the soil conditions most closely approach those in Calcutta when in its driest state—namely, during and towards the end of the rains, at which period alone the soil conditions of the former area approximate to those in Lower Bengal during the greater portion of the year. After bestowing the most careful consideration on this matter,

* Many years ago a well was sunk in Calcutta, to a depth of 481 feet, through successive layers of sand, clay, peat, and pebbles; that at 380 feet was a layer of fresh-water shells, resting on a bed of decayed wood, indicating that this must at one time have constituted the surface, but that it has since sunk and been covered by a soil formed by deposits from a river.

and after endeavouring to examine it in all its phases, Drs. Lewis and Cunningham have come to the conclusion "that the theory of cholera which regards conditions of the soil as essentially determining the *production of the cause of cholera* in a locality is much more in accordance with the phenomena of its seasonal prevalence, as manifested throughout the Bengal Presidency, than any of the other doctrines appear to be." But large plains of laterite also crop up, as in the Orissa, Midnapore, and Beerboom districts; or the soil consists of beds of clay, of a yellow colour, strongly coherent, and abounding in *runkur*, on which Patna, Dinapore, Benares, and Mizapore are built. An idea prevails that cholera is less liable to occur in lateritic districts than in others in India—a matter deserving the attention of those who have the opportunities of inquiry. *Lateritic soil* is a compound of clay and oxide of iron, very porous, and possessing the property of hardening on exposure to the atmosphere (LEWIS and CUNNINGHAM).

Although very great differences of opinion thus prevail as to the part which obvious local causes bear to the production or spread of cholera, yet it is almost universally considered that they are necessary for the development and propagation of this disease in its epidemic forms. M. Bayer, in 1832, followed by Drs. Barton, Carpenter, Pettenkofer, and Snow, all agree in this general proposition. Dr. Pettenkofer, of Munich, maintains,—(1.) That a certain condition of soil is necessary to the local development of epidemic cholera. The soil must be a stratum of earth, inhabited by men, pervious and permeable to water and air, so that the air in the soil bears a relation to the air above it. The air in the pores of the soil is constantly kept moving by the force of the wind passing over the surface of the ground; and by the laws regulating the intermingling of gases—change of temperature, diffusion, and so forth; while gravelly and sandy soils act much more quickly than those of mud and clay. (2.) It must possess a particular degree of *humectation*, depending upon the position of the subsoil water to the surface; but a considerable fluctuation, temporarily, in the degree of humidity of this stratum is necessary; which fluctuation shows itself in the simplest and surest manner by the difference in the level of the subterranean waters, the most dangerous moment being when the level of the water sinks after having attained a considerable height. (3.) It must be charged with organic, especially excrementitious, matter. These he believes are essential conditions, *as to soil*, for the development of epidemic cholera. But, (4.) The nidus, or cholera poison, or cholera *ejecta*, once finding admission to such a soil, undergo those developmental changes which are characteristic of its rapid dissemination as an epidemic. Such a condition of soil fosters the multiplication of the cholera poison. It is not alleged that it generates the poison *de novo*, but by means of the evacuations of those sick of the disease—or even the evacuations of men who are healthy, but who came from infected places, produce the same result—the fermenting contents of choleraic intestines find their way into such soils. Thus Dr. Pettenkofer believes that two elements are indispensable for the production of cholera,—(1.) The importation of the choleraic germ into the locality; the nature of which he does not define or characterise in any way. (2.) A peculiar constitution and condition of the

soil. Neither the first nor the second is sufficient of itself; but the simultaneous action of both is necessary to produce certain emanations which give rise to choleraic infection. Apart from the local conditions as to soil insisted on by Dr. Pettenkofer, there is very strong evidence to show that the decomposing choleraic discharges (already in a state of active fermenting change in the intestine of the cholera patient) will alone produce the disease—which will go on changing and multiplying a virus capable of spreading through the air as well as through the medium of water. In the outbreak at Southampton in 1866, there were instances both of transmission by water and by air (PARKES, in Mr. SIMON'S *Ninth Report on Public Health*, p. 253). The discharges need not pass into the ground to decompose or ferment there. They may decompose equally well in sewers, and, of course, can propagate their specific fermentation to the contents of such sewers, and such contents may find their way as gases, or more material elements, in the air or in the water. *Impure water, lowness of site, and the emanations arising from the decomposition of animal excreta*, are the local conditions now satisfactorily determined to have a more or less constant connection with the development and propagation of cholera. That *impure water* has a powerful influence over the intensity of cholera outbreaks is now unquestionably established by the observations of Drs. Acland, Sutherland, William Budd, the late Drs. Parkes and Snow, the specific inquiries of Dr. Farr, as represented by the Registrar-General, and lastly, Mr. Simon. Yet still it is found that *impure water* is not a necessary element in the generation of the cholera poison, as shown in the report of Dr. Baly (pp. 201-205), Budd as to Bristol, Pettenkofer as to Munich, and Günther as to Saxony. From their evidence "cholera can do its very worst where the drinking water can play no possible part in its dissemination" (*Brit. Med. Journ.*, April 13, 1867, p. 416). The localised attacks at Theydon Bois in 1865, in the east of London and in Southampton in 1866, all point unequivocally to impure water. With the *general outbreak* at Southampton, however, impure water had nothing to do. It had only to do with the production of the disease on board the steamship "Poonah," just before her arrival from Gibraltar, where she took in a tankful of very foul water, which, from its peculiar smell, evidently contained sewage (PARKES).

As regards London, it has been shown by Dr. Farr that *the elevation of the soil* has a more constant relation to the mortality from cholera than any other known element, the mortality from cholera being in the inverse ratio of the elevation. Yet, like the condition of the water, the elevation of the soil has not been always found to be a necessary localising condition; and there is now only left to be noticed *the influence of an atmosphere contaminated by the effluvia arising from decaying animal matter and excreta*. Dr. Cullen long ago remarked (and every industrious dissector knows), that the effluvia from very putrid animal substances readily produces diarrhoea. Yet it appears that the nature of the decomposing matter, and of the transforming process it undergoes, have some influence in modifying the effects on the human constitution. Districts in which the most putrid odours tainted the air have sometimes almost entirely escaped, whilst others contiguous to them have suffered severely. In dissecting-rooms, where the process of animal putrefaction goes on to a

great extent, diarrhoea is comparatively rare, if the rooms are kept clean. During my experience as Demonstrator of Anatomy in the University of Glasgow, for a period of six years (including the severe epidemic of cholera there in 1848-49, and during which time almost all the subjects for dissection had died of cholera), not a single student suffered from cholera; and when the proper agents are used, such as the injection of arsenical solutions into the dead body, which have the effect of arresting and modifying the putrefactive changes, I believe the production of diarrhoea is an exception, and may be found to have as significant a cause in errors of eating or drinking as in too close an attendance in the dissecting-room. That the poison of cholera does not attach itself to the *dead body* (in a certain state of decomposition at least) is a fact confirmed by the experience of those connected with the dissecting-rooms in Edinburgh and Glasgow. It is certain that these were supplied during the greater part of 1848-49, as they were in 1832, almost exclusively by cholera subjects, and in neither years was there a single case of the disease among the numerous students attending these rooms (Dr. ALISON).

Much pains has been taken by Dr. E. H. Greenhow to investigate in this country the precise conditions which, from their more uniform co-existence with cholera, might be supposed to produce or to aggravate epidemics of it. The result of his observations tends to confirm what Mr. Orton (*London Med. Gazette*, Vol. X., p. 222, 1832) was the first of English writers to show—namely, that “an atmosphere impregnated with the products of *fermenting excrement* is at once the most obvious and most constant concomitant of cholera (*the privy or fecal contamination theory*). Such exhalations were often found, even in a concentrated form, in houses where the existence of any palpable cause of insalubrity would scarcely be suspected, and thus the fact is in some measure explicable, that the pestilence, sometimes passing over slums and rookeries, knocked at the door of the comfortable annuitant or the wealthy tradesman. It was found that persons appeared to suffer in proportion to the contamination of the air they breathed with the ‘*privy odour*,’ and that immunity from this appeared to secure immunity from cholera.” The observations of Mr. Orton and of Dr. Greenhow are confirmed by the investigations of Dr. Pettenkofer at Munich and at the village of Gaimersheim. Dr. Barton, of New Orleans, Dr. Milroy in his report on the epidemic at Kingston, Dr. Buckler in his account of the outbreak in the Baltimore almshouses, and Dr. Parkes at Southampton;—all give similar evidence confirmatory of the influence of the *fermentive decomposition of animal excrement* as favourable to the spread of cholera. The outbreaks of cholera in some of the camps in Bulgaria and the Crimea, especially at Aladyn and Alma during the war, also furnish sufficient illustrations; and I believe the outbreak of cholera at Scutari, in November, 1855, which suddenly commenced in the camp of the Osmanli Horse Artillery, was similarly influenced as to its spread.

(c.) *Propagation of Cholera by Human Intercourse*.—When cholera appeared in its epidemic form in this country in 1831, the majority of European practitioners were decided contagionists. Subsequently to that period a reaction of opinion occurred, and the question was discussed for many years without any definite result. In 1848, when the disease

again became epidemic, many of the higher authorities coincided with "the solemn declaration of the Board of Health, that the malady was not in any way contagious, and that no danger was incurred by attendance on the sick. "A large body of evidence, however, now renders it certain that human intercourse has at least a share in the propagation of the disease, and under some circumstances it is the most important, if not the sole, means of effecting its diffusion" (Dr. BALY). Very positive evidence has now accumulated in abundance to prove the transmission of the disease by human intercourse. Healthy men carry the disease with them by their clothes, by their ships, and by their caravans. That such is the case, we have now ample evidence in the *Bengal Report*, of 1824, by Dr. Jameson; in cases related by Mr. Orton, in 1832, and by Dr. J. Y. Simpson, in 1838; in *The Edinburgh Monthly Journal* for 1849, by the late Dr. Cruickshank at Dalmellington, in Ayrshire; by Dr. William Robertson, detailed in *The Edinburgh Monthly Journal* for August of that year; and more recently the account of the outbreak at Arbroath, in Scotland, in 1853, by Dr. T. Trail; and cases by Dr. Alison, in 1854, in the paper already noticed; in the report of Dr. Berg, of Stockholm, in 1848; in the Norwegian Reports of 1850-53; in the Report of the College of Physicians of London, in 1854; and, the several reports of 1865 and 1866. (See especially also the more recent works of MACNAMARA and BRYDEN, and the reports of the various Indian Sanitary Commissioners during the past decade for evidence of a similar kind). These records afford undoubted instances which show that human intercourse is occasionally influential, in some way, in transmitting cholera into detached localities, where it may seize upon two or more individuals, and then cease, or spread as an epidemic.

But it is no less certain that its general extension over the world cannot be accounted for by human intercourse alone, to the exclusion of aerial contamination. It is curious that in India, the birthplace and headquarters of the disease, the doctrine of contagion is almost universally disbelieved in by our professional brethren. The opinion generally entertained in India is indeed opposed to the doctrine of contagion (MOREHEAD, *Indian Annals of Med. Science*, Vol. I., p. 456). On this important point Lewis and Cunningham show that the normal course followed by the fluctuations in the prevalence of cholera in Calcutta is not merely inexplicable by the theory of contagion; but is strongly opposed to it. Such difference of opinion may admit of explanation, in the fact that in India all the causes of cholera in its original home are in constant operation, and hence, no sooner does the specific poison of cholera become active, than the disease spreads with such rapidity as to resemble an epidemic invasion; and so long as the conditions supposed to secure development of the poison are not distinctly defined, it is evident that an endemic locality affords little field for the "soil-theory" in regard to the relation of prevalence of the disease to the influences calculated to favour its production. Cases, however, of undoubted contagion are not wanting in India. Mr. Barry, of the Bengal service, records an outbreak of cholera at Gowalparrah, in Upper Assam, in 1853. In this instance cholera was evidently imported into a healthy place by a body of Sepoys coming from an infected locality. Every case of the disease could be traced to com-

munication with the sick: a large number of attendants on the sick were seized, but those who separated themselves escaped in every instance. (*Ind. Annals*, Vol. I., p. 448.—See also the instance of Dominica, twenty-two miles from Guadaloupe, referred to by Mr. SIMON in the *Ninth Report of Public Health*, pp. 25 and 26. Also those referred to in the *Army Medical Departmental Report* for 1865, p. 349, given by Dr. PARKES, from Dr. GUNTHER's work on the cholera in Saxony.)

Dr. Wm. Budd, of Bristol, maintains, with most cogent reasoning and evidence, that the poison is cast off by the intestine of the cholera patient in the characteristic rice-water discharges, and that it may be transmitted to other and uninfected persons in the following principal ways:—(1.) By the soiled hands of attendants on the sick—a mode of communication probably very common within the limits of the family circle. (2.) By means of bed and body linen, and other articles tainted with the rice-water discharges. (3.) Through the medium of the soil. The discharges being liquid, the great bulk of them find their way to the ground, from which the poison may be propagated in three ways,—(a.) By rising into the air as a product of evaporation; (b.) By percolating into the drinking water; (c.) By atmospheric dispersion in the form of impalpable dust, after it has passed into the dried state. It is, of course, difficult to establish these modes of propagation by direct proof; but circumstantial evidence, and evidence by analogy, is so cogent and weighty, that no reasonable doubts can now be entertained regarding these modes of propagation. By *experiment* the *enthetically* contagious poisons (*e. g.*, vaccine, variola, worara, &c.) are known to retain their properties in a dormant state for indefinite periods of time after having been dried up, and to recover these properties again when moistened. Evidence almost as certain as experiment demonstrates the same regarding the poison of scarlet fever, malignant pustule, glanders, and syphilis. Therefore it is probably true of cholera, and the more so that the numerous and well-authenticated instances of the propagation of the disease through articles of dress shows that the poison, during its transit, must necessarily have been in a dry condition—a condition which entirely protects organic bodies from certain molecular changes; so that, so long as the material-holding poison remained in this dry state, no definite limit could be stated as to how long the morbid agent might retain its specific powers. From this point of view a single case may give rise to a widespread infection; and as cases multiply, it becomes more and more impossible to trace their lineal succession. The relative share which the modes of propagation (here indicated) take in the propagation of cholera must vary with season and climate, with temperature, with the habits of the people, with the condition and nature of the soil, with the water supply, with the prevailing wind, and with general sanitary arrangements.

The experience of 1865 and of 1866 confirms all previous experience as to the propagation of cholera, so well summed up by Mr. Simon in his official memorandum of July, 1866. In it he assured the public that cholera is so little contagious, in the sense in which small-pox and typhus fever are commonly called contagious, that, if proper precautions are taken where it is present, there is scarcely any risk that the disease will spread to persons who nurse and otherwise closely attend upon the sick. But he

admits it is not less true, that all matters which the patient discharges from his stomach and bowels are infective; that the patient's power of infecting other persons is due entirely, or almost entirely, to these discharges; that these, however, are comparatively non-effective when first discharged, but afterwards, while undergoing decomposition, acquire their maximum of infective power; that if cast away without previous disinfection, they impart their own infective quality to other excremental matters; that if they get access, even in the smallest quantity, to wells or other sources of drinking water, they may infect very large volumes of it; that the infective influence of choleraic discharges attaches to whatever bedding, clothing, towels, and like things which have been imbued with them; and that thus even a single case of cholera may exert a terrible power over large masses of population, *if local circumstances co-operate*.

The rapidity with which the rice-water discharges must pass into a dry state under the burning rays of a tropical sun, renders it highly probable that in India "dust," bearing the poison of cholera in a dry state ("cholera dust"), has a large share in the mode of propagation; and when the disease has prevailed, the poison may be left behind in a dormant state, from being simply dry, so that seeds of a new outbreak may exist in the soil co-extensive with the first. Hence the imprudence of encamping on old encamping grounds (so often referred to); and, in short, the views of Dr. Budd appear to explain, in the most natural way, almost all the leading facts which characterise the diffusion of the pestilence. They explain especially the relation of cholera to filthy habits and defective drainage—its predominance in low levels—its striking tendency to follow the natural line of water-shed—its communication to persons who not only have never been in the presence of the sick, but who are stationed at a distance from them—contamination of those only who visited one particular or single privy, into which the rice-water evacuations had been discharged from the first casual case—and the operation of tainted privies in propagating the disease in workhouses, barracks, prisons, and places of public resort (Dr. BUDD's "Letters" addressed to the *Association Med. Journal* in 1854-55; Dr. L. LINDSAY's able papers; and Dr. ALISON's paper "On the Communication of Cholera by Dejections," in *Edin. Med. Journal*, 1855; also Mr. SIMON's *Ninth Report*, for 1866). Under circumstances, therefore, of great concentration, or otherwise, some unknown poison is communicated, probably by *fomites*, through human intercourse, and as emanations, of some kind or other, passing through the air, they act as poisons on the gastro-pulmonary mucous membrane of susceptible persons. Dr. Parkes has shown, in his Indian experience of cholera, that it may pass with extreme slowness even against the wind (and even the trade or monsoon wind), which only retards its course, but that a favourable wind promotes its transmission; and that it sometimes travels in this way, and not by the shortest route of human intercourse, or even by the route of greatest intercourse between places.

The communication of cholera by so-called premonitory diarrhœa (*i.e.*, the early stage of cholera) is now beyond dispute. "An instance in point occurred at Southampton in 1866, where no cholera prevailed at the time. A man in the diarrhoeal stage of the disease landed at Southampton, and went to his house, a clean, airy place, where his wife and young

child lived. He was labouring under great diarrhoea when he landed on Monday. On Wednesday following his child was attacked with cholera, and died on Thursday, and on Thursday the man became worse, and died of cholera on Friday." Here the observations of Parkes confirm the observations of C. T. Kiærulf, near Bergen, and of Mr. Orton, in 1832, already referred to. The evidences of importation or transmission of the disease in this country, in 1866, by human intercourse, was sufficiently abundant, and as demonstrative as possible, consistent with the nature of such evidence; and there can be no doubt, as Mr. Simon concludes, that if a quarantine could be conducted with the extreme rigour and precision of a chemical experiment, cholera could be kept out of any part of Europe wherever such extremely difficult conditions could be absolutely fulfilled. Experience has proven, however, that they can never be fulfilled, that quarantine has always been a "futile expedient for arresting an epidemic" (BRYDEN), that all such quarantine arrangements are beset with insurmountable difficulties, and that ineffective quarantine is worse than useless (TOWNSEND). (See *ante*, p. 665, under *Plague*.)

(d.) *Theories regarding the Pathology of Cholera*.—They are as follow:—
I. One doctrine has been very extensively and very generally accepted regarding the pathology of cholera—namely, that it is a miasmatic poison which *has been absorbed*, and that, after a longer or shorter time, it produces a primary disease of the blood; that it undergoes enormous multiplication in the living body of the cholera patient, as a result of the morbid process so established; and that changes are induced in the function of respiration directly consequent upon this alteration of the blood by the poison—

"Whose effect
Holds such an enmity with blood of man,
That, swift as quicksilver, it courses through
The natural gates and alleys of the body;
And, with a sudden vigour, it doth posset
And curd, like eager droppings into milk,
The thin and wholesome blood."

—*Hamlet*, Act I., Scene v., line 64.

So wrote our greatest English poet three hundred years ago as to the effects of poison on the blood; and by such characters it has been ably shown by Dr. Wm. Budd, of Bristol, Sir Thomas Watson, and others, that cholera identifies itself with that group of contagious disease-poisons which give rise to acute diseases in which the blood is primarily affected. It is this multiplication, and the disturbance to the vital functions, which in each case constitutes the disease and destroys life. In small-pox the work of reproduction is seen in results directly appreciable to the eye. And although the fact may not yet be opened to evidence so precise, yet the circumstantial evidence is conclusive which shows that, in any case of Asiatic cholera, its specific poison is multiplied in a ratio at least as great. The facts on which Dr. George Johnson rests his belief that a morbid poison in the blood is the essential cause of cholera, may be stated as follows:—(a.) There is as much circumstantial evidence to show that the poison may be inhaled as well as swallowed, and that being so inhaled, it must enter the blood before it can reach the alimentary canal. In this respect there is some analogy between the action of a poison

producing malignant cholera and the action of poisons producing some forms of ordinary diarrhoea. It is a notorious fact, that when diarrhoea has been excited by the inhalation of foetid gases or emanations, which are inhaled, and perhaps also swallowed at the same time, the stools have the same peculiar foetor as the foul air which has excited the disease. I have frequently experienced this result from foul *post-mortem* examinations, and also when at daily work, for many years, in the dissecting room. (b.) "In many instances of malignant cholera, and perhaps in all, there are symptoms of general constitutional disturbance prior to the occurrence of diarrhoea." These symptoms, Dr. Johnson believes, correspond with the initiatory fever of small-pox and other acute exanthemata—an initiatory fever, as regards cholera, which the thermometer will one day be found to indicate. (c.) In most cases of cholera the urine is albuminous; so it is in diphtheria and in scarlet fever, as a rule—facts pointing to the existence of a blood poison. (d.) The foetus *in utero* has been killed by cholera, the characteristic discharges being found in the alimentary canal of the foetus.

"The belief," writes Mr. Simon, in his *Report on Public Health* for 1866, "that the proximate cause of cholera is a 'poison' first acting in the blood is common to Dr. Parkes and Dr. Johnson, as well as to many other writers; and Dr. Johnson builds on that belief his advocacy of a particular principle of treating cholera—the principle, namely, of 'assisting nature,' by emetics and purgatives, in what he deems to be her 'salutary and curative efforts' of vomiting and purging; but Dr. Parkes's doctrine of the state of the circulatory system in collapse, and Dr. Johnson's doctrine of the dependence of that state on spasmodic closure of the minute pulmonary arteries, are doctrines which do not necessarily involve an acceptance of the 'eliminative treatment' of cholera, nor presuppose any belief that cholera begins as a blood-disease. It is important that the different questions should not be jumbled together as one; particularly important now, since the notion of a primary blood-poison in cholera seems to be a question still at issue." And "in the present state of our knowledge," Mr. Simon writes, "I do not find it proven, nor do I see any theoretical convenience in taking for granted that cholera begins as an active blood change capable of producing primary collapse. The facts, so far as I know them, can all be reconciled with the belief that cholera begins as bowel disease, producible by direct contagion, without even a passive intervention of the blood, and that all asphyctic phenomena of the disease are supervenient sympathetic phenomena. That, so far as they are facts of cardiac paralysis and arterial contraction, they may be attributed to nervous sympathy between bowels and circulatory system, without reference to the greater or less humoral effect of the coincident flux from the bowels, is at present a tenable view. At the same time, I hesitate to accept as proven that cholera collapse is independent of humoral sympathy. In questioning the fact of a primary blood-poisoning in cholera I, of course, do not intend to deny that the blood *during* cholera is poisoned. From our earliest knowledge of the disease it has been on record that, when pregnant women have cholera, the intra-uterine offspring almost invariably dies; and more recently, in proportion as the anatomy of the disease has got to be better studied, cases have accumulated, giving detailed evidence in support of an opinion which had from the first been

entertained, that the infant in such cases dies of true choleraic infection. Waving particular reference to earlier cases of this sort (for which see, for instance, PHÆBUS, 1833, op. cit. § 51,* and BUHL, 1866, in the famous Bavarian report), I may quote some statements made by Dr. Goldbaum in the report to which I have already referred. In the three last epidemics, he says he has carefully anatomised twenty-two such infants, and never failed to find appearances which, collectively, he deems characteristic of cholera. 'In the stomach and upper part of the small intestines always there was a fluid like rice-water, sometimes a thick mass, consisting of exfoliated bowel epithelium; the heart was always ecchymosed; at the back of the tongue there were swollen papillæ, as there are in greater degree in adult cholera corpses; and in the kidney the yellowish cortex contrasted strongly with the more blood-holding medullary substance.' It may, I think, be assumed for certain that the death of the fœtus is death by cholera, and that the fœtus is infected through its blood. And since its blood is a mere derivative of the mother's blood, the fact seems to be beyond dispute that the mother's blood had cholera contagium in it. In relation to our main argument, however, the question is virtually unchanged. Is there any reason to suppose that the cholera contagium in the mother's blood was not a secondary product of disease—was not let into her circulation from the ferment seething interior of her bowels? In this point of view the case may be usefully illustrated by another and closely kindred fact. Dr. Thudichum (see p. 477, *Ninth Report on Public Health*, 1867) has made the important observation, that sometimes in cholera the blood, like the rice-water of the intestinal canal, contains butyric acid. He does not believe that this poisonous product of fermentation is primarily formed in the blood; he believes that it is only to be found there when, after collapse, absorption from the bowels has recommenced, and when evidently the presence of that and other like matters in the blood can be interpreted as a fact of secondary infection from the bowel."

There is thus evidence, both chemical and physiological, to show that the organic infecting matter of cholera is absorbed into the patient's blood, at all events to some extent, during the deep collapse of cholera.

II. From observations made during the epidemic of 1848-49, Dr. J. Snow promulgated his belief that the poison of cholera is swallowed, and acts directly on the mucous membrane of the intestines. It is at the same time reproduced in the alimentary canal, and passes out, much increased in quantity, with the discharges; and that these discharges afterwards, in various ways, but chiefly by becoming mixed with the drinking waters in rivers and wells, reach the alimentary canals of other persons, and so produce the like disease in them.

III. In 1849, Dr. W. Budd, of Bristol, expressed a somewhat similar belief—namely, that the disease depends on a living organism—a distinct species of fungus, which, being swallowed, becomes infinitely multiplied in the intestinal canal, and the action thus excited causes the flux of cholera. These organisms Dr. Budd believes to be disseminated by contact with food, but principally through drinking water of infected

* "Among the cases given in Phœbus's work is one where the infant was not actually born dead, but died an hour afterwards with all symptoms of the epidemic disease."

places. The idea of cholera depending upon the presence of a fungus growth affecting the epithelium of the intestinal canal had previously originated with Boehm in 1838, who described and gave drawings of forms of cryptogamic growth amid the *debris* of the epithelium in choleraic dejecta. He remarks that *after death* from cholera, the matters found in the intestines “teem with vegetations of micro-fungi, and that innumerable round oval or elongated corpuscles are to be found in all the vomits and dejections, as well as in the intestinal canal; sometimes single, sometimes two, three, four, or more joined, end to end, as links of a chain.” In 1849, Dr. Swayne also published drawings of “cholera cells” in the *Lancet* for October 13, 1849; and these were subsequently shown by Mr. Busk to be the spores of a species of *Uredo*, and other extraneous matters introduced into the intestinal canal with the food. This so called “fungus theory” of cholera is now no longer believed in. The careful investigations of Lewis and Cunningham in India have failed to confirm the hypothesis.

IV. Dr. W. Farr, reporting on the epidemic in 1852, states that Asiatic cholera is induced in man by a certain specific matter—the zymotic principle of cholera, which he called *cholerine*. “A variety of that matter was produced in India in certain circumstances; it had the property of propagating and multiplying itself in air, or water, or food, and of destroying men by producing in successive attacks a series of phenomena which constitute Asiatic cholera;” and he argues,—that the fact of *cholerine* being an organic matter cannot be doubted by those who have studied the whole of its phenomena and the general laws of zymotic disease. But three great questions remain,—Is *cholerine* produced in the human organisation alone, and propagated by excreted matter? Is it produced and propagated in dead animal or vegetable matter, or mixed infusions of excreta and other matters out of the body? Is it propagated through water, through air, through contact, or through all those channels?

V. In 1855, Professor Pettenkofer, of Munich, attributed the propagation of cholera to the rice-water stools of patients in a state of fermentation (a revival of the doctrine of Bayer in 1832), but that this ferment can only act where it meets with suitable local conditions (*localising conditions*). Thus a special leaven sets up a *zymosis* or series of decompositions in the impure soil itself, and the *special poison of cholera* is a *miasm generated out of this earth-fermentation*. “While, therefore, Pettenkofer considers the presence of a special ferment as essential to the production of a cholera epidemic, he also insists upon the co-existence of certain local peculiarities; namely, a damp subsoil, sufficiently porous to be penetrable by the decomposition products of human and animal excrements. It is only on such a soil, thoroughly impregnated with this peculiar organic matter, that the specific cholera poison is generated. Hence the susceptibility or otherwise of towns is in exact proportion to their soil relations (GREENHOW); and the ferment necessary to set up the peculiar decomposition of which cholera poison forms one of the products is the matter of the dejections of cholera patients. A cholera miasm is the result which becomes diffused through the atmosphere of dwellings in common with other exhalations; and thus, although formed in the ground, the air is the vehicle for its transmission to the patient; and he believes further, that the dejecta of

persons suffering from *diarrhoea* or *cholérine*, is equally capable with the cholera dejections of producing the pestilence.

VI. Dr. Macnamara maintains a theory which is a modification of that propounded by Dr. Snow, in conjunction with the views of Drs. W. Farr and Pacini, that Asiatic cholera depends upon a specific organic matter, derived from the *feces* of a person suffering from the disease. This organic matter, when in a certain stage of decomposition, is capable of imparting its specific action to the epithelium lining the walls of the intestinal canal of a person swallowing it. If swallowed in this stage (which is a vibrionic stage of decomposition) it excites, by conversion of force, changes in the epithelium of the intestinal canal similar to those which it is itself undergoing. The epithelium being thus destroyed, the serum of the blood is allowed to drain away from the capillary arteries of the intestine, and at the same time venous absorption is prevented from taking place through the mucous membrane. The characteristic rice-water stools of cholera are thus produced, which consist of serum of blood and the altered epithelium and mucus, together with gland-cells, from the walls of the intestinal canal. The result of these changes is a further outpouring of serum from the denuded walls of the intestines, while at the same time absorption of fluid through the mucous membrane is stopped. In proportion to the rapidity of this drain of fluid from the intestines the patient's blood becomes viscid; and not being able to circulate freely through the lungs, the blood becomes imperfectly aerated, its corpuscles are incapable of carrying oxygen, and the *vis à fronte* of the capillaries is in consequence suspended. At the same time, the circulation is further impaired by the hydrostatic pressure of the blood on the walls of the vessels being suddenly withdrawn, which tends to destroy the contractile power of the muscular fibre of the heart and other parts of the body. The patient is thus speedily killed, unless the outpouring of fluid from the intestines is suspended, by the vessels of the part becoming occluded with the viscid blood they contain; in which case the water remaining in the tissues flows into the dehydrated blood in the vessels, rendering it sufficiently fluid to circulate through the system until absorption can take place by means of the newly-formed intestinal epithelium. Dr. Macnamara believes that this organic matter is the sole cause of cholera; and, with the exception of this specific cholera-infecting matter, he entirely ignores all other causes, or combination of causes, as capable of producing this disease. It may be preserved in a dry state for years; and that whether fresh or old, it undergoes rapid changes in water. The first change is what he terms the "*vibrionic stage of decomposition*," lasting from two to five days in water *slightly* contaminated with cholera matter, and exposed to a high temperature. When the organic substance therein becomes oxidised, the water is no longer injurious. Furthermore, the acids of the healthy stomach, and in fact all acids, are capable of destroying the action going on in the cholera matter, so as to render it harmless. Certain degrees of temperature, both high and low, have a similar effect."

VII. Dr. Bryden, with regard to cholera in India, does not directly state a theory, but he adopts the conclusions of Dr. Beasley, of Midnapore (*Report on Cholera* of 1866-68, p. 57), who writes, that "the cholera

germs lie hidden in the ground. Occasionally they are extricated in sufficient quantity to develop a few cases of cholera; but when the atmospheric condition (which is air and water) presents itself to the cholera seeds, their vitality is roused into full play. This atmospheric condition progresses through the air, and it is attracted to, and is resuscitated by, swampy lands and localities that revel in sanitary defects." He considers there are sufficient grounds for ascribing much of the advance of an epidemic to currents of wind. He thus fully acknowledges the influence of meteorology, of bad sanitary conditions and localities in augmenting, and of personal communication in propagating, the disease. He believes that there is a *movement of epidemic bodies of cholera in India* which, springing up among its teeming population, overflows its endemic boundary from time to time, and commences a destructive march, invading other regions of Asia, Africa, and Europe. Much cholera within the endemic area forebodes danger to the districts beyond it; and, conversely, little cholera is unfavourable to its epidemic advance. The ultimate cause or *materies* of cholera Dr. Bryden does not define; but, whatever it may be, its behaviour is more like an organised vitalised body than anything else. It has a period of growth, existence, decay, revitalisation, and death—all of which are under the influence of time, place, and atmospheric conditions. The direction which any epidemic may take depends on the point at which cholera overflows the epidemic boundary; and, once started on its epidemic course, its life-period as such for Northern India appears to be four years.

VIII. Drs. Lewis and Cunningham, while they would not be understood to imply that the causes productive of malarious fevers and cholera are identical, or that localities providing for the conditions necessary for the development of the one must therefore provide those for the other also,—the tenor of their writings point to "a localised generation of the disease," that there is a "marked parallelism between cholera and malaria;" and conclude by putting the following question, namely—"Does it not seem more reasonable to infer that the disease was generated at or near the place of its occurrence, in the same manner as outbursts of malarious fevers? There is nothing more remarkable in the production of an attack of cholera, than in the production of an attack of ague; in some respects, indeed, the latter is the more remarkable of the two, seeing that once acquired the symptoms may recur at long periods after the original attack, and without subsequent exposure to the influences that originally produced it. It is true that although malarious fevers are not so appallingly fatal as cholera, nevertheless it has not been always so. During periods when cholera was either unknown in Europe, or a far milder form of it prevailed, malarious fevers almost depopulated whole tracts of country—many parts of England suffered terribly; and Sir Gilbert Blane states, that the mortality in London from ague during 1558 was so great that the living could not bury the dead. We would not for a moment have it supposed that we consider the two affections as mere gradations of the same disease; all that we desire to urge is that cholera has as good a claim as malarial diseases to a telluric origin. What the essential cause may be remains unknown in both cases; but the fact that the production of malaria is so greatly under the control of improvements in local conditions

warrants us in looking confidently to similar results with regard to the cause of cholera also."

On looking into the distractingly confusing history of this disease, it will be found that there are quite as many *false facts* (SO-CALLED *facts*, which are not facts) as there are *false theories*. All the theories given regarding the pathology of cholera agree in assigning an Eastern origin to its poison, which is believed to have reached this country either by means of direct human intercourse, as by fomites or individual contagion; or the poison itself is assumed to be migratory, and to have come hither by a kind of wave-like extension from its endemic area in India. All of the theories, with the exception of that propounded originally by Dr. Snow, consider the existence of certain local conditions, or of a predisposition in the inhabitants of infected districts, as usually necessary to give strength and vitality to the poison. It is also to be observed that, while each of the theories is apparently supported by a large amount of evidence, direct and circumstantial, each is also opposed, to some extent, by a "considerable number of obstinate facts," which the particular theory does not explain. In opposition to the view that a specific fungus is the cause of cholera, there seems now to be positive evidence to show that there is no special cholera fungus in India. In opposition to the theory that cholera springs from some special condition of ground water, there is evidence that cholera has assumed great intensity where there is no ground water. In opposition to the view that cholera excreta are the special *foci* of cholera, the disease has spread where the excreta have been carefully dealt with in conformity with the indications of this theory, and ceasing to spread in the hospitals of Calcutta, where the excreta had not been so dealt with. In opposition to the belief that cholera spreads by its specific poison getting into water, it has been known to spread where no such occurrence could possibly have taken place.

(e.) *Morbid Anatomy*.—After death, during collapse, the following lesions have been noticed:—

External Appearances.—The face is often distorted; the eyes sunk deep in the orbits, and surrounded by wide cyanotic or dusky rings. The eyelids, only partially closed, expose portions of the eyeballs as dry as parchment. The deeply-sunken cheeks give undue prominence to the nose, which is itself shrunken at the point. The lips are livid, or even deep blue; and many parts of the body are cyanotic, especially the terminal phalanges of the fingers and toes, the skin of which may be shrivelled and wrinkled like that of a washerwoman from constant immersion in soapy water. Where the skin is cut through, the hardness and dryness of the subcutaneous connective tissue are remarkable, and also the dark-red colour of the muscles. The follicular gland structure of the intestinal canal has been found to be swollen as large as hemp seeds, and the intestine filled more or less with a turbid, inodorous, semi-diaphanous fluid, usually compared to a thin starch or rice-water, the remains of that immense secretion which has taken place during life, and which, being tested, has been found always alkaline. It is found in its most unmixed condition in the small intestines. It consists of a thicker and thinner portion, and it appears to be the latter which chiefly constitutes the "rice-water" stools, which may be passed off without admixture of the

thicker substance. A layer of greyish mucus has also been found coating the whole of the mucous membrane of the alimentary canal, but without a trace of bile, although the gall-bladder is usually filled with that fluid. If the first stage has been prolonged, the mucous membrane of the alimentary canal is of a livid colour, and in some instances has presented a mammillated appearance, caused by an enlargement of the tubular glands, from which a white opaque fluid can be squeezed out, and the mammillated appearance effaced. The mucous membrane of the whole canal is in a state of uniform arborescent injection, especially near the ileo-colic valve; or, venous congestion may exist in patches, leaving other parts pale and bloodless; and spots of ecchymoses are common in these congested patches. The small intestine is relaxed and baggy, having a peculiar rosy appearance before it is opened into. The tissue is swollen from oedematous infiltration. These appearances, seen after death, are equivalent to extensive catarrh of the intestines.

The mesenteric glands are always found hyperæmic, and infiltrated with a whitish granular exudation, like the milt of a herring. The liver, the spleen, and the kidneys have in general been found gorged with blood as to their veins, and the veins of the kidneys are quite as visible as from "contracted mitral-valve" disease (SUTTON). This engorgement extends even to the bones, which, Louis says, appear like those of an animal fed on madder. The capillary vessels are empty—the natural condition in persons dying of such very acute diseases as do not remove the colouring matter from the blood (Professor W. T. GAIRDNER, *Edin. Med. Journ.*, July, 1859). The gall-bladder is almost always distended with thin brown or green bile. The urinary bladder is contracted and empty; but the mucous membrane of the urinary passages is coated with white mucous epithelial accumulation. The membranes of the brain and cord are in general congested, and the substance of the brain dotted with more *puncta cruenta* than usual. Venous congestion, and serous effusion beneath the membranes, are the prominent pathological changes found in the cranium.

In the lungs the most common appearances are the presence of blood, collected chiefly in the ramifications of the pulmonary arteries; the collapse and the deficient crepitation—arising from the more or less complete absence of air in the air-cells, which readily makes its exit through the dry bronchi when the cavity is opened; the existence of blood in the minuter capillaries, and the approximation of the vesicular parts of the pulmonary substance. The ultimate tissues of the lungs are pale and bloodless, remarkably dry, and free from hypostasis and oedema. In other cases—about one-half of the whole—there is more blood in the minute structure, congestion throughout, a corresponding dark colour of the lung, and a variable amount of frothy serum. Dr. Johnson gives the following diagrammatic representation of the state of the heart, lungs, and large vessels, when death has occurred during the stage of collapse in cholera, from an original drawing by Mr. John Wood. The lungs weighed very much less than usual, often not half their usual weight; and on section they appear dry. The blood they contain is all in the branches of the pulmonary arteries: it is black-looking blood—thicker than usual, but still fluid; so that on puncturing

a vein, such as the jugular, it escapes in such quantities that the right side of the heart is emptied in a few minutes (SUTTON). The anterior portions of the lungs were of a grey colour—very much paler than normal; the posterior portions and the bases of the lungs were much darker in colour, soft in consistence, and easily broken down. A section of the lung substance rapidly became of a bright scarlet colour on exposure to the air. The right side of the heart and the pulmonary arteries were

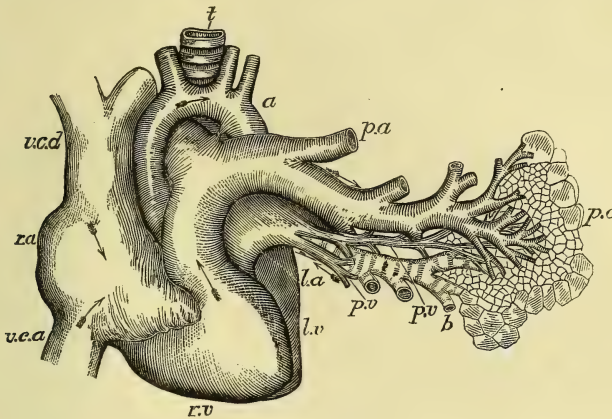


Fig. 91.*

generally filled, and, in some cases, distended with blood; the left side and aorta are generally empty, or contain only a very small quantity of dark blood; the left side evidently had received little or no blood, but had continued to contract, in some cases even violently, on the last drop of blood which had entered it (so-called "concentrical hypertrophy").

But the question may be also put in another form, as it has been already so well put by the able reviewer of Dr. Johnson's book in the *Saturday Review*:—"Why has the circulating blood stopped here (in the pulmonary arteries), and by what means has it been brought to a stand? Were the arrest of motion due to gradual thickening in consequence of the continued abstraction of its liquid portion, it would be found stagnating in the capillaries as well as in the arteries. It must be borne in mind that one characteristic symptom of cholera—that symptom which, irrespectively of the fatality of the disease, renders it truly a disease to be dreaded—consists in very painful cramps of the larger muscles of the body. These contractions, it may be assumed, are produced by the choleraic poison, just as we know they are producible by the poison of strychnine. Dr. Johnson supposes that a similar spasm or cramped state

* EXPLANATION OF THE FIGURE (after Dr. Johnson).—The venæ cavæ, the right cavities of the heart, and the pulmonary artery with its branches, are seen to be distended; while the pulmonary capillaries, the pulmonary veins, the left cavities of the heart, and the aorta with its branches, are comparatively empty:—(v.c.d.) vena cava descendens; (v.c.a.) vena cava ascendens; (r.a.) right auricle; (r.v.) right ventricle; (p.a.) pulmonary artery; (p.c.) pulmonary capillaries; (p.v.p.v.) pulmonary veins; (l.a.) left auricle; (l.v.) left ventricle; (a.) aorta; (t.) trachea; (b.) bronchus.

of the muscular fibres which embrace, and by their natural contractions regulate the size of, the minute pulmonary arteries, is caused by the same choleraic poison, and bars these slender channels against the advancing blood. The thickening of the blood is a consequence, and not a cause, of the collapse. Precisely in the same way does a similar condition of the muscular fibres of the smaller air-tubes of the lungs constitute a fit of spasmodic asthma. . . . Thus the emptiness of the systemic arteries accounts for the extinction of the pulse at the wrist, for the cadaverous sinking in of the eyeballs and falling of the features, for the blueness and coldness of the skin, and for the absence of syncope. The circulation stops, not from debility of the heart, as in exhaustion, but in consequence of a direct mechanical impediment to the onward course of the blood. We can understand the impotence of brandy against this condition; and how, on the other hand, bleeding may help, both by relaxing the spasm and by unloading the distended right heart, to restore the circulation. Into this explanation Dr. Johnson presses, plausibly enough, the singular effect of the injection of fluids into the veins of these patients. It appears that, to be influential at all, the fluids must be hot; and he concludes that they act chiefly by relaxing, through their warmth, the spasm of the smaller arteries. The blood then flows on again, and the symptoms of collapse are for a time removed. Again, the husky whispering voice is owing, not to muscular weakness, but to the small volume of tidal air in the respiratory currents. As but little venous blood reaches the lung tissue proper, there is but little demand for air to meet and decarbonise it. The respiration accordingly becomes shallow, and the vocal pipe, feebly blown through, refuses to speak. Under the temporary impulse of the warm injections, the voice regains its usual tone and note. Once more, there are chemical and less obvious changes already noticed which receive their explanation from this theory." The contraction of certain muscles after death is a phenomenon very common in cholera, by which the extremities, especially the fingers and toes, are moved after death, so as to change their position from that which they held immediately before death. This no doubt has given rise to the belief that many supposed to be dead were yet alive; and to the still more painful belief, that so they may have been prematurely buried.

The icy coldness of the body in the stage of collapse passes away after death, when the temperature is said to have risen sometimes to 102° or 104° Fahr. *Rigor mortis* is very difficult to overcome; and I have often noticed in the dissecting room that cases of death from cholera would frequently have the muscles ruptured—especially the *rectus abdominalis*, within its sheath.

Such are the appearances which the body has presented when the patient has died in the first, the asphyxiated, pulseless, or collapse stage. The enlargement of the solitary follicles of the intestines is believed to be peculiar to those cases in which diarrhoea, or other disorder of the alimentary canal, had for some time preceded the fatal attack. This enlargement bears no relation to the intensity of the disease, being often most conspicuous in the least severe cases; and it is an appearance considered of secondary importance. It has been found in about two-thirds of the cases (Dr. W. T. GAIRDNER). When the patient has survived

until reaction has taken place, and the second or febrile stage has been formed, the body no longer presents that shrunk, worn, and livid appearance which is seen on death taking place in the first stage; but, on the contrary, rather a full and plump appearance. The injection of all the large organs disappears, the blood being recalled to the surface of the body. The alimentary canal is no longer distended with the turbid secretion peculiar to cholera, but contains a thin yellowish *purée* of faecal matter, having the usual odour. The mucous membrane of the alimentary canal has now, however, been found more or less diffusely inflamed, sometimes in all its divisions, but more especially in the pyloric portion of the stomach, and also in the duodenum. The glands of Peyer, as well as the solitary glands, though occasionally found enlarged, were seldom found ulcerated; but when that was the case the corresponding mesenteric glands were also enlarged, being sometimes pale or purple, and when cut into gave issue to a dark liquid blood.

The *post-mortem* appearances, and the order of the symptoms tend to show that the blood has a difficulty in passing through the lungs, mainly on account of its viscid and thick condition; and that the loss of animal heat, embarrassment of the respiration, and gradual arrest of circulation, are produced by some aberration of the proper respiratory changes, or impediment to them. But as the mechanical part of respiration remains perfect, and as there is no impairment in the voluntary command of the respiratory muscles, and as the heart evidently beats in many cases till stopped by the want of blood on the left side, and by its accumulation on the right side, "we are compelled to look," says Dr. Parkes, "for the cause of such arrest of the circulation in the only remaining element of respiration—namely, in the blood itself" (*Researches into the Pathology and Treatment of Asiatic or Algide Cholera*, p. 107). The most important researches on the chemistry of the blood in cholera have been made by Dr. Parkes and Dr. Garrod, of London, and Schmidt, of Dorpat. The latter observer has attempted to trace out the exact chemical steps which attend the period of transudation *from* the blood into the intestinal canal. The most prominent phenomena of cholera, during this period of transudation, consists in "separation of the water and of the salts of the intercellular fluid (of the blood) through the mucous membrane of the intestinal canal, and the retention in the blood of an important excess of albumen, and of blood-cells, with apparently less, but in reality with great diminution of the salts and fibrine." The period during which this transudation takes place is generally one of definite duration (about thirty-six hours), and in it the serum and fibrine (intercellular fluid of the blood) are first affected. Water, salts, and a small portion of albumen, pass off, and form the well-known liquid stools. The order in which the constituents of the serum are affected is thus stated by Schmidt:—The water transudes before the solids of the serum; the inorganic before the organic solids; the chlorides before the phosphates; the salts of soda before the salts of potash; and the order is very much the same as takes place during the action of purgative medicines, such as *elaterium*. Very soon after this transudation commences an important change occurs in the blood; the normal diffusion currents between its fluid part and the fluid in the blood-cells

alter; and the constituents of the blood-cells transude into the serum, in the same order as the constituents of the serum transude into the alimentary canal; that is to say, the water diffuses more readily than the solids; the inorganic solids more readily than the organic; the chlorides (and of these the soda salts) more readily than the phosphates. The result of all these changes in the fluid of the blood, and in the blood-cells, is, that at the height of the transudation-period the constitution of the blood is profoundly altered. The inorganic constituents, if compared to the water, are during the first four hours increased, because at this time the water is passing off with great rapidity; afterwards, as the salts pass off, the disproportion is lessened, and after eighteen hours or so the proportion of salts is greatly diminished, and, if compared with the organic constituents, the diminution is enormous. With respect to the individual salts there is in the blood a relative preponderance of phosphates over chlorides, and of potash salts over soda salts. By the end of eighteen hours or so, the blood-corpuscles are left in a most abnormal condition. The great loss of water and of salts, especially of the chloride of potassium (a most important constituent of the blood-cells), at once leads to the conclusion that their functions must have been greatly impaired. The amount of oxygen contained in them was lessened by one-half (SCHMIDT). The fibrine is usually in large amount, and coagulable with great firmness (Dr. W. ROBERTSON). On the other hand, defective or imperfect coagulation of the blood in cholera was observed by Dr. Parkes as occurring in little less than a quarter of the whole number of cases observed by him. The presence of fibrine was not indicated by any coagulation either in or out of the body; and, whether coagulated or not, the blood has usually a dark colour; but it generally acquired an arterial tint when brought into contact with the air in thin layers. Dr. Robertson's observations were made on the cases occurring in the Edinburgh epidemic of 1848 and 1849; while Dr. Parkes's observations were made on two severe epidemics of cholera in India in 1843 and 1845. He also made the interesting observation, that a few drops of the thick substance taken from the intestines had sometimes the effect of restoring the vivid arterial colour of the blood, a result which the transudation of salts just described may in some measure explain. During the transudation into the intestinal canal, it appears that the diffusion currents *from* the blood into various structures are diminished; while, on account of the density of the blood, the inverse currents *from these structures to* the blood are augmented in rapidity. In this way fluids are drawn from the muscles, the viscera, and, in fact, most of the tissues; and it is probable that these fluids are charged with substances (such as sugar, &c.) which, under ordinary conditions, are taken very much more slowly into the blood, and are soon decomposed when they get there. The extent to which the blood is contaminated and injured by this admixture, and also by the retention of urinary constituents, is not yet accurately known. "When we remember," says Dr. Parkes, "the great share taken by the blood-globules in the respiratory and heat-furnishing processes, it is scarcely possible to avoid concluding that their loss of salts is connected with the characteristic cyanosis and lowered temperature in cholera. In *most* cases there is vomiting and purging before there is loss of heat, though this very soon follows in a

slight degree, and then gradually augments. In other words, the diarrhoea coincides with the first or early chemical changes in the blood—the transudation of some of the constituents of the serum. The lowered temperature follows afterwards, at the time when we know that diffusion from the blood-cells into the serum must be taking place, and augments gradually as the diffusion increases.” In all the cases examined by Dr. Marcus at Moscow, in 1832, the clot and serum evinced acid qualities on the application of litmus, except in four cases, where the discharges were watery and the reaction alkaline. The phenomena of the disease may thus be traced from the transudation of serum constituents as the starting point. All the other chemical changes in the blood, and the most marked symptoms (such as the abnormal respiratory process), follow as a matter of course.

Such is the nature of cholera, according to the observation of Simon, Parkes, William Robertson, Schmidt, Niemeyer, Johnson, and Sir Thomas Watson, and thus “an early theory of the nature of this disease has received the support of the best physicians and chemists of the day—namely, that the blood, if not the primary seat of the disease, becomes eventually contaminated by the action of a specific poison.”

THE MICROSCOPY of the body in cholera, and especially of the stomach and the intestines, has led to the re-discovery in Germany (by Drs. Thomé and Klob) of microscopical bodies like *fungi*, innumerable and vehemently multiplying, whereof swarms are shed with prolific and infective power in each characteristic evacuation of the sick. At the International Medical Conference on Cholera, which met at Easter of 1867, at Weimar, and which Mr. Simon attended, Professors Hallier, of Jena, and De Bary, of Halle (two of the leading mycologists of Germany), were associated with Drs. Thomé and Klob to make, in common with them, a statement and appreciation of the facts which had been observed, and which are in substance that—“Both observers find in cholera evacuations and in the intestinal mucus of the dead body definite organic structures, consisting of excessively fine granules, clustered more or less densely in the interspaces of a jelly which surrounds them. The granules divide and subdivide themselves, to form beaded threads, which interlace in immense numbers into felted masses in the mucus. The further development of these organisms has been determined by Thomé and Hallier. By sowing or cultivating them, these observers have got, after some time, larger round cell-like bodies, which rapidly multiplied, and also abundant filamentous fungi (*cylindrotenium*), on which grew cylindrical spores, capable of developing again to filaments” (*Ninth Report* of Mr. Simon, p. 31). In 1849 the question of *fungi* in cholera stools was repeatedly discussed on the repeated observation of several independent observers; but the objects then described do not seem capable of precise identification, or as being exactly the same as those now described. It would appear that Boehm in 1838 described “the whole extent of the intestine as teeming with a vegetation of *microfungi*; that innumerable round and oval, or more elongated corpuscles, are to be found in all the vomit and dejections, as well as in the canal, sometimes single, sometimes two, three, four, or more, joined end to end, as links of a chain, and these chainlets sometimes branching; that such forms are held together in mucous floccules, and come best to light when *liquor*

potassæ is used; that within the small intestine they are often so numerous that not the smallest specimen will fail to show numbers of roundish *fungi* forms amid the *debris* of epithelium" (*Über das vorkommen der Gährungskeime (Pilze) in Nahrung-Kenäl der Cholera-Kranken*, quoted by Mr. SIMON, *Ninth Report*, p. 518). In the microscopic examinations of the "rice-water stools" of cholera in 1848, made by the late Dr. Parkes (whose observations were at the time confirmed by Drs. Sharpey and Jenner, and by Bowman, Hillman, Ellis, and Quekett), "peculiar corpuscles" were observed and were variously named as such, or as "dark-yellow granules," or as "organic corpuscles (about the size of the pale corpuscles of the blood), finely granular on the surface, and containing from six to twelve dark-yellow or black granules." Without being able to state what these "peculiar corpuscles" are, Dr. Parkes showed that they were not mucous, but that their existence and development are confined to the deep algide period—that they were not seen in the premonitory diarrhoea, nor after the algide stage, and that they disappeared when the pulse and the warmth of the surface were returning. They co-existed in their greatest perfection with the purest type of the cholera fluid (*London Journal of Medicine*, 1849, p. 144, *et seq.*) Thus Dr. Parkes, and those observers who aided him, recognised certain "peculiar corpuscles or granules" in the rice-water stools of cholera; but neither of them suspected the peculiar corpuscles or granules to be of vegetable or fungoid origin. They appeared then to Dr. Parkes to be "but modifications of the same substance—namely, fibrine." He, however, recognised "vib- riones, in great numbers, and two or three oval transparent bodies placed end to end. When the stool was kept, these fungi increased in numbers" (Ob. 6). In the descriptions and plates of Klob, Thomé, and Hallier, the corpuscles and granular bodies which they figure were recognised by Dr. Parkes as similar to those which so attracted his attention in 1849. He again noticed these bodies in 1865 and 1866, when they vividly recalled the previous observations to his mind. Dr. Parkes concludes, from these and his own observations, that this fungus development really exists, and invariably in cholera dejections. He found them in every stool which he examined in 1849 and in 1854; and he again saw them in 1865 and 1866. He is familiar with all the forms described by Klob and Thomé; and believes them universal in cholera stools; and they form the major part of the white flocculi of the true "rice-water stools" (*Report on Progress of Hygiene in Army Med. Dep. Reports for 1865*). In 1854 Dr. Lauder Lindsay also recognised large bodies, which he named "gonidic," from their resemblance to the gonidia of the lichens. They appear quite globular, usually larger than pus corpuscles; have a distinct wall, colourless, and transparent; frequently a distinct central nucleus, also colourless, round which are aggregated a number of rounded granules of a bright greenish-yellow or orange colour, resembling the chlorophyl grains in the cells of plants. These bodies have occurred in greater or less abundance in the evacuations of *all* the cholera patients under Dr. Lindsay's charge; and they pass through the digestive apparatus both of man and the dog apparently without change (*Edin. Med. and Surg. Journ.*, 1854). With reference to such positive assertions regarding the presence of fungi of a specific character in cholera discharges, and the spread of the disease

through their agency, it became very desirable that those points should be thoroughly examined by men specially trained for the inquiry,—the examination of microscopic fungi and their identification being of extreme difficulty and delicacy, while an interpolation of their pathological significance requires an extensive knowledge of disease. In June, 1868, two medical candidates then at Netley were considered specially qualified to undertake this investigation, namely,—Drs. Lewis and Cunningham. They went out to India on 12th December, 1868, where they received every attention from the authorities; and several reports have since been published by them, after long and elaborate investigations into the fungoid forms referred to by Hallier and others. The following conclusions have been arrived at as the results of their investigation referred to in the *Sixth*, and subsequently in the *Tenth Annual Report of the Sanitary Commissioner with the Government of India for 1869 and 1873* (Calcutta):—

“(1.) That no ‘cysts’ exist in choleraic discharges which are not found under other conditions; (2.) That cysts or ‘*sporangia*’ of fungi are but very rarely found under any circumstances in alvine discharges; (3.) That no special fungus has been developed in cholera stools, the fungus described by Hallier being certainly not confined to such stools; (4.) That the still and active conditions of the observed *animalcules* are not peculiar to this disease, but may be developed in nitrogenous material even outside the body; (5.) That the flakes and corpuscles in rice-water stools do not consist of epithelium, nor of its *debris*, but that their formation appears to depend upon the effusion of blood-plasma; and that the ‘peculiar bodies’ of Parkes found therewith correspond very closely in their microscopic and chemical characters, as well as in their manifestations of vitality, to the corpuscles which are known to form in such fluid; these are generally to a greater or less degree associated with blood cells, even when the presence of such is not suspected, especially as the disease tends towards a fatal termination, when the latter have been frequently seen to replace the former altogether; and, (6.) That no sufficient evidence exists for considering that *vibriones*, and such like organisms, prevail to a greater extent in the discharges from persons affected with cholera than in the discharges of other persons, diseased or healthy; but that the *vibriones*, *bacteria*, and *monads* (*micrococcus*) may not be ‘*peculiar in their nature*’ (for these ‘*do*’ vary), and may not be the product of a peculiar combination of circumstances, able to give origin to peculiar phenomena in predisposed persons,—is not proven.” Dr. Macnamara also asserts his conviction, after a long and attentive study of the subject, that fungi peculiar to cholera dejecta have not yet been discovered; that no doubt fungoid growths appear in this as in other nitrogenous matters undergoing decomposition—notably the *oidium lactis* and *mucor ramosus*; but neither are peculiar to decomposing cholera dejecta. Moreover, he desires particularly to affirm, that neither these nor any other fungoid growths can be discovered in *fresh* cholera stools. The more recent the specimen the clearer this fact appears. Dr. Macnamara has examined the contents of the intestinal canal for the appearance of mycelial threads or *sporangia*, and even after protracted collapse, has absolutely failed, in numerous instances, in detecting any characteristic elements of the kind, and he states that he is reluctantly compelled to abandon his faith in the existence of any such growth. So

far, therefore, as *fungi* are concerned in the spread of cholera, I am satisfied that we have no grounds for such a belief. In the blood also there is a total absence of bacteria, fungi, or other extraneous bodies; but a great abundance of white blood cells, in a state of great activity, or of bodies which cannot be distinguished from the white blood cells. The red corpuscles have also been shown to be peculiarly diffuent.

(f.) *Chemical Changes undergone by the Body in the Progress of Cholera.*—Dr. J. L. W. Thudichum, at the instance of Mr. Simon, made important observations during the epidemic of 1866, which are published in Mr. Simon's report of that year. The following is a summary of his results:—

The blood after death during collapse contains urea in variable abundance. The rice-water-like evacuations contain butyric acid, and yield nitrogen and carbonic acid, but no urea. In bodies dead at an early period there was no urea; and more seemed to accumulate after a protracted algide stage, and much more after three to six days' torpid condition. The greatest amount of urea was found after a long algide stage, with rise of temperature at the end. The blood loses water, albumen, and salts, and is incapable of passing the capillaries with the usual freedom. It retains most of its colouring matter in its normal chemical composition; and Dr. Thudichum's observation led to the conclusion that any fermentation of the blood in the manner in which the intestinal contents are fermented was very improbable. The blood absorbs water from the tissues; and there is no chemical evidence of any special cholera poison in the blood. The epithelium of the inner surface of the blood-vessels becomes detached and mixes with the blood, and the blood adheres to the blood-vessels with great pertinacity. The secretion of bile is completely arrested; and in extreme cases a clear white fluid percolates through the hepatic ducts, free from bile, colouring matter, and albumen. It seems to be simply water with a trace of alkali and a vestige of mucus. In some instances the fluid is coloured, but contains no bile acids. The bile ducts shed their epithelium.

In all of this description *post-mortem* results are unfortunately mixed up with vital phenomena; and it is to be regretted that evidence is so contradictory on a matter of fact so definite as the shedding of epithelium during life in cholera patients. This latter fact can only be determined *after death*, when all ducts shed epithelium, in cases of all diseases or modes of death that I have examined. So long as the patient lives and passes the characteristic rice-water evacuations, I have never seen them containing shed epithelium in any notable quantity. In August and September, 1849, I examined numerous stools from patients dying in the collapse of cholera immediately after they were passed; and on reference now (1871) to my notes and drawings, I conclude that it is only in the *post-mortem* contents of the bowel (small intestine) that cylindrical epithelium is to be found—the result, no doubt, of maceration, and the usual desquamation that follows death. If they were shed so abundantly during life, as some say they are, how do they come to be so abundant in the contents of the intestine after death?—a statement which is not denied. Epithelium after death is alike found in the stomach, urinary, and gall bladder. Denudation of the intestinal villi, or desquamation of the epithelium, is no essential lesion of cholera. Boehm's statement seems to have

been adopted as one of authority, without further question; and while competent observers have agreed with him (*e.g.*, Beale) on the one hand, no less competent observers have not been able to confirm it on the other (*e.g.*, Parkes, Gairdner, Lauder Lindsay). The observations at the London Hospital (*Report*, Vol. III.) and those of Dr. Bruberger, of Berlin, who examined the stools of 540 cases, confirm the statements of Parkes, Gairdner, and Lindsay (*Virch. Arch.*, 1847, p. 361, quoted by PARKES in *Army Med. Report* for 1865). It may be, however, that a distinctive change in the epithelium, described by Dr. Macnamara, may be so complete as to prevent its recognition as such. He thus describes a rice-water stool:—"They are always alkaline, consisting of a watery gruel or cream-like fluid, composed of thinner and thicker portions. Its consistence varies according to the varying quantity of its component parts. The thicker portions are flaky, stringy, curdy, or clotted. When first passed, the rice-water evacuations soon separate into two portions, the flocculent curdy matter sinking to the bottom of the glass or vessel in which it is contained, leaving a whitish fluid above. This separation of the material into two parts takes place rapidly in many instances, say in from one to three hours, and is evidence of the severity of the disease; for if the more solid matter of the dejecta collects in the lower part of the fluid very speedily, it indicates the complete death and disintegration of the organic matter. On the other hand, if the separation of the fluid and more solid components of the rice-water product takes place slowly, it is on account of the evacuation containing a considerable quantity of comparatively healthy mucus, and the case so far allows of a more favourable prognosis. The flocculent matter of the stools is composed of epithelial cells, and the mucous lining of the intestinal canal in various stages of decomposition; but the perfectly fresh dejecta in the active stages of the disease contain no *vibriones*. Towards the end of collapse, when the evacuations are passed less frequently, probably remaining in the intestine for some hours, *vibriones* may be seen in the fluid immediately after it is passed." The epithelial cells are often shed in large quantities during the early stages of cholera. For instance, in the case of a little boy—three years of age—whom I was called to see, and who had gone to bed apparently in perfect health, and slept soundly through the night; on rising next morning he had a call to stool, and the mother noticed something peculiar in his appearance; on examining the pot she discovered it to be full of a dirty brown-looking fluid. The child lay down, and seemed much exhausted. I was sent for, and within half-an-hour arrived at my patient's house, to find him almost pulseless, and passing fast into a state of collapse. The first stool had been kept for my inspection. I secured a specimen of it, and had it under the microscope within two hours of the time it was passed. It contained a vast number of columnar epithelial cells; all of them were more or less under the influence of the *molecular change* I have so frequently referred to. The stools passed by this poor child at 10 P.M. contained comparatively few cells that could be identified as such, though I am not prepared to say that much of the flocculent matter was not composed of disintegrated epithelial gland-cells and mucus, doubtless combined with albumen and other organic matters in a state of decomposition." Again, he states that "the changes in epithelium occur

during the life of the patient, and are evidence of the rapid destruction of these cells taking place before death. The cells under a quarter-inch object glass contain a vast number of minute dark specks, precisely similar to those seen in a pus corpuscle. The majority of the epithelial cells are invaded by this molecular matter. In a few hours this increases, so that the outline of the cells becomes altered, their margins jagged, and ultimately *their shape is completely destroyed*, the molecular matter increasing at their expense. The result at the end of a few hours is *an irregular mass of molecular matter—a granular, diphtheritic, or amorphous deposit*, as it has been variously called by authors. It is an aggregation of these molecular masses which constitutes the bulk of the flocculent substance noticed in the rice-water stools of cholera patients." I have underlined certain portions of this description to draw attention to its bearing on the varied accounts given as to the presence of epithelium shed, and seen as such. As form is one of the chief modes by which objects are recognised under the microscope, and as *ultimately the shape of the epithelium is completely destroyed*, and *the result is an amorphous deposit*, it must be very difficult to identify this resulting matter as having been originally epithelium. The simple question of fact regarding the shed epithelium has yet to be decided by more extended observation, just as the question of *fungi* has been. Niemeyer states that *after death*, the most important appearance in the intestines is the great loss of epithelium. The intestinal villi are stripped of their protecting covering, or it is elevated by a serous effusion, and still loosely adherent to the villi; or it lies on the intestinal wall as shreds of mucus. Also, on the authority of Bruberger, he states that the white flocculi floating in the serum rarely consists of perfect cylindrical epithelium, but generally of its remains, in the shape of fine loose nuclei, with coarse or fine granular masses imbedded in a mucous basement substance, and of round nucleated, coarse, or finely-granulated cells. *The tissues* generally are doughy to the feel, from want of water. The muscles become dry, and are affected with cramps; the spasms, beginning at the most distant parts, rise gradually to the centres; and the cramped muscles show deposits; urea accumulates in them, and nuclei of their sarcolemma multiply.

The contents of the colon half an hour after their removal were observed to evolve gas, and to lift off the heavy glass stopper of the bottle in which they were contained. Their smell was putrid, but not fecal. The flocculent deposit which formed on standing filled one-half of the bulk of the fluid. It consisted of intestinal epithelium in patches, single cells, and cells in all conditions of disintegration, and great numbers of vibriones. The fluid filtered but very slowly, and the filtrate was not clear. Its reaction was strongly alkaline. On dialysis of the alkaline rice-water, the *dialyate* gave an acid reaction, from which, on being neutralised by baryta water, and the fluid evaporated, several matters could be obtained, namely,—(1.) A body crystalline like leucine, and combining with nitric acid; (2.) An oily substance, which was soluble in water, and with nitric acid gave a peculiar pink reaction; (3.) Butyric acid, combined with the added barium; (4.) Inorganic salts in considerable quantity; (5.) No urea could be discovered in the rice-water stools. Volatile acids, butyric and

acetic, were also obtained, the latter apparently prevailing in quantity. They were combined with ammonia in the original fluid. The rice-water evacuations, therefore, contain the following ingredients:—Vibriones, cells from the surface of the intestine, granular *debris* of cells, mucine, modified hemochrome, albumen, albuminous body giving rose-pink reaction, butyric acid, acetic acid, ammonia, leucine, inorganic salts. Nearly the same results were obtained by Dr. Lauder Lindsay in 1853. The gas evolved is at first composed almost entirely of nitrogen; soon, however, carbonic acid prevails, and ultimately nothing but carbonic acid is evolved. At one period some hydrogen is developed. In 1848 Dr. Parkes examined many cholera stools, and his observations coincided with those of O'Shaughnessy, Vogel, Wittstock, and Andrew Buchanan (of Glasgow). The thin fluid was always alkaline, and contained an abundance of alkaline chlorides, phosphates, and sulphates, and a certain proportion of albumen. The *odour* was always peculiar. If it is admitted that the cholera evacuations acquire infective powers only after a period of fermentation, it is also easy to understand that the specific infecting power may belong to albumen or mucine at a particular stage of disintegration or chemical cleavage. The next knowledge which it is necessary to acquire is evidently this—namely, the exact period at which the rice-water stools acquire infective properties, and their chemical composition at that period. The most dangerous period of the choleraic stools is believed to be when they become very ammoniacal. This begins to occur usually immediately they are passed, but not to any extent for some time; and anything which makes and keeps them acid prevents the ammoniacal change (PARKES).

(g.) *Circumstances predisposing to Cholera; or presenting or favouring Resistance to the Attack.*—Influences are chiefly apparent in the age and sex, food, fatigue, housing, ventilation, filth, misery, and intemperance of the people. Both sexes, and all ages, including new-born children, are liable to the disease. Dr. Farr's results show that males suffered more than females at all ages under twenty-five years; but between twenty-five and forty-five the females suffered more than the males. The deaths from cholera in Paris were estimated at 18,402 in 1832; and it was remarked that the mortality was least from six years to twenty, greater from thirty to forty, and greatest of all in old age. The influence of *sex* in predisposing to cholera can hardly be said to be determined; for in Calcutta, of the native inhabitants attacked with cholera, the males were to the females as four to one, while in Bombay the proportion was as seven to twenty-five. In Canada, the soldiers' wives were observed to suffer nearly in an equal proportion with their husbands; and this was the case among the civil inhabitants of Gibraltar. In all countries the *lower classes* have always suffered in a much greater proportion than the upper classes. In Calcutta, the disease ran a wide career of destruction in the native town, while the "City of Palaces," inhabited by the English, was much less affected in proportion to their numbers; and the same disproportion has been observed in Bombay. In general, it has been observed, among the native inhabitants of India, that the Brahmin and Banian merchant suffered less than the ryot or farmer, while the poor outcast Pariah suffered the most of all. In every town in Europe it has been observed that the lower classes, and especially those resident on the banks of rivers,

have suffered infinitely more than the upper classes. In military life it has been believed that the Sepoy suffered more than the European soldier living in India. This perhaps, is true in some instances; but the returns of the Madras army show this not to have been the fact in that Presidency. There the European soldiers attacked appear to have been as one to three, while of the Sepoy force it was only one in four and a half. The influence of cholera on different races attacked at the same stations in India, and at the same dates, is stated by Dr. Bryden in the following general results:—European troops were *attacked* in the proportion of 89·20 *per thousand*, while Native troops were *attacked* in the proportion of 9·56 only. Of these there *died per thousand*, of Europeans, 53·68; of Natives, 4·11 only. Again, out of every 100 cases there *died*, of Europeans, 60·19; and of Natives, 43·02. The Goorkhas suffer even more than Europeans from cholera. In the Indian army, also, it appears to have been universally observed that the officer suffered in a less proportion than the soldier, the cavalry than the infantry, and the infantry less than the hard-labouring, ill-fed camp follower.* The troops on march likewise universally suffered more than the troops in quarters; and this influence of long marches appears to indicate something more powerful than mere fatigue in bringing about the disease. Dr. Balfour has proved that of the native soldiers of the Madras army thirty-two died of cholera in cantonment, and eighty-six when marching, to an average of 10,000 strength; the number attacked being respectively 85 and 200 in 10,000. Dr. Lorimer's reports show that the men were more frequently attacked on long than on short marches, the men (as Dr. Farr observes) being longer exposed to the causes of disease. These causes are those which are incidental to the life of a soldier on the march, such as lying by the banks of rivers, on low marshes, jungly grounds, sleeping on the ground, and encamping amongst the filth of encampments recently occupied, but abandoned. Of this latter indiscretion there were many melancholy examples during the war with Russia in 1854; for example, the occupation of the evacuated camping ground at Aladyn, in Bulgaria, and that on the heights above Alma, previously occupied by the Russians, the consequences of which were so fatal to the first and fourth divisions of our army. The effects of a poor diet in predisposing to cholera will perhaps be better understood by stating that the European suffers less than the Mohammedan, and the Mohammedan, who is better fed and better clothed, than the Hindoo, except during their rigid fasts, when the Mohammedans suffer in a much larger ratio. During the epidemics of 1848 and 1849, in Edinburgh, Dr. William Robertson, of that city, found that anæmic persons were those most predisposed to cholera.

* The Madras Sepoy, of whom alone Dr. Balfour wrote, invariably carries his family with him. At the end of a long march he puts off his accoutrements, and hastens back, without tasting food, to assist his family out of the difficulties incident to a country in which the roads are often mere tracks. He thus performs nearly double the route march, and finally encamps on ground which for years has been used for the purpose, and is saturated with the excretion of former sufferers from the disease. Moreover, for a long time the authorities in Southern India were most reckless in sending regiment after regiment in one another's footsteps, through districts known to be infected; and as they all occupied the same encamping ground, the last regiments pitched in places saturated with cholera evacuations, and surrounded by the half-buried remains of the dead. These facts to some extent explain the effects of marching on Sepoys (W. C. MACLEAN).

Symptoms and Various Forms of Cholera.—*Cholera Indica* has many degrees of severity, and hence many pathologists have divided it into *Cholera Indica mitior*, and into *Cholera Indica gravior*. The French have termed the slighter forms of the disease *Cholerine*, and this name has been also recently used by Dr. Farr to designate the specific poison or zymotic matter of cholera. *Malignant Cholera* is divided into two stages,—the cold, pulseless, or asphyxiate stage, and the subsequent febrile stage, when the patient outlives the first. This latter stage, however, is not essential to the disease, and has been observed in India in a small proportion of the cases only. In Europe, however, the febrile paroxysm has followed in the majority of instances. The duration of the cold stage varies from a few minutes to twelve, twenty-four, forty-eight, or even more hours; while the febrile stage lasts from four to eight or more days,—making the total duration to vary from a few hours to two, three, or even four weeks. The attack of this fatal disease is most commonly sudden, the patient at the time of his sickness being apparently in his best health; yet not unfrequently slight diarrhoea or other general indisposition has preceded it. Some hold that premonitory diarrhoea is always present. In India, in some cases, the premonitory symptoms are vertigo, and noise in the ears, the latter sometimes so loud as to have been compared to the humming of a swarm of bees, to the beating of drums in the camp, or to the roaring of the surf as on the Coromandel coast.

A period of incubation has been variously fixed at from one to fourteen days. According to the accurate observations of C. T. Kiërluf in the vicinity of Bergen, it appears that, when the disease is propagated by human intercourse, from one to four days elapsed from the supposed period of infection to the outbreak of the disease. Most frequently the disease appeared on the second day after exposure to the infection; and he found that the diarrhoea, so frequent during the invasion of cholera, is a part of the disease, and itself capable of infecting others with true cholera. This, Mr. Orton also showed, was the case in 1832, (l. c.) The extreme shortness of the period of incubation is an important element to be remembered in all investigations regarding the course of events in cholera epidemics. According to Dr. Budd's observations it seldom exceeds three days; and where the disease is virulent, there is evidence to show that it may not exceed six hours. Niemeyer fixes it at not less than thirty-six hours, and not more than three days. Dr. Parkes records an instance which fixes the incubative period so very closely, that it could not have been more than sixty hours, and was doubtless less (*Army Med. Dep. Reports*, 1870, Vol. XI., p. 206).

A classification may be made of the disease into three principal varieties, which coincide in their phenomena with many of the changes known to take place in the blood (PARKES). (1.) The slighter forms commence with much watery purging and vomiting, and pass into the second and third varieties in varying times. There may be from ten to fifty copious watery stools, but losing neither their odour nor colour, and frequent copious vomiting, before there is any great loss of heat and failure of circulation. When purging commences twenty-four hours, or two or three or four days, before the violent symptoms, such as vomiting, purging, or cramps, such patients are said to have "pre-

monitory diarrhoea." But there is always some degree of loss of heat and failure of circulation even in the slightest cases, else the case would be mere watery diarrhoea, attended only by exhaustion, and not by the symptoms peculiar to cholera. Cramps are seldom present till the stools put on the *true choleraic character*—viz., *copious white flocculi suspended in a watery fluid*. The algide symptoms come on gradually, and are less intense than in the following forms; recovery is also more common. In the epidemic in London (1866), Dr. Sutton gives forty-one examples in which there was undoubted premonitory diarrhoea, the duration of the diarrhoea being as follows:—In three cases, 12 hours; in one case, 18 hours; in one case, 19 hours; in seven cases, 1 day; in one case, 1 day and 9 hours; in twelve cases, 2 days; in six cases, 3 days; in two cases, 4 days; in two cases, 5 days; in one case, 6 days; in one case, 7 days; in two cases, 2 weeks; in one case, 5 weeks; and in one case, 8 weeks. In more than half the number of cases the diarrhoea preceded the marked symptoms by one, two, or three days; and of the forty-one cases, the diarrhoea in twenty-six was limited to the first three days. During an epidemic of cholera, all such cases of severe diarrhoea ought to be regarded practically as cases of cholera, with reference to sanitary measures; seeing that the transportation of cholera is now known to have been effected by persons suffering from such diarrhoea, which sometimes passes into the severest form of cholera. (2.) If the poison acts with greater intensity, we have the second variety, in which there is less physical alteration in the fibrine, and the circulation is carried on for a longer time. Consequently, the characteristic change is not evidenced solely or chiefly in the interior of the vessels, but it is partly transferred to the exterior of the vascular system. The albuminoid constituents, fibrine, and perhaps albumen, are effused in large quantities, and in all parts of the body, though chiefly on the free surfaces of the skin, alimentary mucous membrane, and more rarely the bronchial mucous membrane. The general nature of this effusion forms two characteristic distinctions between cholera and diarrhoea; for diarrhoea is a disease confined, in the first instance, to the eliminating part—viz., the large or small intestines, as the case may be—and is unattended, as a general rule, by the effusion of albumen and fibrine. The worst forms of this variety are seen in those cases in which, after two or three choleraic stools, severe and long-continued cramps come on, accompanied and followed by intense algide symptoms. After death the small intestines are generally found distended with the thick, white, flaky substance. Other cases of this variety present infinite modifications in severity, according as watery elimination is added to effusion of the fibrine; in other words, according as they tend towards the slighter forms. (3.) If the final change should occur at once, and should there be a complete and rapid arrest of the circulation, either from the intensity of the cause or from constitutional predisposition, the worst variety is produced, in which "a mortal coldness comes on from the beginning." As the circulation is soon almost entirely arrested by physical alterations in the blood—presumably, changes in the fibrine—there can be little purging and comparatively little sweating; there is always some effusion of the thick white substance into the intestines, but often little of the watery part of the blood. The symptoms might be inferred from a statement of this

condition; we might have presupposed a very rapid loss of animal heat, loss of voice, deafness, and vertigo, total arrest of all secretions, defective aëration of the blood, consequent dark colour of the surface, and early and deep coma. The more usual course of the disease in this country, when limited to the cold stage, is as follows:—After the patient has been troubled for a few days with diarrhœa (the more insidious and dangerous because it is *painless*), but more commonly while he is yet in perfect health, and has retired to rest, and has slept soundly till the middle of the night, or even till early morning, he is suddenly seized with an unaccountable sickness and vomiting, together with a most profuse discharge from the bowels. More persons were seized from twelve o'clock at midnight to three o'clock in the morning than at any other time (Dr. SUTTON, *Health Report*, p. 371, for 1866). These profuse evacuations are attended with severe pains down the thighs, and more especially by an indescribable and subduing sense of exhaustion, the patient often fainting in the water-closet. In an instant the physical powers of the body are not only exhausted, but its temperature sinks rapidly below the natural standard. An icy coldness benumbs it; while the skin is sometimes rendered so insensible that it has been known to resist even the action of boiling water or other powerful chemical agents. The breath, as it issues from the mouth, communicates a glacial sensation to the back of the hand; still, notwithstanding this great loss of temperature, the patient complains of being oppressed, and is incessantly throwing off the bed-clothes; while cold water, copiously and eagerly drank, is grateful to him; and, although it does not seem to afford relief to his insatiable thirst, it ought not to be withheld, but given in small quantities, or as ice to melt in the mouth, for the symptom is a most torturing one. The extreme coldness of the first stage is further accompanied by a blue, livid, or purple discolouration of the hands and feet, extending not only a considerable way up the arms and legs, but sometimes over a great part of the body. These parts often become, in a few minutes after the seizure, not merely shrunken, but singularly wrinkled, like the hands of a washerwoman after a day's hard labour. These symptoms are rendered still more distressing by the shrieks and groans of the poor sufferer, tortured by spasms, which affect the fingers, the toes, the arms, and the legs—spasms which clench the jaw, fix the walls of the abdomen in contact with the spine, or draw the trunk into singularly contorted forms. The patient thinks he obtains some relief by the use of friction, and his cries to the attendants are incessant to “rub hard.” The calves of the legs are especially liable to these cramps, and I have often seen the *gastrocnemii* and *rectus abdominalis* muscles ruptured after death from cholera. As the disease proceeds, the countenance assumes a character peculiar to this great struggle, the “*facies cholericæ*,” the eye being deeply sunken, red, and injected; while the aqueous humour, transuding its coats, leaves the cornea flat and depressed, as in the dead body; a broad and livid band encircles the lower portion of the orbit. Every feature, moreover, is sharp and pinched, as after a long wasting disease; the complexion thick and muddy; the lips and tongue purple. All these great changes have been known to take place in a few minutes. In addition, the vomiting is constant, the purging most incessant, and the pulse, though often natural, sometimes rapid, yet in some cases is not to

be felt, even after the first hour of the attack, either in the large superficial arteries or at the wrist. The voice is strangely altered; its firm and manly tone changes to a low, feeble, and unnatural sound. The urinary secretion is likewise entirely suppressed, while no bile flows into the intestines. The only organ which seems to preserve its powers is the brain; and the patient often to the last moment of his life retains the power of thinking, and of expressing his thoughts distinctly, sometimes full of hope, while at other times he seems indifferent to the fate which too often inevitably awaits him. Most patients are apathetic. Reflex excitability is also diminished, irritating vapours will not produce coughing nor sneezing; the eyelids refuse to wink if the finger is approached to the conjunctivæ, and patients do not wince if water is suddenly dashed on the naked skin. On the accession of the spasms, the vomiting, and the purging, the disorder is fully developed, and the crisis is at hand, which in a few hours must decide the fate of the patient. The termination may be favourable or unfavourable; if unfavourable, he may die with all the symptoms just narrated strongly marked; or, should it be favourable, they may abate, and a happier prognosis be formed. Unfortunately, however, it too often happens that, although the stomach retains what is taken, and the purging appears checked, and the patient falls into a sleep, yet the weakness, the entire cessation of the pulse, the coldness and lividity of the surface, and the ghastly expression of the countenance, show that a few hours must close the scene, often with so little struggle that death is only marked by the phenomena of cadaveric contraction, which sometimes continues active in the muscles for some hours after death. The largest number of deaths take place betwixt the hours of 7 and 11 A.M., and between 7 and 11 P.M., both with respect to males and females (SUTTON, l. c.) These might be considered as the critical hours; and a knowledge of such times may be of some importance in the treatment, and especially the expectant treatment of the disease. The aim of expectant medicine is not simply to stand by and do nothing, but it is to watch the disease—to see if it is running its “natural” course—to judge whether the patient tends to do well; and if not, to ascertain how he tends to die, and to strive to counteract such tendency, and thus to gain time. It is very important, therefore, to know the hours when the vital powers are likely to become feeble, and the vital functions almost brought to a stop: we may then assist the struggling patient at these particular times. “In any future registration of such facts,” writes Mr. Simon, “it would be desirable to make separate enumeration of deaths in collapse, as distinguished from deaths in reaction and fever. Of course, too, in discussing the subject of the hour of death, regard must be had to the hour of so-called ‘attack’—*i.e.*, the hour of manifestation of severe symptoms; and if this should seem to be governed, at least locally, by some general law, the determining influence of local modes of life would need consideration.”

If the patient should happily survive the cold stage, the disease may terminate by a rapid recovery, or it may pass into the second or febrile stage. The former is the more usual course in India, the latter in Europe. The first symptom of returning health is shown by the patient falling into a sleep of unusual soundness, during which the respiration becomes light and easy, the pulse freer, while a gentle, warm perspiration bedews the

whole body. This grateful pause in the disease appears to be the result of the returning powers of life, uninfluenced by medicine, for it has often occurred when none has been given. After this balmy slumber the patient awakes refreshed, and often recovers so rapidly that, in the natives of India, it almost resembles a restoration after syncope. In all the Presidencies, indeed, and especially in Bengal, the recovery of the European has, in general, been followed by a stage of reaction, usually slight, but in some cases assuming the form of the bilious remittent fever of the country, and which has occasionally terminated fatally. In Europe, restoration after the cold stage, and without febrile reaction, is by no means so frequent or so rapid as in India. Sometimes the reaction is trifling, and sleep may indeed have ensued, fecal evacuations containing bile may have passed, the urine may again have flowed, the purging, vomiting, and spasms may have subsided, the pulse may have risen, the blueness may have disappeared, and the temperature of the body may have increased, yet in many instances this amelioration of the symptoms has been only temporary; the patients relapsed and died. In most cases, however, the reaction was more considerable, and the patient, in a few hours after the subsidence of the cold stage, laboured under a severe form of fever, in no degree dissimilar to, and not less fatal than, typhoid fever. These *typhoid* symptoms, common in Europe and America, are said to be unknown, or nearly so, in India, where, if a secondary fever ensues, it assumes the form of the remittent fever of that country. But remittance is characteristic of typhoid fever; and this character may only be more expressed in India than in Europe. For the first few hours after the febrile reaction commences the tongue is white, but it quickly becomes brown and dry, while black sordes encrust the teeth and lips. The eye becomes deeply injected and red, the cheek pale or flushed, the pulse rapid, and the temperature of the body a little above the natural standard. The patient, either delirious or comatose, then lies in a state resembling the last stage of the severest typhoid fever of this country. This struggle usually lasts from four to eight days, when the symptoms either gradually yield or death ensues. In a few mild cases the fever assumes an intermittent type, or sometimes a quotidian, sometimes a tertian form: all these cases usually recover.

The blood in cholera varies according to the state of the disease. In the cold stage it is usually of an unnaturally dark colour and thick consistency, so that it flows with difficulty from the veins, and very imperfectly separates into clot and serum. Blood taken from the temporal artery has been found equally black and thick. After the secondary fever is formed, the quantity of serum increases, till at length it is much more abundant in the blood than natural; and it is singular that this takes place notwithstanding that the secretion of urine is re-established. The phenomena resulting from the changes in the blood in cholera are the proper and distinctive symptoms of the disease; and the term "*algide*," first used by the French pathologists, very properly designates one of its most remarkable and constant symptoms—namely, the diminution of animal heat. The sensation of cold communicated to the observer has been compared to that experienced on touching a moist bladder or the skin of a frog. It is "reptile-like." The algide symptoms, therefore, essentially constitute the characteristic phenomena of this disease. In

proportion to them is the malignity and rapidity of the case. They afford the only measure of its severity, and from them only can a correct prognosis be formed. The vomiting, purging, and cramps are considered as non-essential phenomena; for authentic cases of cholera are on record entirely divested of these symptoms; and the suddenness with which the disease sometimes extinguishes life is extremely remarkable. Instances of death taking place in two, three, four, or six hours, are by no means uncommon. When the disease broke out at Teheran, in May, 1846, Dr. Milroy states that those who were attacked dropped suddenly down in a state of lethargy, and at the end of two or three hours expired, without any convulsions or vomitings, but from a complete stagnation of the blood. In Bulgaria, during the outbreak of cholera in the allied armies, in the summer of 1854, the rapidly fatal character of the early cases was notorious.

The Urine in Cholera Reaction.—As the complete suppression of the urinary secretion in collapse, lasting for hours or days, is one of the most striking and peculiar features of cholera; so, its reappearance is amongst the earliest and most auspicious signs of beginning recovery. The first secretion in reaction mostly contains evidence of mechanical obstruction of the minute channels of the kidneys, and of the general death of the epithelia of the urinary passages. It also contains *the sign* of continued resistance to the blood current through the kidneys, in the form of *transuded albumen of the blood*. In many cases it carries small quantities of abnormal ingredients, which may perhaps be products or remnants of processes engendered by the choleraic process in the blood. The quantity is at first very small; urea being much diminished.

The Temperature in Cholera (as determined by Dr. Thudichum) falls steadily from normal to 5.4° or 7.2° Fahr. below it, and in most cases very rapidly. The lowest temperature is quickly reached in deepest collapse; and the minimum temperatures of all cases observed in the algide stage are below the lower limits of the fluctuation of health. The maximum temperature of the majority of cases observed are below the upper limits of the fluctuation of health. The lower the temperature, and the longer the duration of the algide stage, the higher and the longer continued is the temperature of the tepid stage, which does not exceed the normal, unless the temperature of the algide stage had previously sunk below 95° Fahr. But the temperature may for a short time reach 95° Fahr., or less, and yet the temperature of the tepid stage not rise above the normal. When the maximum temperature of a case of cholera remains below the normal average, the case will probably be fatal. Among thirty-nine cases observed, all such cases (seven in number) proved fatal. On the basis of thermometric observations alone, cholera may be divided into two stages,—the first or algide stage, from the beginning of symptoms to that period where temperature returns to the normal limits; and the second or tepid stage, in which temperature either remains within the normal limits, or rises more or less above them—in some cases even to febrile height, afterwards descending again to normal limits. Only observations taken in the *rectum* or *vagina* are of service as a standard of the temperature in *cholera*. Great differences between the axillary and rectal temperature is decidedly unfavourable; for, as soon as there are any

indications of *asphyxia*, the temperatures become more divergent, and the axillary temperature lower than normal. In death, during the *algide* stage, the temperature in the vagina and rectum has been as high as 108.32° Fahr.; and any considerable rise of temperature, or any considerable fall (especially with quickened respiration), indicates great danger. As temperature rises, the alvine discharges tend to diminish; but if the rise is high, rapid coma is likely to occur. *The less the temperature fluctuates the greater is the chance of recovery* (WUNDERLICH).

On the basis of all the pathological phenomena and clinical data the following seven stages of cholera may be distinguished:—(1.) *Fæcal diarrhœa*; (2.) *Choleraic diarrhœa and vomiting*, quick sinking of temperature; leading to, (3.) *Asphyxia or collapse*, in which lowest temperature is reached; (4.) *Reaction*, which may be defined as the cessation of collapse and the beginning of the re-establishment of the suppressed functions; (5.) *Torpid stage*, or secondary period of algide stage, in which (reaction notwithstanding) temperature remains below the lower normal limits, and then gradually or suddenly rises to the normal average; (6.) *Tepid stage*, in which, during continued reaction, temperature rises to normal or its upper limits, more rarely somewhat above; (7.) The *febrile stage*, only reached in cases where the entire algide stage has been very long, or where there are complications, or secondary lesions arising out of the choleraic process. Reaction does not always terminate the algide stage. For although, from the moment of the beginning of reaction, temperature rises somewhat in most cases, in exquisite cases it does not reach the lower limits of normal fluctuation. The algide stage is evidently continued into the state of reaction, and the tepid stage is the result only of continued reaction. Reaction begins mostly with absorption from the intestinal canal within thirty-six hours from collapse, possibly also with some actual secretions.

The influence of the temperature of the external air upon the temperature of the choleraic process was imperceptible during the observations recorded by Dr. Thudichum, as its range was very equable during the time of observation, the wards being mostly at 66.2° to 68° Fahr. The lowest temperature observed in any case which recovered was 92.8° Fahr. It is at present uncertain whether there is a minimum temperature below which the body (axilla) cannot be cooled down without fatal results. If there is such a temperature it will probably be about 92.3° Fahr. *A low minimum temperature is at present of less significance than a low maximum.* All cases of cholera, the temperature of which ranges persistently below the lower healthy limits, even if no very low minimum temperature was reached, seem to have a fatal prognosis. "It is all-important to remember that the thermometric observations recorded in this instructive section of Dr. Thudichum's report are exclusively of *external* temperature. In order to a complete understanding of the thermal phenomena of cholera, observations of this kind require to be supplemented by observations of *internal* temperature" (SIMON).

In forty cases of collapse treated in the London Hospital in the late epidemic, temperatures were measured, simultaneously in the rectum or vagina and in the axilla, by Mr. F. M. Mackenzie, assistant resident

medical officer of the hospital.* Another important set of double observations has been published in Germany by Dr. Güterbock. From these observations Mr. Simon concludes that "the choleraic affections of the bowels is a heat-making or inflammatory" process, on which the development of inflammatory fever, by circulation of blood from the inflamed parts, would as a matter of course attend, were it not that circumstances special to the disease (but accidental) suppress or circumscribe the manifestation. In a typical case of collapse the axillary thermometer shows a temperature perhaps little above 90° Fahr., while a thermometer in the rectum or vagina is marking a temperature high above the normal. With the superficial pulselessness of collapse, the suspicion cannot fail to arise that this vast difference of temperature between external and internal parts denotes mainly the failing blood supply of the former; a state which, in so far as it does not equally affect all parts in the aortic circulation, may not improbably "be deemed to depend on the muscular contractility of peripheral arteries." It is not yet determined whether the general temperature of the blood in cholera is not febrile. As regards this point, the following conclusions were arrived at by Surgeons A. Leith Adams and F. H. Welch during the epidemic at Malta, of which they have given so admirable a report (*Army Med. Dep. Report*, Vol. VI., 1864, p. 341):—

"(1.) That a strongly marked, rapid downfall from the average normal temperature, 97°, takes place soon after the setting in of the cholera symptoms, and the extent in proportion to the dose of the poison; the downfall being characterised in the healthy young, and up to middle age, by elevations and depressions, each succeeding one of the latter reaching a lower point than the preceding one; in the aged, weak, or debauched constitution, by an uninterrupted sinking of the thermometer. The average fall from normal temperature into collapse was 11°, the extremes 7° and 15° Fahr. (2.) That the highest temperature at which the general symptoms of collapse became apparent was 90°, the lowest 82°, the average 86° Fahr. (3.) That during the period of collapse the temperature underwent but slight variations in the aged, weak, or debauched; while in the young, and up to middle age, it was characterised by undulations. (4.) That the stage of complete collapse is not marked by any characteristic unvarying point of temperature. It would seem that an excessive dose of the poison is accompanied by a corresponding dose of heat; but when the vital stamina is deteriorated by drunken habits or delicacy of constitution, either the general symptoms of collapse are present when the thermometer makes no great fall, or an excessive lowness is reached with no corresponding general indications; while, on the other hand, a hardy, well-used constitution does not betray signs of failing until the respiratory function is much interfered with. The lowest point reached during life was 73°. (5.) That a general brightening up of the patient, unaccompanied by any change of temperature, often preceded the final downfall, and was exceedingly deceptive until appreciated rightly. (6.) That the general signs of reaction were pre-

* Mr. Mackenzie's notes of these observations are published in the third volume of the London Hospital Reports. Besides his observations, others, also made in the East London epidemic, are mentioned more or less fully in the same volume—viz., a few made in the London Hospital by Messrs. M'Carthy and Dove, and some made in the Wapping Cholera Hospital by Dr. Woodman and Mr. Heckford.

ceded by a marked elevation of the temperature; and when convalescence ensued, this reaction was characterised by fluctuations tending towards reinstatement of normal temperature. The average rise from complete collapse into full reaction was 6° , the extremes 8° and 4° . (7.) That when death ensued, whether preceded by reaction or not, the fall of the thermometer was most marked and rapid. (8.) That after death a rise of temperature ensued in the cases of great severity and quickness of course; but when the disease was prolonged, the patient falling into that senseless condition well expressed as 'death in life,' the contrary was the rule. (9.) The readings of the hands and epigastrium followed the breath's variations, though not always in the same ratio. As will be seen, the epigastrium specially showed a great tardiness in assimilating itself to the others, and was very tenacious of its heat.

"Thus, the thermometer indicated that in the aged and delicate the vital powers gave in to the poison, step by step, commensurate with the dose; collapse reached, a comparative quietness ensued, followed by the system asserting its superiority, or succumbing rapidly. The course from the onset to the termination was gradual, with no marked deviations. Not so, however, with the young and healthy. Although the system was compelled to give way to the attacks of the virus, it was not without a struggle; the collapse was marked by constant attempts at reaction, and this having once set in, the vital powers seemed to overreach themselves in their eagerness to resume their normal condition.

"As helps to prognosis, it may be said that a rapid and marked fall at the onset, a temperature below 86° , a further loss of heat during collapse, a setting in of general symptoms of reaction not preceded by rise of temperature of breath and hands, each and individually indicated badly; but it must be borne in mind that the previous habits of life were great influencing causes, and this more especially when drunkenness was the deteriorating agent. This vice, *per se*, appeared to predispose to the disease; but no words can express its baneful effects as demonstrated by the manner in which its devotees succumbed to the poison, even when in a minor state of intensity."

Duration of Cholera.—There are good reasons for believing that the cholera process runs a definite course of from twenty to thirty hours. While the "cold stage" is always present more or less, the "hot stage" of reaction may be absent, and is no essential part of the disease. It varies very much when present. In mild cases it is very short and scarcely appreciable. In severe cases it is long and protracted in this country. The phenomena of collapse appear not to be limited to any definite time. Some patients became collapsed very early; others not until vomiting and purging have continued several hours; and then often *not* protracted. In the milder cases the algide symptoms were scarcely, or even not at all marked; and all experience has shown that the collapse of cholera is not always present, while subsequent phenomena may be fatal.

Relation of Vomiting and Purging to Algide Symptoms.—When patients passed into collapse the vomiting and purging very greatly diminished, and in some cases entirely ceased. Some of the worst cases—cases which seemed almost sure to prove fatal—had very little, and often not any, purging. Thus cases characterised by the most continued purging and vomiting were not by any means the worst class of cases—for the most part the very opposite. It was the exception for such cases to pass

into collapse; and if so, the algide symptoms came on slowly. It by no means follows, because a patient is very much purged, and vomits violently and very frequently, that he is in a worse condition than another patient who vomits and is purged much less. Dr. Sutton brings forward evidence which clearly shows that the algide symptoms were not in regulated proportion to the frequency of the vomiting and purging. He shows that a patient may be purged hour after hour, may almost continually vomit, yet may not pass into collapse; whereas another patient is purged and has vomiting for two or three or five hours, and passes into deep collapse. If the collapse be dependent solely on the loss of fluid, it is difficult to understand why in the very class in which there is the protracted purging there is the least collapse; and even on the assumption that one patient passes in two or three evacuations more water than another does in double the number, and admitting that it is so in some cases, we are yet called upon to explain how it is that a patient who is not purged at all—that is, has had no discharge from his bowels—dies very suddenly; that another who has only been purged four times passes into collapse and dies; while others are purged twenty or thirty times without ever showing any well-marked symptoms of collapse. It is difficult to conclude that one patient passes more fluid in one evacuation than another does in twenty; and any explanation of this difficulty, to be satisfactory, must take into consideration not only the quantity of fluid withdrawn from the blood, but the rapidity with which it is withdrawn. In the worst forms of cholera a considerable quantity of water and other constituents of the blood are very suddenly withdrawn from the system; and there seems to be a decided relation between the severity of the collapse and the rapidity and violence with which the cholera process sets in and is carried on.

Evidences of Reaction.—When a patient dies in advanced reaction, or in complete reaction, the different organs of the body regain their weight, sometimes even weighing heavier than usual. This is especially the case with the lungs. In collapse the lungs and spleen weigh much lighter than normal. In favourable cases of reaction the wakefulness characteristic of true collapse gives way gradually to sleep. The colour returns, the pulse becomes distinct and more perceptible, and in some cases the patient may sleep quietly for some time. The thermometer is of all *single* guides the best, but cannot be absolutely relied on, unless the temperature in the axilla be very low indeed; and, in the rectum very high—*e.g.*, if the temperature in the axilla be 92° Fahr., and in the rectum 102° Fahr., the patient is still in collapse; but if the temperature in the axilla were 95° or 96° , imperfect reaction may be commencing (Dr. SUTTON). Some parts of the body appear to pass into reaction before others; and imperfect reaction is sometimes associated with bloody evacuations; and if a patient is pulseless, but with a natural colour and a greasy perspiring skin and a coated tongue, he will in all probability pass bloody evacuations, and then will certainly die. Dr. Sutton thus recognises a class of cases having the following symptoms:—The patient is seen lying on his back, eyes open, looking very wakeful, mind collected, voice weaker than natural, at times the typical choleraic voice, colour natural, lips natural, complexion greasy, tongue sometimes cold, livid, of grey

colour and covered with white fur; at other times the tongue is warm and coated with yellow fur. The hands are of a livid red colour, cold, and shrivelled. The temperature in the axilla is generally lower than usual. Respiration is laboured, and generally accelerated—often 25, sometimes 40, a minute. The pulse at the wrist may be only just perceptible, and very often such patients are pulseless; there may be no purging for hours together, and very little vomiting. There may be profuse perspiration, the face and hair wet with it. The patient may lie for hours like this, and even one or two days. Bloody evacuations appear on an average about twenty-eight hours after the violent symptoms of cholera set in. When the algide symptoms are most severe the reaction is greatest and most protracted. The longest reaction was seventeen days, the shortest sixty hours (Dr. SUTTON). In the mild cases the longest reaction was seven days, and the shortest twelve hours. The duration of suppression of urine is also in proportion to the severity of the algide symptoms. In one case no urine was passed, and none discovered in the bladder for six days and ten hours; in two cases none for five days, and in two none for four days. In the milder class of cases three days was the longest period of suppression, and the shortest ten hours.

Prognosis.—The mortality from cholera in all countries is very great, being from one to two-thirds of the number attacked. The period of the epidemic greatly influences the mortality; for, on the first onset, nine-tenths of all those attacked perish, then seven-eighths; and the proportion of deaths forms a gradually decreasing series of five-sixths, three-fourths, one-half, one-third; till, towards the close, a large proportion of those attacked recover. The uniformity of this law in every country affected with cholera, whether Europe, America, India, or China, is remarkable. The chances of recovery are much diminished in young children and in the aged—the age of greatest number of recoveries being from fifteen to twenty. The feeble in constitution, the anæmic, the sick, and the convalescent from other diseases, were in all cases the surest victims of cholera. But whatever the age of the patient, every case which becomes pulseless is likely to die.

Treatment.—There are few diseases for the cure of which so many different remedies and modes of treatment have been employed as in cholera. In considering the treatment of this disease there are three periods to be provided for,—(1.) The period of diarrhœa, which so frequently precedes cholera—“*premonitory diarrhœa*.” This indication of intestinal trouble must be distinctly recognised as the starting-point of all other symptoms, and the true source of danger. (2.) The algide period, or collapse; and, (3.) Period of reaction.

(1.) *The Period of Premonitory Diarrhœa.*—To check or arrest the diarrhœa is the practical result aimed at by a variety of formulæ. The evacuations ought to be checked as soon as possible. Those in which opium is the main remedy have acquired the most amount of confidence. The management of a case embraces the following conditions:—(1.) That the horizontal position of the body be maintained; and patients ought to take to bed at once. The necessity for the horizontal posture of the patient is, that it aids the efforts of the circulative powers, which tend to weakness. (2.) That the administration of opium, with or without cordial

stimulants, be at once commenced. With regard to opium, its dose must be regulated by, (a.) The extent of the nervous prostration; (b.) The rapidity of the dejections; (c.) The extent of vascular depletion. In the cases which present these phenomena in the extreme a much larger dose of opium is required to be given at the outset at one time than in the cases less urgent. (3.) That the induction of perspiration be brought about.

The extensive experience of my friend, Dr. Fergus, of Glasgow, in whose practice I was privileged to assist during the epidemic of 1849, led him to the conclusion (in which I concur) that "there is a first stage at which cholera is curable and preventible"—namely, that of laxity of the bowels *previous* to vomiting, spasms, or uneasiness of any kind. The relaxation of the bowels may not even amount to diarrhoea, and may be to the extent of only two or three stools a day, where one only was usual. Such relaxation was generally thought of no moment, being attended with no pain. On the contrary, the evacuations often gave a feeling of relief. This is the only stage at which opium is to be given, and that in a full dose. At this stage, in combination with a stimulant, it is often of the highest value. It is *only* to be given "*if the evacuations are still bilious, the pulse fair, and the skin warm.*" When vomiting, "rice-water" purging, and cramps set in (the phenomena of collapse) it is then too late for opium. Drs. Macpherson and Macnamara record, as the result of their extensive experience in India, that if administered at the proper stage no remedy is so effectual as opium; and the drug may be safely trusted to cure the premonitory diarrhoea of cholera. A dread has arisen of using opium from fear of secondary effects; but the same writers justly observe, that if it has a curative effect, we must not shrink from its use from the dread of ulterior consequences. "The vast majority of practitioners have found opium extremely useful in the early stages of the disease. Men like Twining and Parkes, who by no means considered the disease to consist merely in vomiting and purging, were agreed that opium is *the medicine* to be given in the commencement of the great majority of cases." It calms the brain, relieves the excessive feeling of constriction at the epigastrium, tends to counteract over-secretion from the mucous membrane, favours cutaneous transpiration, and assists the action of such other remedies as *stimulants, astringents, carminatives, or absorbents* (GUBLER). The following formula for pills, each containing a grain of opium, with stimulants, is well known as a most useful anti-spasmodic pill in the early stage of bowel evacuation:—

R. Pulv. Opii, gr. xii.; Camphor, gr. xxx.; Pulv. Capsici, gr. ix.; Spt. vine. rect., q. s.; Conserv. Rosar, q. s.; Misce et divide in pil. xii.

Moderate doses of opium or morphine, either alone or combined with stimulants (as the *pulvis cretæ aromaticus cum opio*) are sometimes sufficient to check diarrhoea. When medical men have charge of large numbers of people, as in the army, navy, prisons, workhouses, asylums, hospitals, and the like, it is incumbent on them to make frequent inspections of those under their care, and to seek out any cases of incipient diarrhoea. Responsible officers should be made to take notice of those who go more than once a

day to the water-closet at times when a cholera epidemic influence prevails. In military practice, frequent inspection of the men is of cardinal importance. Every man in a regiment should be seen at least three times a day by some medical officer, who should also visit the various guards. By walking down the ranks at roll-call, and picking out the men who show the earliest symptoms, cases are thus caught in the stage of premonitory diarrhoea, and saved. The following rules were drawn up by Dr. Fergus, now President of the Faculty of Physicians and Surgeons of Glasgow, and printed and hung up in prominent places, for the direction and management of large numbers of men, in the factories and offices of that city, over which he had charge when cholera was epidemic :—

“(1.) Do not be afraid of cholera, or make it the topic of conversation. Fear and all the depressing passions are injurious. (2.) Do not take brandy; it is not a preventative; and it does harm by disordering the action of the stomach and bowels. (3.) Do not make any change in your usual diet, if it is simple and of easy digestion; take it moderately, and at regular intervals, as long fasting is injurious; and carefully avoid excess in any intoxicating beverage. (4.) Take no excessive fatigue; if overheated, beware of any sudden chill, and see that the skin is kept comfortably warm. If the disease appears in winter, much benefit may be derived from wearing a flannel belt round the body, covering the stomach and bowels. (5.) As soon as cholera appears in a town, a bottle of *solution of morphia* or *laudanum* (and a graduated measure) should be kept in every house, place of business, factory, or wherever, in fact, there are a number of people gathered together. Persons travelling should always have it with them, or easy of access. During the existence of the epidemic, one person in each factory, &c., should take the charge of the health of the inmates, and should act as ‘house physician,’ warning all under his or her care to attend to the slightest relaxation of the bowels. He should remind them that the less pain the more danger, and therefore the more need of immediate and energetic action. (6.) Should the slightest diarrhoea occur, the individual so attacked should at once receive forty minims of *solution of morphia* or *laudanum*.* If from home or at business, the patient should be at once conveyed home in a cab, put to bed, and kept warm. If chilled, warm water bottles may be put to the feet. If the first dose has not checked the looseness, the patient should take a second, and then have a flannel cloth thoroughly dipped in turpentine, placed all over the stomach and bowels for from forty minutes to an hour, or a large, soft, warm poultice of linseed meal and mustard for one or two hours. If the second dose has not effectually checked the diarrhoea, and medical assistance has not arrived, a third dose may be taken. (7.) The patient must remain in bed two or three days after the diarrhoea is checked. *I insist strongly on this*, for the patient often feels so well that it is difficult to get him to attend to it. (8.) To relieve the thirst, a piece of ice may be given, or a mouthful of iced water, or soda water; but in no case must more fluid be taken at a time, and all food should be abstained from till from fifteen to eighteen

* If a measure is not at hand, a small teaspoonful. Of course this dose is for adults. Below that age the doses should be a drop for each year till twelve or fifteen, and after fifteen a drop and a half for each year, up to forty minims, or a small teaspoonful. More portable than laudanum, and of equal efficacy, would be pills composed of a grain and a half of opium, and a grain of cayenne pepper in each pill, three of which may be taken with safety, till medical assistance arrives.

hours after the opiate has been administered. *Then*, and for two or three days, the diet should consist of such food as rice, sago, arrow-root, Indian corn flour, tea and toast, &c.; about the third day beef-tea or chicken-soup might be taken. (9.) These rules are for the *first stage, and for it only*—i.e., the *diarrhœa*. If a person has neglected the first warning, and is in the second stage—i.e., has cramps, vomiting, and stools like rice-water, without smell—you should, till medical assistance arrives, place the patient in bed surrounded with bottles of hot water, and give him a little ice, and mouthfuls of soda and water. If the cramps are severe, you must rub the limbs with turpentine, or chloroform and oil" (*Glasgow Med. Journal*, 1866).

The following *cholera mixture* (as it was called) was proposed by the Board of Health during the prevalence of cholera, and was no doubt useful in many cases of diarrhœa:—

R. *Pulveris Aromat.*, ℥iii.; *Tinct. Catechu*, f℥x.; *Tinct. Cardam. Comp.*, f℥vi.; *Tinct. Opii*, ℥i.; *Mist. Crete*, preparat. ad f℥xx. Of this mixture the dose is one ounce.

Bulky doses of remedies are obviously objectionable. The usual remedies known as "astringents" (compared with each other, or with *opium*) have no decided influence for good. "Astringents," as such, have merely a negative effect.

Of the various preparations of *opium*, Dr. Macnamara gives preference, as Dr. Fergus does, to the *tincture*. *Thirty drops of laudanum in water should be administered as soon as possible after the first watery motion has been voided, and this dose must be repeated if the purging does not stop after the first dose has been given.* A pill composed of one grain of *opium* and two grains of the acetate of lead, to be taken every hour till the purging stops, is a useful formula, and more easily distributed than laudanum. Supposing thirty drops of laudanum to have been administered; and, that within half an hour the patient is again purged, Dr. Macnamara does not hesitate to repeat the dose at once. It is not advisable to wait until two or three motions have been passed. Watery painless purging should be at once stopped by such means. When the disease is endemic, and still more so when it is epidemic, every family should have a bottle of laudanum, to be resorted to at once after the first watery motion. If the motion is accompanied by a feeling of exhaustion, and a faintish sensation at the præcordial region, the dose of laudanum should be followed by "a stiff glass of brandy and water." "No more erroneous practice can possibly exist in India, or any other place where cholera exists, than that of allowing diarrhœa to go on upon the supposition that the attack is a bilious one, and that the purging is a salutary or a harmless process. More deaths from cholera have been occasioned from this than from any other mistaken notion on this subject. The issue is simply this:—Supposing the attack to be a bilious one, we stop it with opium, and may subsequently have to administer a dose or two of blue pills and rhubarb; or, perhaps, a little judicious starving may answer the same purpose. But if, under the impression that the diarrhœa is bilious, we allow it to run on, it may become, in the course of a few hours, the second stage of cholera, and our opium may then be of little or no use."

In the case of children the dose of laudanum must be regulated by the age of the child.

But there are certain cases in which, although the diarrhoea may be altogether checked by such remedies, yet the disease is not cured. Symptoms characteristic of the *algide* or collapse stage supervene. These are the cases which have been held to justify that method of treatment which has for its object elimination by the promotion of purging and of vomiting—excretion of the poison by the alimentary canal. The advocates of this method of treatment also lay stress upon the fact that those are the worst cases in which the diarrhoea is the least; and that those cases are the most hopeful in which diarrhoea and vomiting are the most severe. Dr. George Johnson's treatment by *castor-oil* has for its object the elimination in this way of the specific poison; and it may be said of it, that it is neither more nor less successful than other remedies of its class. It, as well as purely astringent mixtures, excites such loathing in most cases that it cannot be persisted in. Dr. Johnson agrees that "diarrhoea during an epidemic season ought not to be neglected even for an hour." He regards such diarrhoea as an indication of the presence of offending material in the alimentary canal—*e.g.*, (1.) Unwholesome, undigested food; or, (2.) A large and unnatural accumulation of the feculent contents of the bowel; or, (3.) Noxious secretions poured from the blood into the bowels, in consequence of the action of a specific blood-poison. To this latter process Dr. Johnson believes *choleraic diarrhoea* to belong. Therefore he lays down the following rule:—"Not to attempt by opiates, or by other directly repressive means, to arrest a diarrhoea while there is reason to believe that the bowel contains a considerable amount of morbid and offensive material; for, such offensive material must come forth before diarrhoea can permanently cease." Purging he considers the natural way of getting rid of the irritant cause; and the safest purgative he believes to be *castor-oil*. So far as meeting the conditions of examples (1.) and (2.) as above stated, the treatment cannot be improved upon; but that it is the best possible treatment to meet the preliminary diarrhoea or relaxation of the bowels in cholera cannot be regarded as established nor warranted by what we know of the pathology of the disease as set forth in the text. Assuming it to be proven that malignant cholera is a specific disease in which there is poisoning of the blood; and believing, as I do, that such is the pathology of the disease; and, seeing that the College of Physicians entertain a similar belief—for malignant cholera has been classed by the College in Section A of the GENERAL DISEASES, concerning which it is written, that "*it comprehends those disorders which appear to involve a morbid condition of the blood, and which present the following characters:—they run a definite course, are attended with fever, and frequently with eruptions on the skin, and are more or less communicable from person to person.*" Assuming the pathology of malignant cholera to be as here represented, I cannot admit that the best indications for the treatment of the disease are those which suggest an attempt to eliminate the poison by the promotion of purging and vomiting. I do not believe we can, by any remedy, nor by the over-stimulation of any eliminative function, ever get rid of any such poisons as those of *small-pox*, *measles*, *scarlet fever*, *typhus fever*, *enteric fever*, or any other general and specific disease-poison comprehended in Section A. No method of

treatment, based on the so-called principle of eliminating a poison, has ever effected a cure of any one of these diseases ; and I do not see any likelihood of the principle being more successful in cases of *malignant cholera* than with other diseases of the class. We have never yet been able to “cut short” any of these diseases by *emetics*, *purgatives*, or *diaphoretics*. Neither *emetics* nor *purgatives* have ever cut short *small-pox*, although, if we are to follow indications of treatment eliminative of poison, we ought to encourage such vomiting and purging which mark the initiative stage of most of the *exanthemata*. The early attempts to cure *small-pox*, *scarlet fever*, and *measles*, by promoting copious eruption on the skin, in the belief that the poison was so eliminated, proved a signal failure. The popular belief that “*better out than in*” was not found to be a safe principle of treatment in the management of those diseases. We have no eliminative method of treatment which will rid the blood of such specific disease-poisons ; just as we have no antidote which will destroy or counteract the influence of snake-venom when once it has got accidentally or artificially into the blood. We do our best when we try only to guide the patient through those specific diseases, *by moderating the violence of the expressions of the malignant phenomena, not by increasing them*. We cannot cure these diseases. The most successful results in the treatment of all of them have been got by those agencies which have led to modified and less malignant forms of disease, such as by vaccination, in regard to small-pox, and by sanitary improvements generally. We do not think of, and dare not attempt to promote, diarrhoea or specific lesion in the intestines during *enteric fever*. Why should we be induced to promote diarrhoea in *malignant cholera*, in the belief that we are thereby to free the blood from the specific poison of the disease ? We have no evidence that the body is freed of the poison in this way. The bowel discharges may be, and undoubtedly are, means (but not necessarily *the only means*) of propagating the disease ; but it does not follow that promoting their discharge frees the blood from poison either in *enteric fever* or in *cholera*. By moderating the catarrhal flow in *enteric fever*, it is not found that we aggravate the disease and lock up specific poison in the system ; certainly by moderating, curtailing, or restraining the diarrhoea of cholera, the only chance is left us of guiding the patient through that disease ; and OPIUM is the remedy which, by actual experience, alike in India and in this country, seems most worthy of reliance ; *but,—only at the commencement of laxity, or relaxation of the bowels, in seasons when cholera is epidemic.*

Treatment based upon conflicting theories (which are but theories after all) as to the nature of the disease, is greatly to be deprecated ; and on this point Dr. Fergus makes the following pertinent remarks :—

“The influence of the theories of Dr. G. Johnson and others is to be seen in the instructions recently issued by the Board of Supervision in Scotland—very excellent as to the sanitary part, but in the medical portion (like most things issued by a committee) an evident compromise. It is a compromise, I believe, between the facts and a theory. . . . We are told, under the fourth section, to take *castor-oil* or *rhubarb* and *soda* for looseness of the bowels, and afterwards the astringent mixture or pills—the former being intended as *eliminators* to promote the diarrhoea, the

latter to check it. If this advice is followed during an epidemic of cholera, the consequences may be very serious. It is well known that during an epidemic there is a general tendency to relaxation of the bowels, and that they become very susceptible of the smallest dose of even the mildest medicine. Any one who has had much to do with cholera must have frequently met with cases which appeared to be the direct consequence or result of a slight dose of medicine. The risk is that the *castor-oil* and *rhubarb* would drive most of the cases into the second stage of cholera, and then the use of astringents and opiates at that stage would increase the danger immensely. As to the remedies ordered, the *quantity* of *laudanum*—viz., five drops per dose—is too small, and there is no necessity for the chalk and catechu. Before the opiate could produce much effect, the stomach would be overloaded with the chalk and catechu, to the extent of inducing vomiting. The pills should be ordered after each discharge from the bowels; but people will find it easier to provide themselves with laudanum than to use complicated mixtures or pills.”

“With regard to castor-oil,” writes Dr. Macnamara, “I was acting as House Physician to King’s College Hospital, in 1854, when Dr. G. Johnson was treating his cholera patients on eliminative principles. I caught some of his enthusiasm on the subject, and came out to India the same year full of confidence and hope in castor-oil. These ideas were destined soon to pass through a severe ordeal; for in the following year I was left at Bhaugulpore in charge of a field hospital. I was the only medical man in the place, when cholera burst out among the Europeans and natives under my care: I went boldly to work with castor-oil, but it absolutely and completely failed; the mortality from the disease was fearful. I have since on several occasions tried castor-oil in cholera, but I have now finally abandoned it, having never seen any benefit arise from its use.” In the second or algide stage the object is to promote reaction and to keep it in moderation. If the patient is not seen till profuse discharges—rice-water-like—have taken place, the time for all active treatment has passed, and efforts must be directed solely to restoration and repair; and, if possible, to rendering the stools acid, by the following draught every half hour for four hours, and then to continue every hour (MACNAMARA):—

R. *Dilute sulphuric acid, and dilute acetic acid, of each ℥ xv.; carbolic acid, half a drop; water, an ounce and a half;* and opium may be added to the first and second doses, as existing circumstances may indicate.

To promote reaction in cholera and diarrhoea, the following *formula* has met with most universal approval in this country and in India. So highly is it valued, indeed, that it is ordered to be always in store, and in readiness in the “*Medical Field Companion*” of the army when on the march:—

R. Ol. Anisi., Ol. Cajeput, Ol. Juniper, ā ā ℥iss.; Æther., ℥ss.; Liquor Acid. Halleri, ℥ss.; * Tinct. Cinnam., ℥ii.; *misce.* *The dose of this mixture*

* The *Liquor* or *Elixir Halleri* consists of one part of concentrated sulphuric acid to three parts of rectified spirit. It is commonly employed in Germany in the treatment of typhus and allied diseases, in doses of five to twenty drops in solution (MURCHISON, l. c., p. 266).

is ten drops every quarter of an hour in a tablespoonful of water. An opiate may be given with the first and second dose, but should not be continued, for reasons already given.

The *nitrate of amyl* has also been recommended in the collapse of cholera by several physicians. It ought to be given internally, or by subcutaneous injection rather than by inhalation (Dr. T. L. BRUNTON, *Brit. Med. Journ.*, of Jan. 13, 1872). The indications generally followed are to treat the case as we should a similar state in typhoid fever, and to moderate the affection of the bowels by mild opiates, by enemata, and by sinapisms to the abdomen; also, to relieve the head by leeches and cold lotions, and subsequently, as the tongue becomes brown, to support the patient with wine, sago, strong broths, and a generally cordial treatment. During the reaction stage Dr. Andrew Clark, of the London Hospital, employed with great success a powder composed as follows:—

Hyd. c. Cretâ, gr. ii.; Pulv. Ipecac., gr. ss.; Pulv. Doveri, gr. iiss.; night and morning.

The cases most benefited by this remedy were those in which the tongue had become dry and hard, salines, lemonade, and chlorate of potash drinks being freely given. Mustard plasters or blisters to the nape of the neck were found to be of use by inducing the return of the urinary secretion.

The sick-room should be supplied with fresh air. Liquids should be assiduously applied to every surface capable of absorbing them, and the patient should be suffered to remain as free from officious treatment as possible. Heat applied in the dry form is to be avoided, but cloths moistened with hot water may be applied; or the patient may be wrapped up in warm, moist blankets, and hot bottles or bags of heated sand placed around his cold and benumbed body. After the temperature is restored, the surface should still be kept moist, by sponging from time to time, or by the use of the wet sheet, to moderate the reaction. Urgent thirst is one of the most distressing symptoms in cholera. There is incessant craving for cold water—doubtless instinctive, to correct the inspissated condition of the blood, due to the rapid escape of the *liquor sanguinis*. It was formerly the practice to withhold water—a practice as cruel as it is mischievous. Water in abundance, pure and cold, should be given to the patient, and he should be encouraged to drink it, even should a large portion of it be rejected by the stomach; and when the purging has ceased, some may, with much advantage, be thrown into the bowel from time to time (MACLEAN). Niemeyer recommends cold wet compresses to be frequently laid over the abdomen; and, also a grain of calomel every hour. Such compresses are said to be grateful to the feelings of the patient, whose sense of heat is great, although he may be cold to the bystander. The practice also is consistent with the lesions, especially the intensely pink vascularity seen in the intestines after death. The following as a drink is generally relished and retained upon the stomach:—"A raw egg beat up with half a pint of milk, and then mingled with about a pint and a half of water, adding as much salt as will give the whole an agreeable taste" (Dr.

ANDREW BUCHANAN). Or whey, milk and water, weak chicken soup, or any similar decoction, may be drunk *ad libitum*. *Enemata* of warm milk, repeated as often as expelled, are of the highest importance if the evacuations have ceased, by supplying an absorbable fluid, by mitigating abdominal pains, and diluting acid secretions. The secretion of urine may be promoted by dry cupping over the loins, by the use of solution of *chlorate of potash*, and the like. But suppression of this secretion is most to be dreaded where *opium* has been too freely used in the treatment. (See p. 738, for conditions under which opium is to be used.) In men of intemperate habits we often see, during the stage of reaction, obstinate vomiting of thick, tenacious, green paint-looking matter, probably bile-pigment, acted on by some acid in the stomach or alimentary canal. It is a symptom of evil omen, and often goes on uncontrolled until the patient dies exhausted, and this although all other symptoms may promise a favourable issue. It may last for a week, resisting all remedies, and proving fatal when the urinary secretion has been restored, and all cerebral symptoms have subsided. *Alkalies* in the effervescent form, free stimulation of the surface, and *chloroform* in small doses, offer the best hope of relief for such cases. The patient should be nourished more by the bowel than the stomach when vomiting is present. *Ice* should be given *ad libitum*, where it can be obtained, not only to dissolve in the mouth, but to swallow in pieces of convenient size (MACLEAN).

A plan, perhaps peculiar to this country, which was practised to bring about reaction (when the inefficiency of medicines was generally admitted), was an injection into the veins of the patient of a solution of half an ounce of *muriate of soda*, and four scruples of *sesquicarbonate of soda*, in ten pints of water, at a temperature varying from 105° to 120° Fahr. This solution was injected slowly, half an hour being spent in the gradual introduction of the ten pints, and the immediate effects of this treatment were very striking. The good effects were rapid in proportion to the heat of the solution, but a higher temperature than what is stated could not be borne. After the introduction of a few ounces, the pulse, which had ceased to be felt at the wrist, became perceptible, and the heat of the body returned. By the time three or four pints had been injected, the pulse was good, the cramps had ceased, the body, that could not be heated, had become warm, and instead of a cold exudation on the surface, there was a general moisture; the voice—before hoarse and almost extinct—was now natural; the hollowness of the eye, the shrunken state of the features, the leaden hue of the face and body had disappeared; the expression had become animated, the mind cheerful, the restlessness and uneasy feelings had vanished; the vertigo and noises of the ear, the sense of oppression at the *præcordia*, had given way to comfortable feelings; the thirst, however urgent before the operation, was assuaged, and the secretion of urine restored, though by no means constantly so. But, these promising appearances were not lasting; the vomiting continued, the evacuations became even more profuse, showing that the remedy did not touch the root of the evil. The patient soon relapsed into his former state, from which he might again be roused by a repetition of the injection. The amendment,

however, was transient, and the fatal period was not long deferred. Of 156 patients thus treated at Drummond Street Hospital, Edinburgh, only twenty-five recovered,—a lamentably small proportion ; and, small as it is, it seems doubtful if the recoveries were final or complete (MACKINTOSH, *Principles of Pathology*, p. 365). Nevertheless, injection of fluid by the veins ought still to be persevered in as a remedy in the right direction—with due regard to temperature—as a means of *prolonging life*. A trial made by Surgeon-Major E. Y. Kellat at the close of an Indian epidemic is deserving of more extended experience, namely :—To inject *distilled water* at a temperature of 99° Fahr. hypodermically, into the connective tissue of the body, or into the cavity of the peritoneum. He thus aimed at establishing an artificial anasarca or an ascites ; experience having shown that of several instances of cholera attacking persons suffering from such conditions, the effused fluid became rapidly absorbed during the course of the cholera—the patients recovering. The distilled water he thus proposes to inject hypodermically, must *filter* through the living tissue before it can reach the circulation ; and hence be more fitted to mingle with the blood, than when thrown into the veins directly from contact with air. Water is the cardinal want. The supply of it fails first : and thus introduced it avoids contact with the morbid surface and secretions of the stomach and bowels, which are barriers to its absorption in that direction.

Stimulants tend to irritate the stomach, and are of no use in exciting the heart to any salutary action. Employed as in typhus, they are worse than useless. All violent remedies are greatly to be deprecated. *Strychnia*, *prussic acid*, *tobacco*, *galvanism*, *boiling water*, the *actual cautery*, or *firing the spine*, or *bags of ice to the spine*, or *injections of spirits*, are proven by trials to be of no use ; nor can their recommendation be supported except upon baseless theories, having no rational relation to the pathology of the disease.

To generalise on the subject of remedies in cholera, the broadest conclusion seems to be, that remedies with an *acid*, rather than an *alkaline* or *neutral reaction*, have been the most beneficial.

Prevention of Cholera.—The following summary, from the writings of Dr. Parkes and Mr. Simon, will best conclude this account of cholera. The importance of the topics noticed, and the uncertainty that yet enshrouds many of them, may justify, it is hoped, the amount of space which the subject has taken up :—

“For the first time in the history of cholera a new system of prevention has been brought largely into play in Europe—viz., the addition to the discharges of a presumed disinfecting substance. This plan, of course, is based on the belief that the principal (perhaps the only) mode of spread is by means of the putrefying evacuations ; and the results obtained by it give certainly some strong evidence in favour of this opinion. In this country the difficulty has been to make the public (and, in some cases, even the medical men) sensible of the importance of this plan, and of the necessity of giving it a complete trial. In some cases in which it has really been fairly tested, it appears to have arrested the spread of the disease, as at Bristol ; and Dr. Budd’s paper in the *Brit. Med. Journ.*, April 13, 1867, gives good evidence on this point. In Southampton also, I be-

lieve, the spread was limited in this way, though it was not arrested so perfectly as at Bristol. In London, and several provincial towns, the method was also tried more or less fully. In Germany, owing to the influence of Pettenkofer, the disinfecting plan was also brought into play, and the system followed has been described by that chemist.* Without analysing all the evidence, I proceed to give the most important practical rules. The dangerous period of the choleraic stools is supposed to be when they become *very ammoniacal*. This occurs sometimes immediately they are passed, but usually not to any extent for some time. It is thought (but of course exact scientific proof is not readily attainable) that anything which makes and keeps them acid prevents the changes which cause the poison. The three principal means of doing this are the use of *carbolic acid*, and *sulphate of iron* (with or without *permanganate of potassium*), and the *salts of zinc*. Each has its advantages, and all may be used. The *carbolic acid*, from its liquid form and from its volatility, is excellently adapted to purify air, and to be used when surfaces are to be washed. It is also useful for sewers and closets. The *sulphate of iron* in substance and strong solution is better adapted for being put in the utensils in a room, as it has no smell, but it may be equally used for sewers and for watering streets. The *sulphate of zinc* (for the *chloride* is too dear) is better adapted for being put on linen or on floors, as it does not ironmould the linen like the *sulphate of iron*. The *carbolic acid* has not been used much in Germany, as it is still too dear; but Pettenkofer makes an observation of importance—viz., that when added to *sulphate of iron* the mixture seems to have more power of preventing *ammoniacal development* than either substance separately. If so, it might be desirable, as a matter of practice, to use the two together as much as possible. The *salts of zinc* (*sulphate* or *chloride*) may be also used, but are perhaps not so good, and in some forms are dearer than the iron salts. *Chloride of lime* does not prevent the *ammoniacal change*, and appears altogether less useful. The quantity in which these substances must be used is as follows:—For each healthy person, daily, about three-quarters of an ounce of *sulphate of iron*, or one drachm of strong (but impure) *carbolic acid*, is sufficient. This amount will entirely prevent any decomposition of the *feces* for several days. In a town, therefore, where sewers are used, the above amount of *sulphate of iron*, or *carbolic acid*, multiplied by the number of persons, should go into the sewers daily, and, if possible, should be passed in from the houses, so as to act on the house-drains as well as on the main sewers. If the place is not sewered, then the disinfectants should be added to the cesspools, middens, latrines, or whatever plans may be in use. If both *sulphate of iron* and *carbolic acid* are used, which is to be recommended, half the quantity of each should be employed. The iron should be dissolved in a good deal of water.

"In Southampton, in 1866, carbolic acid was chiefly used; and the average amount was about twenty gallons daily for a town of 50,000 people: it certainly appeared useful. If an aerial disinfection is needed, *sulphurous acid* (obtained by burning sulphur) is perhaps the best. *Nitrous acid fumes* are certainly very powerful; and one or other of these substances should be used for half an hour daily in all privies or latrines. For washing clothes the iron salts are not applicable, as they stain linen. *Carbolic acid* gives a disagreeable smell. Either a watery solution of *sulphurous acid*, or a solution of zinc salts, should therefore be used. Baking

* "Cholera Regulativ," von H. Griesinger, M. Pettenkofer, and C. A. Wunderlich, *Zeitschrift. für. Biologie*, Band. II., p. 435.

the clothes, at a temperature of 250° , or boiling, should be used. In hospital wards, dead-houses, &c., it is a good plan to sprinkle sawdust on the floors, and to moisten it with the weak *carbolic acid* (one part of crude acid in sixty or eighty of water). These measures should be commenced when cholera is apprehended. Every privy and sewer should have twice daily the mixed *carbolic acid* and *sulphate of iron* solution. If cholera is introduced, the amount should be doubled in the privies of all the adjacent houses, while the closet of the affected house should never act without a portion of the disinfecting liquid being placed in it. If the disease breaks out, a plan recommended by Dr. Budd is worthy of imitation—viz., to place a layer of *carbolic acid* powder (carbolic acid and lime) in the bed, under the breech of the patient. The disinfection in this way of the closets and privies of hotels, railways, and workshops, should be commenced very early."

A saline antiseptic, "chloralum," has been recently much commended; and on the subject of prevention Mr. Simon has also made some valuable suggestions:—

"Wherever cholera is known to exist too much importance cannot be attached to the precaution of thoroughly disinfecting, without delay, all discharges from the stomach and bowels of persons suffering under the disease, and of disinfecting or destroying all bedding, clothing, towels, and the like which such discharges may have imbued; and, of course, neither choleraic discharges, nor any slops which may contain traces of them, should ever (even when supposed to be disinfected) be cast into any position from which they may get access into drinking water. But, although the duty of observing those precautions is one which ought never to be neglected, populations cannot prudently stake their lives on the chance that it will be completely fulfilled for them. Apart from all questions of negligence, the degrees of cholera are too many, and the slight and incipient cases far too apt to escape observation, for any such defence against its infection to be more than partial. And, therefore, the main object for endeavour must be to secure everywhere such local circumstances that the infective material, though not disinfected, would be unable to spread its influence among the population. The dangers which have to be guarded against as favouring the spread of cholera infection are particularly two. First, and above all, there is the danger of water-supplies which are in any (even the slightest) degree tainted by house refuse or other like kinds of filth; as where there is outflow, leakage, or filtration from sewers, house-drains, privies, cesspools, foul ditches, or the like into springs, streams, wells, or reservoirs from which the supply of water is drawn, or into the soil in which the wells are situate; a danger which may exist on a small scale (but perhaps often repeated in the same district) at the pump or dip-well of a private house, or, on a large and even vast scale, in the source of public waterworks. And secondly, there is the danger of breathing air which is foul with effluvia from the same sorts of impurity."

"Temporary security must, as far as practicable, be sought in measures of a palliative kind:—(a.) Immediate and searching examination of sources of water-supply should be made in all cases where the source is in any degree open to the suspicion of impurity; and the water both from private and public sources should be examined. Where pollution is discovered everything practicable should be done to prevent the pollution from con-

tinuing, or, if this object cannot be attained, to prevent the water from being drunk. (b.) Simultaneously there should be immediate thorough removal of every sort of house refuse and other filth which has accumulated in neglected places; future accumulations of the same sort should be prevented; attention should be given to all defects of house-drains and sinks through which offensive smells are let into houses; thorough washing and lime-washing of uncleanly premises, especially of such as are densely occupied, should be practised again and again. (c.) Disinfection should be very freely and very frequently employed in and round about houses wherever there are receptacles or conduits of filth, wherever there is filth-sodden porous earth, wherever anything else, in or under or about the house, tends to make the atmosphere foul.

"In the absence of permanent safeguards no approach to security can be got without incessant cleansings and disinfections, or without extreme and constant vigilance against every possible contamination of drinking-water."

Then again as to the risk of contagion Mr. Simon writes:—

"Cholera in England shows itself so little contagious, in the sense in which small-pox and scarlatina are commonly called contagious, that, if reasonable care be taken where it is present, there is almost no risk that the disease will spread to persons who nurse and otherwise closely attend upon the sick. But cholera has a certain peculiar infectiveness of its own, which, where local conditions assist, can operate with terrible force, and at considerable distances from the sick. It is characteristic of cholera (and as much so of the slightest choleraic diarrhoea as of the disease in its more developed and alarming forms) that all matters which the patient discharges from the stomach and bowels are infective. Probably, under ordinary circumstances, the patient has no power of infecting other persons except by means of these discharges, nor any power of infecting even by them, except in so far as particles of them are enabled to taint the food, water, or air which people consume. Thus, when a case of cholera is imported into any place, the disease is not likely to spread, unless in proportion as it finds locally open to it certain facilities for spreading by indirect infection. In order rightly to appreciate what these facilities must be, the following considerations have to be borne in mind:—First, that any choleraic discharge, cast without previous thorough disinfection into any cesspool or drain, or other depository or conduit of filth, infects the excremental matters with which it there mingles, and probably more or less, the effluvia which those matters evolve; secondly, that the infective power of choleraic discharges attaches to whatever bedding, clothing, towels, and like things have been imbued with them, and renders these things, if not thoroughly disinfected, as capable of spreading the disease in places to which they are sent (for washing or other purposes), as, in like circumstances, the patient himself would be; thirdly, that if, by leakage or soakage from cesspools or drains, or through reckless casting out of slops and wash-water, any taint (however small) of the infective material gets access to wells or other sources of drinking-water, it imparts to enormous volumes of water the power of propagating the disease. When due regard is had to these possibilities of indirect infection, there will be no difficulty in understanding that even a single case of cholera, perhaps of the slightest degree, and perhaps quite unsuspected in its neighbourhood, may, if local circumstances co-operate, exert a terribly infective power on considerable masses of population."

The inquiries made through the Epidemiological Society show that special hospitals, or special wards in hospitals, ought to be devoted (with a separate and special staff of attendants for day and night duty, or more frequent periods of relief) entirely to the management of cholera patients.

With regard to India much may still be expected from a thorough improvement of its sanitary condition; and especially of the filthy towns and *bustees*, or native villages, constructed without any plan or arrangement, without roads, without drains, ill-ventilated, and never cleaned—the abodes of misery, vice, and filth—the nurseries of sickness and disease. These are the localities within the endemic area of cholera which require special attention and improvement in the first instance; and which are now receiving the attention of the Indian authorities through their special sanitary administration.

DIPHTHERIA.

LATIN EQ., *Diphtheria*; *Synanche Contagiosa* (SENATOR); FRENCH EQ., *Diphthérie*; GERMAN EQ., *Diphtherische Entzündung der Rachenschleimhaut*—Syn., *Diphtheritis*; ITALIAN EQ., *Difterite*.

Definition.—*A specific disease commencing with fever, and producing a membranous exudation in and upon the mucous membrane (of one or other, or of all of it) covering the tonsils, uvula, and soft palate, the root of the tongue, the larynx and air passages, the posterior wall of the pharynx, or the nasal cavities. The disease is attended with great prostration of the vital powers; also, by a very early appearance of albumen in the urine, which may continue for a very short time only, or may become persistent. In many cases a remarkable series of nervous phenomena are apt to supervene, characterised by progressive paralysis, and sometimes by fatal syncope. The disease is contagious, and apt to be epidemic.*

Pathology.—(*a.*) *Historical Notice.*—The writings of the older physicians prove that from time to time the disease to which we now give the name of “Diphtheria” has been epidemic, or at least very common, in many parts of England; and the accounts of it show that it has preserved its essential character and nature from age to age. Ever since the end of the “sixteenth century, diphtheria has been observed in every region of the Old and New World.” At first it continued for a time in Spain; and during nearly forty years it was noticed in different parts of the Peninsula. Rather later, all Italy was successively afflicted by it. Towards the middle of the last century especially, epidemics of the disease have occurred, less general and less prolonged, but more multiplied, in England, in France, in Sweden, and in America, and particularly at New York and Philadelphia. It terminated the life of the celebrated Washington and of the Empress Josephine. The outbreaks have usually been limited in extent, sometimes not spreading beyond a single dwelling, building, village, or quarter of a large town. The disease, however, has only been known by its present name since Bretonneau investigated the epidemic at Tours in 1818, and gave to it the name of *Diphtheritis*, which has been subsequently modified to *Diphtheria*. The latter term has the

advantage of being the shorter word ; and is that adopted by the Registrar-General and by the College of Physicians. Etymologically, the terms are derived from *διφθέρα* *vel* *διφθερίς*, signifying the prepared skin of an animal; while *διφθερίτης* *vel* *διφθερίνης* signifies that which is covered with a fur, or with a leathern coat. It is even now sometimes described in medical certificates as "putrid sore throat" or "Cynanche maligna."* It prevailed as a severe epidemic in the north of France in 1855-57 and was fatally prevalent in England in 1859. Since that time many monographs have been written on the subject in our own country, generally recognising the disease as specific, infectious, and sometimes epidemic. It first appeared as a separate heading in the Registrar-General's nosological classification in the Report relative to the year 1859. But it was M. Bretonneau's investigations and writings which gave to diphtheria the characteristics of a substantive affection. The literature of the various forms of sore throat will show how the disease has been confounded with "*the croup*" on the one hand, and *malignant scarlet fever* on the other. Recognising the existence of *diphtheria* expressing itself first by a lesion in the larynx, and so constituting *croupal diphtheria*, or *diphtheria* with the *local symptoms of croup*; or, to put it in another form—recognising that there is a form of *croup* due to *diphtheria*, there is yet abundant evidence to show that true *croup*—"the croup"—is a different disease—a disease different from *diphtheria*, which is named as such by the College of Physicians, and which will be described in its proper place, together with *laryngeal catarrh* and *acute laryngitis*, each of which has also been confounded with *croup*. Of the historical accounts of diphtheria, the best is that given by Mr. William Squire, L.R.C.P., London, in Reynolds' *System of Medicine*; and Mr. John Chatto's valuable account of the bibliography of diphtheria since Bretonneau's first essay will show the confusion of ideas entertained and expressed upon *diphtheritic croup* and *sore throat*. One source of confusion is to be traced to the different uses of the term *croup*, a term which the French have always used in the sense

* Dr. H. Senator of Berlin now proposes the term *Synanche Contagiosa*, as there is at present no uniform designation for it—none used in the same sense by all physicians, none which embraces all the various symptoms. On the one hand, the term diphtheria includes the idea that the course of the disease must always be characterised by the formation of a membrane, which is not always the case. On the other hand, there is a difference of opinion even on this point; diphtheria being applied by some only to certain apparent coatings of pseudo-membranes adherent to, and not separable from, the mucous membrane; while others, on the contrary, lay stress upon the condition of membranes rather than on the place where they are found, and apply the term diphtheria only to that disease which leads to their formation in the *pharynx*. The same term is also employed to express a certain condition of wounds and ulcers, irrespective of the part of the body in which they occur, without regard to any possible relationship with the disease under consideration here. The term *diphtheria* is used sometimes in a clinical, sometimes in an anatomical, and sometimes in an etiological sense; and the same idea is not always associated with it, even by those who use it in one and the same anatomical sense. With such confusion in the use of terms, it is difficult, if not impossible, to come to an understanding with regard to the many points in dispute connected with the theories of the disease about to be described. There is scarcely any other field of pathology in which the confusion is so great—a confusion which will last until a general determination is arrived at to abandon all terms at present conventional, though used in different senses; and to select others less ambiguous, involving as little pre-judgment as possible, and not compelling the adoption antecedently of any particular idea as to points in dispute, or as to anatomical or etiological conditions.

of diphtheria.* “Croup” simply means roughness of breathing, and has no more pathological significance than the word “cynanche,” which formerly comprehended all diseases of the throat and air passages. But the disease known and to be described as “the croup” is quite another thing from “diphtheria.” The two diseases are now as much requiring recognition and differentiation as typhus and typhoid fevers required differentiation before Sir Wm. Jenner called attention to their differences rather than their resemblances.

(b.) *Causation and Propagation.*—Like enteric fever, diphtheria must be regarded as one of the “filth diseases;” and the question of the *de novo* origin of its cause seems to be in the same position as that of enteric fever—*i.e.*, whether its poison is produced *de novo* in decomposing masses of

* On the subject of nomenclature Sir John Rose Cormack thus writes:—“Confusion is apt to arise in the minds of the uninitiated students of foreign medical literature when they begin to compare French and German authorities on diphtheria and croup. To avoid bewilderment, it is necessary to bear in mind that the physicians of France and Germany respectively attach absolutely different meanings to these words. The great majority of French writers, considering the question from a purely clinical point of view, regard diphtheria as a disease which is general, specific, and contagious, specially characterised by the formation on the affected parts of false membranes, which, when removed, are reproduced, and which have a great tendency to extend to neighbouring parts, and to become more or less general. French writers designate all pseudo-membranous laryngeal affections by the name of croup; but some of them limit the use of the term croup to diphtheritic laryngeal membranous exudations. German writers, on the other hand, adopt a purely anatomical division. Completely abandoning the original signification of the Scottish and Swedish word, by which was simply meant croupy or crowing respiration, they apply the term croupal to all affections which are characterised anatomically by the formation of a fibrinous exudation or superficial false membrane upon a subjacent mucous surface in a state of integrity; hence we find them speaking of croupal nephritis and croupal gastritis. They apply the term diphtheritic to deep or interstitial pseudo-membranous exudations, accompanied by infiltration and elimination of the parts subjacent to the false membrane. An example will make clear the essential difference between the German and French nomenclature. The term croupal pneumonia is generally employed in Germany to designate acute pneumonia with fibrinous exudation (*pneumonie aiguë, franche, avec exudation fibrineux*). Some German authors, looking to the pseudo-membranous and ulcerative character of dysentery, give it the name of intestinal diphtheria. As to the unfortunate term *croup*, it would be well to expunge it from British scientific medicine, or be careful to employ it unambiguously, which can always be done by the use of a descriptive prefix, such as diphtheritic, herpetic, spasmodic. The word croup was first employed in scientific medical literature in 1765, by Dr. Francis Home of Edinburgh, in his *Inquiry into the Nature, Cause, and Cure of Croup*. Till then, it was only the popular term in Scotland and Sweden for stridulous breathing, for crowing or croupy respiration. An attentive perusal of the cases detailed by Dr. Home leads to the impression, that the epidemic described by him was an epidemic of diphtheria, and identical with that studied by Bretonneau at Tours in 1826, and described by him under the name of diphtheritis, which he created. It must always be remembered, however, that, although Bretonneau created the name and indicated the true pathology of the disease, his first work—*Recherches sur l'Inflammation spéciale du Tissu Muqueux, en particulier sur la Diphthérie*, published at Paris in 1826—was far from complete. It was, however, a grand unveiling of the truth, and the foundation of one of the most brilliant clinical inquiries which adorn the history of medicine. The term croup was not vulgarised in France till the publication, in 1809, of F. Ruette's translation of Home's *Inquiry*. In the same year, Ruette published at Paris a translation from the English of an account by Starr, in the *Philosophical Transactions* for 1750, of an epidemic *morbus strangulatorius*, which he had observed in 1748 at Liskeard, in Cornwall. In concluding this notice of the history and meaning of the terms diphtheria and croup, I may add that, in the current medical literature of Italy and Spain, I find that croup and diphtheria are employed exactly as in France, with this occasional difference, that, in place of croup, the Italian writers often use the term laryngeal diphtheria” (*Brit. Med. Journ.*, 1875, Vol. I., p. 544).

vegetable and animal tissue, or whether our sewers and cesspools are simply the hotbeds in which the living contagia are rapidly propagated? As with enteric fever, so with diphtheria, it is a question which cannot be solved from cases occurring in large cities (see page 358, *et seq.*, *ante*). For though many such cases must have been exposed to cesspool gas or sewer air, it is fair to assume that the specific poisons of all our contagious and specific diseases are constantly present in our air. Nevertheless, "filth" in its various forms, especially excrementitious, has been shown to be an important element in the development and propagation of cases of diphtheria; but, unlike most other diseases of this kind, it is far more fatal in rural than in urban districts (FARR). Such an example is to be found in the history of an epidemic in the Warwickshire village of Brailes, situated in the rural sanitary district of Shipston-on-Stour, which was the subject of special investigation by Mr. W. Power, in which 48 per cent. of the cases proved fatal. In an analysis of 112 cases of diphtheria, one-fourth of the cases occurred on the *first-floor* of houses *without cellars*; one-third on *first-floor with cellar*, showing that *dampness* with filth is a powerful predisposing cause. With regard to the season of prevalence—the year being divided into a warm, dry season, from April to September, in which there were thirty-seven cases; and a cold, damp season, from October to March, in which there were seventy-five cases—December having the largest number, twenty-six; June and July the least, each having three. (Dr. G. A. VAN WAGENEN, *New York Medical Journal*, Vol. XXVIII., p. 588, 1878.) These figures correspond with those of Wibmer for the epidemic of 1868-69 in Berlin.

The disease is contagious or infectious; and family constitution seems to influence its development, progress, and results. Most observers agree in assigning to diphtheria a very short period of incubation. It may be stated positively to occupy from two to five or seven days (OERTELS, SENATOR); and from twelve to twenty-four hours after artificial inoculation upon the surface of wounds, a greyish-white discoloration may be detected—a dirty greyish layer, with other signs of infection. In exceptional cases the period extends to three or even four weeks (SENATOR, W. CLAES, JENNER, GREENHOW, SANDERSON). Of the 112 cases analysed by Dr. Wagenen, fifty-nine followed direct known exposure; thirteen were probably exposed, the disease being near by; while forty knew of no exposure. Thus almost two-thirds can be accounted for by contagion; and as the chances of a child exposed are as three to one in favour of contagion, diphtheria must be acknowledged to be a *very contagious disease*, the contagiousness bearing a pretty constant relation to the malignancy of the case. As the breath becomes offensive the danger increases; and a malignant case will *usually* engender another malignant case. As years increase the danger from contagion decreases; and as a single attack does not seem to exhaust susceptibility to this disease, it may be classed as a disease of childhood. Direct contagion by inoculation is only probable. There is no reason to suppose that greater power of propagation attaches to the fibrinous exudation than to the general secretions and exhalations from the sick. The presence of one sick person in a house is sufficient for communication of the disease to the susceptible, however carefully kept apart. Therefore, to some extent, the material of infection must

be diffusible in the air, so that by some the danger of infection is greater from this source than from direct contagion (SQUIRE). Like scarlet fever and erysipelas, the specific *miasm* of *diphtheria* clings, with great tenacity, to particular houses or apartments. Convalescents from diphtheria also retain the power, for an indefinite (unknown) period,—the power of disseminating the disease (SQUIRE, JENNER).

In relation to other diseases there are cases which show that where diphtheria is said to have complicated scarlet fever, the resulting contagion set up diphtheria (only) in others. Zum Sande relates in full the results of several successive infections in the members of a large family. Some were attacked by diphtheria only; in others a scarlet fever eruption (followed by desquamation) broke out on the eighth day of the former affection; but he does not consider these to be cases of scarlet fever, inasmuch as the eruption did not precede, but broke out eight days after the pharyngeal diphtheria; and the contagion of the patients who presented this eruption set up a fresh simple diphtheria. (*Berlin Klin. Woch.*, 1873, 315; and *B. Retrospec.*, 1873-74, N.S.S., 1075.)

(c.) *Morbid Anatomy*.—The prominent and characteristic lesion in diphtheria is a specific exudation upon certain parts of the mucous surfaces, also upon wounds and cutaneous abrasions. As regards the localisation of this exudation, in 104 cases analysed by Dr. Wagenen it *began* on the tonsils, or some part of the pharynx; and, of these, thirty-three extended up into the posterior nares, and fourteen downward into the larynx. In six the exudation *began* in the nares; in all of which it extended into the pharynx; but in none of them did it reach the larynx. In four it *began* in the larynx, of which in two it extended upwards to the pharynx. This characteristic local exudation is specific, and has its primary seat on the mucous membrane, covering a tonsil, or the arches of the palate, or the posterior surface of the soft palate, the uvula, the nares, the pharynx, or the larynx. It thus constitutes the "false membrane," which "may extend continuously from the nares to the bronchi—may be moulded on to any anfractuosity of the nasal passages, which may line the whole pharyngeal cavity, adhere to both surfaces of the epiglottis, cover the interior of the larynx, form a cylindrical cast of the trachea, and may thus be removable from even the smaller bronchi. More rarely it is found to extend in this manner throughout the œsophagus" (SQUIRE). The membrane has been found even in the gall bladder, on the vulva, and in the vagina.

At first there is redness and swelling; and the normal mucous secretion is so altered in its physical properties, that it adheres by its own increased viscosity to the mucous membrane. A white or grey patch is seen to form on the membrane, which indicates the presence of a layer of lymph on the reddened surface. This layer of lymph may thus spread from one or from several centres over the reddened surface; and this redness may involve the whole mucous membrane within reach of the eye. If the lymph be torn from the mucous membrane, a raw, bleeding surface is exposed, which in a few hours is again covered by a new layer of lymph. The lymph of *diphtheria* has a variety of appearances. Sometimes it is granular, with very little consistence or tenacity. Sometimes the part is covered with a pulpy substance of a white or grey colour; but this pellicle is constant in some form or other. In microscopical characters it does not

appear that this "fur," "pellicle," or "false membrane" of diphtheria can be distinguished from the concrete exudation of blistered surfaces, or that which forms in the angina of scarlatina (EMPIS). Generally it may be stated that there is the greatest possible variation as to the extent, the consistence, the colour, and adherence of the pellicle. The diphtheritic lesions begin in close contact with blood-vessels in the mucous membrane beneath the epithelium, the submucous layer of which is transformed or replaced by the exudation. The exudation which thus forms the diphtheritic membrane (a false membrane) cannot be detached without leaving a bleeding surface; and if detached the exposed surface is very quickly recovered with another layer of exudation. So long as this exudation continues and exists, the surface will not cicatrise, neither will the membrane become organised. Unhealthy secretion is discharged, resulting from the interstitial death of tissue invaded by the exudation, and so there is sometimes a superficial ulceration. Decomposition, however, takes place rapidly in the exudative membrane itself; and hence the very fetid effluvia proceeding from the mouth of the diphtheritic patient. Sometimes the particles of the lymph exudation are so thin, soft, and separated from each other, that the term membrane can scarcely be correctly applied to it. At other times it is tough, elastic, and as much as an eighth of an inch in thickness. In one case the lymph resembles cream in consistence, in another it resembles wash-leather; and between the two extremes we meet with all intermediate conditions as regards consistence and tenacity. Pus, granular corpuscles, oleo-protein granules, and epithelium constitute the bulk of the softer forms of the so-called lymph; while such fibres as we see in the buffy coat of blood-coagula constitute the bulk of the toughest variety of the lymph pellicle. In reaction it is alkaline, swelling up and becoming transparent in strong acetic acid. It is disintegrated and dissolved by caustic alkalies. It has all the characters of pure fibrine, with no albumen and no gelatine. Vegetable growths, as the *oidium of muguet* (VOGEL), occur in the pellicle of diphtheria from time to time, and have been reported by some as a constant occurrence. It is, however, by no means so; and the accidental existence of such vegetable growths is no evidence that epiphytic growths have any essential connection with cases of diphtheria (Sir WILLIAM JENNER).

The essential difference between the false membrane of "*the croup*" and that of "*diphtheria*," is that the former affects only the epithelial investment of the mucous surface, while the diphtheritic membrane is the product of the entire thickness of the mucous coat. Hence it cannot be separated without tearing blood-vessels; and hence also the origin of epistaxis and bloody expectoration in diphtheria, which never occur in "*the croup*" (TURNER and SANDERS). The pellicles of diphtheria are so closely incorporated with the substance of the mucous membrane, that it is quite denuded of its epithelium. The false membrane of diphtheria is a fibrillated texture of more or less dense and compact consistence, and yellowish-white or reddish-grey in colour. At the same time it is very friable, and breaks away in particles or in shreds resembling chamois leather. When forcibly detached, blood-vessels are torn and bleeding occurs. General tumefaction of the parts exists, and when ulceration takes

place it involves or exposes subjacent muscular tissue, while foci of supuration dip down to the cartilages. The membrane as a whole can never be thrown off by the patient (J. B. HOGG). On the other hand, the false membrane in "*the croup*" is a delicate film, which is semi-transparent, delicate, and tender to handle, somewhat gelatinous, and of a pale yellow colour, easily separable from the subjacent surface as a more or less perfect cast. After death and preservation in spirits it becomes opaque, and may be seen to be composed of more than one layer. During a violent fit of coughing this cast is generally thrown off, with immediate relief to the more urgent symptoms; and as there is no intimate connection with the mucous membrane there is no bleeding. It is simply a clean cast of the superficial epithelial layer.

"The histological differences are much more strongly marked. Diphtheritic membrane requires a good deal of teasing out to fit it for microscopic examination. A power of 350 diameters reveals an aggregation of granular matters, nucleated epithelium, fat-molecules, and minute crystals, held together by interspersed bands of connective tissue. Muco-purulent corpuscles often entangle foreign bodies; and in throat affections, the spores of the *oidium albicans* are rapidly developed. The membrane is, in short, a laminated fibroid mass of the superficial and deeper seated structures, in a later stage involving the submucous tissues, muscles, glands, and cartilages. In sections made from various preparations, portions of all these structures have been well seen, and sometimes I have found considerable hypertrophy of the connective and fibrous tissues. A portion of a croupous cast, however, examined under the same power, consists of numerous cylindrical and pavement epithelial cells, granular matter, fat-corpuscles, and mucus, with some occasional foreign bodies, entangled in a protoplasmic homogeneous matrix. The columnar epithelial cells retain their cilia, and are filled with clear sarcode, and nucleated. It is, therefore, surmised that these casts are not long retained; probably they are thrown off soon after their formation. Fungus-spores are rarely found entangled in these films, which are characterised by an excessive cell-proliferation rather than a transudation or true exudation. Although some of the croup casts differ a good deal in colour and consistency, connective or fibrous tissue never enters into their composition." (Mr. J. B. HOGG, *Lancet*, March 15, 1873.)

So also Professor Henoch, in a communication to the Berlin Medical Society, writes that the diphtheritic process has been anatomically distinguished from true croup ("*The Croup*") by the peculiarity that the mucous membrane becomes infiltrated with the secreted matters; while in croup there is a deposition of a free membrane on the surface of the mucous membrane.

Croup, by some, is regarded as a diphtheritic process under all circumstances; but while it is admitted that diphtheria may commence in the larynx, and be identical with what may be called a diphtheritic form of membranous croup, there can be no doubt of the existence of an independent disease—"The Croup," a primary disease which has nothing in common with diphtheria as an infectious disease beyond the local lesion being in a site common to both. It may be also conceded that most cases of membranous croup are diphtheritic; for "*the croup*" is a comparatively

rare disease, whereas diphtheria is a much more common disease than had been suspected.

The lymphatic glands, to which the lymphatics of the pharynx lead, are found in cases of diphtheria to be larger, redder, and moister than natural; and if the disease has continued long, they become brittle, pale, and of a brightish red colour on section—characteristic of inflammation of their substance. These enlarged glands may be felt during life behind the angle of the lower jaw on either side, as well as down the neck by the sides of the larynx, when that organ is implicated. Such enlargement of the glands is just in proportion to the severity and depth of the local, nasal, pharyngeal, laryngeal, and tracheal disease; and when the discharges from the pharynx are fetid, and the mucous membrane ulcerating, not only are the glands behind the angles of the jaw enlarged, but the connective tissue in which they are placed is the seat of the effusion of serum, and even the exudation of lymph, so that very great general swelling of the parts is the result. But suppuration rarely occurs in the cervical glands.

The Urine in Diphtheria.—Albumen is found in many cases; 50 per cent. (LEE), 66 per cent. (BOUCHUT, EMPIS), in the majority (MANGIN), and in all cases examined by Sanderson. Its quantity appears to be sometimes enormous, so that the urine becomes quite solid from heat and nitric acid (PARKES, l. c.) Although it is not established that albuminuria is an essential element in the disease, yet it is a most important symptom, both as connected with the pathology of the disease and with its prognosis. The first discovery of the relation of albuminuria to diphtheria was made by Dr. Wade of Birmingham, and was communicated to the Queen's College Medico-Chirurgical Society in December, 1857. During the following year Bouchut and Empis made a similar discovery in Paris. These observers attach very great importance to the renal complication, as affording an anatomical explanation of the fact that, in many cases of diphtheria in which death occurs neither by suffocation nor by septic poisoning, it cannot be due to local lesion. Bouchut considers it a sign of the commencement of purulent infection in diphtheria, and coincides with very great gravity of the disease. The blood then assumes the tinge of bistre; and numerous masses of pulmonary apoplexy may be found after death, resembling those which precede the development of metastatic abscesses in the lungs. In several cases related by Dr. Sanderson, the cessation of albuminuria was coincident with amelioration of the patient's condition and the disappearance of the most alarming symptoms. The early period of the disease at which the albumen appears, and the short time during which it lasts, are facts of great importance. He is of opinion that either—(1.) The kidneys must be the seat of the primary morbid process; or that, (2.) the albuminuria must depend on an original change in the blood. That it is not due to the former of these is evident from the fact that the renal disease is only coincident with disease elsewhere (*e.g.*, in the fauces), so that the special morbid condition of the blood induced by the *diphtheria miasm* must be regarded not only as the primary cause of albuminuria, but of all the other symptoms. Dr. Sanderson's observations still further show that, at the acmé of the disease, when the urine was intensely albuminous—when there was complete anorexia, and the ingesta reduced to a minimum—that then the

quantity of urea excreted in twenty-four hours was about twice as great as that excreted during a similar period when convalescence was established—when the patient was eating, with an appetite, the ordinary hospital diet, with extras. Thus diphtheria agrees with the other pyrexiae in being attended with a marked increase in the excretion of urea; and the condition of the kidney implied by albumen and fibrinous casts in the urine does not necessarily interfere with increase in the elimination of nitrogenous material. There is, therefore, no reason to apprehend the occurrence of uræmia as a consequence of the renal complication in diphtheria, this complication not being the cause of the blood-poisoning, but merely the index of its existence (*Brit. and For. Med.-Chir. Review*, Jan., 1860, p. 196). The kidneys are rarely found healthy under microscopic examination after death. There is a special change in the *intra-tubular* structure. With conspicuous congestion of the Malpighian tufts, there is great opacity of the tubules from engorgement of their interior with finely granular epithelial cells, in which oil globules are sometimes abundant, and occasionally blood.

Symptoms.—The *prodromata* which forebode an attack of diphtheria may be set down as general *malaise*, anorexia, slight fever, dysphagia, and glandular swelling in the neck. General constitutional symptoms supervene very gradually and insidiously, so that we rarely have an opportunity of observing the first elevation of temperature (which is rapid), or of following it with a thermometer, possibly up to 104° Fahr. The fever is, however, indicated by feelings of depression, prostration, and muscular debility, attended by headache, nausea, diarrhoea, chilliness, and bluish colour of skin; or by convulsions in children. There is a sense of stiffness about the neck, back of the head, and throat; and the drowsiness which attends the accession of an attack of diphtheria may lead the patient to fancy he has caught a slight cold in the throat while indulging in a short sleep. Cases of diphtheria may be arranged into,—(1.) *Malignant*, in which the general constitutional symptoms are very prominent; where the membrane forms rapidly, and is very heavy; where the submaxillary and cervical glands are very much swollen, and the breath becomes rapidly offensive. (2.) *Mild* cases, where constitutional disturbance is moderate; the membrane spreads slowly; the glandular enlargement is not great; and where the offensive breath can be controlled by antiseptics. (3.) *Diphtheritic sore throat*, where there is little constitutional disturbance beyond the first day or two; where the exudation is not extensive; with glandular tenderness rather than enlargement. Sir William Jenner has grouped cases of diphtheria into *six* varieties, as follow:—(1.) *The mild form of diphtheria*; (2.) *The inflammatory form*; (3.) *The insidious form*; (4.) *The nasal form*; (5.) *The primary laryngeal form*; (6.) *The asthenic form*.

In the *mild form of diphtheria* the general symptoms and the local lesions are trifling, and no sequelæ follow. Febrile disturbance prevails to a slight degree; and there may be the least possible soreness of the throat on swallowing. No albumen occurs in the urine, and no nervous symptoms follow. Many inflamed throats, when *diphtheria* is epidemic, have their origin, it is assumed, in the *diphtheria miasm* (whatever that may be), just as many cases of *diarrhoea*, when *cholera* is epidemic, originate in the *cholera miasm*; and it is as difficult to say in some cases that an inflamed

pharynx is not due to mild diphtheria, as it is to say that a serious diarrhoea is not cholera. In the *inflammatory form of diphtheria*, symptoms of severe *cynanche pharyngea* precede the exudation of lymph. There is redness, of a vivid or dusky hue, and swelling of the mucous membrane covering the arches of the palate, the uvula, and the tonsils. The swelling is often considerable, from the effusion of serum into the submucous tissue, which becomes of a jelly-like transparency and aspect. The pain in the act of swallowing is great, so that deglutition becomes impossible. The febrile disturbances may be extreme or moderate; and although the pulse is frequent, it soon becomes weak, and there is the sense of considerable prostration. In from twelve to forty-eight hours after the first symptoms of the throat affection supervene, a layer, more or less extensive, of tough lymph coats the inflamed surface, and death may follow from extension of the exudative process into the larynx or trachea. The urine may contain albumen, and sometimes the joints are swollen, hot, and tender. The *insidious forms of diphtheria* are dangerous, because they seem sudden and unexpected. The general symptoms are not severe. There is no marked soreness of the throat, no notable swelling of the lymphatic glands; but suddenly laryngeal symptoms supervene, and death rapidly follows from suffocation by stenosis of the larynx. This variety of the disease may be confounded with primary croup, if the pharynx has not been examined. The inflammation in such cases extends to the posterior part of the dorsum of the tongue, then to the epiglottis, and so to the interior of the larynx, and farther downwards into the trachea, and even into its branches. The invasion of the larynx is marked by alteration of voice—a peculiar hollow barking cough, and increasing want of breath, and intercurrent attacks of asphyxia as stenosis increases, any one of which may be fatal.

In the *nasal form of diphtheria* a sanious discharge from the nose attracts attention, after some febrile disturbance of a low type. The glands about the angles of the jaw begin to swell, the arches of the palate and tonsils become red and swollen, muco-purulent fluid bubbles in quantity from the narrowing isthmus of the fauces, and is apt to prevent the physician from seeing clearly the state of the pharyngeal mucous membrane. After a few days the disease may subside so completely as to leave its nature doubtful; but it may, on the other hand, spread to the larynx or the pharynx, when laryngeal or pharyngeal symptoms prevail, and the diagnosis is easy. This form is also apt to spread to the conjunctival mucous membrane. There is no sharp line of distinction, however, between these several varieties; and the ulcerated portions of the mucous membrane of the throat, with their membranous sloughs, are very apt to become the seat of putrefactive changes, for nowhere more than here is there such a combination of all the conditions for putrefaction—that is, the fetid decomposition of nitrogenous materials,—warmth, air, moisture, decaying animal tissue, muco-purulent secretion, possibly also some adhering remains of food.

In *primary laryngeal diphtheria* the disease begins with painful deglutition, and is attended by redness and swelling of the mucous membrane of the pharynx, arches of the palate, uvula, and soft palate. Laryngeal symptoms rapidly supervene; and lymph may be seen on

the arches of the palate, the exudation being more abundant at the base of the arch than above it, looking as if it spread from the larynx. Death threatens from *apnoea*. In the *asthenic form of diphtheria* the patient dies from the constitutional effects of the general disease, which may begin with general and local symptoms of very moderate severity. The pulse, however, soon becomes rapid and feeble; the sense of weakness and of illness becomes extreme; the skin has a feverish pungency of heat to the touch; the complexion assumes a dirty looking, pallid, and opaque aspect; and from an early period of the disease the tongue is brown, with *sordes* on the teeth. More or less lymph may be seen on the pharyngeal mucous membrane; and this lymph is of a granular, pulpy, or soft form. The patient may also swallow with perfect facility, and the throat symptoms may appear to be trivial in degree, even when the pharyngeal mucous membrane is covered with lymph. In some cases, however, the pain on swallowing is extreme. The exudative process may extend to the larynx, and this extension is indicated by a little huskiness and want of power in the voice, and imperfect laryngeal breathing. As *asthenia* progresses, so do the local exudations increase. Death tends to supervene by about the tenth or twelfth day of the disease, preceded or not by delirium, which may commence at an early period of the disease.

The temperature is highest in the malignant cases, probably owing to septicæmia; and is highest at the outset— 102° , 103° , and even 106.5° Fahr. After the second day it falls to nearly normal; and when it suddenly rises after its first fall, it is generally due to occlusion of the nares, with consequent septicæmia; or to rapid spread of the exudation.

Diagnosis.—*Croup* and *scarlet fever* are the two diseases most likely to be mistaken for *diphtheria*; and, as already noticed, there is a form of *diphtheria* commencing in the larynx (*laryngeal diphtheria*, so-called *membranous croup* or *diphtheritic croup*), which has been described as a form of croup, but which is *diphtheria*, and not *croup*—a disease quite *sui generis*; and which, beyond the site of the local lesion being the same, has little else in common with diphtheria. The anatomical characters of the lesion in diphtheria have been fully noticed (page 750, *ante*), and in connection with constitutional symptoms are generally conclusive as to the nature of the disease. The diagnosis between diphtheria and the several kinds of non-diphtheritic sore throat may sometimes be established by applying a small blister to a convenient spot, such as on the fore-arm. If the sore throat be a local manifestation of the constitutional affection—diphtheria—then and then only the blistered surface will become covered with false membrane, the diphtheritic membrane. There are also cases of confluent herpes of the throat, which it is difficult by mere examination to distinguish from cases of diphtheria. Diphtheria is attended with less febrile disturbance than scarlet fever; but records of temperature have not yielded results of practical value.

Dr. Bernard Kelly, the Medical Officer of the Fifth District of St. Olanes Union, Rotherhithe gives the following terse summary of the differences between diphtheria and croup:—

“While croup confines its action to the larynx, trachea, and bronchial tubes, which may be considered its proper territory, diphtheria, on the

contrary, extends its ravages not only to these parts, but also to the nares, fauces, tonsils, and, in severe cases, to the œsophagus and whole buccal cavity. The membranes exuded in both diseases are physically and histologically different. In croup, the membrane is uniform in consistence, whitish, brittle, inodorous, evenly moulded to the air-passages, and easily detached either by operation or by the expulsive efforts of vomiting; it is also non-vascular. In diphtheria, the membrane is uneven, elevated in parts, depressed in others, greyish or gangrenous in appearance, elastic, and strongly adherent to the subjacent tissues, from which it is with difficulty separated, and gives rise to a sanious, ichorous discharge, highly offensive in odour. It is also highly vascular, and bleeds freely to the slightest touch, or in the efforts of coughing or vomiting. These blood-vessels, no doubt, are the channels through which the putrid fluids, generated on its surface or interior, are carried into the circulation, and produce those characteristic symptoms of septicæmia so constantly seen in the last stage of the disease. The one (croup) assails the florid and robust; the other singles out the pale and feeble, or those just convalescing from exhausting diseases, such as measles and scarlatina. The one (croup) is sthenic and inflammatory, and destroys life mechanically by obstructing the air-passages and preventing the due oxygenation of the blood; the other is asthenic and constitutional, either in its inception or progress, and proves fatal not so much by impeding the respiration as by tainting the system in general. Hence the cyanosis and other symptoms of asphyxia which invariably precede death in the one case, and the putrid and typhoid phenomena which usher in a similar termination in the other. The one (croup) is as innocuous to the nurse, medical attendant, and others in its vicinity, as is a pleurisy or a pneumonia; the other is as contagious as small-pox or scarlatina. Croup, I may further add, is, *par excellence*, a disease of childhood; diphtheria may attack persons of any age."

Prognosis.—About 33 per cent. of the cases die, most of them being malignant cases from the first; and of them one-half or 50 per cent. die. The younger the patient, the greater the danger. The danger of death bears a marked ratio to the severity of the symptoms. The growth of the membrane from the pharynx into the nares increases the danger; but the most fatal symptom is its extension to the larynx. Most of the cases die on the fifth and sixth day, hence the chances of recovery become better after these days are safely passed. *Sequelæ* occur in about one-tenth of all the cases. The cervical glands do not tend to suppurate, but induration often remains for some time. Usually the glandular enlargement decreases rapidly as the diphtheritic membrane disappears; and there is not the same danger either from suppurating lymphatics, nor from acute nephritis, as attends scarlet fever.

The duration of cases of diphtheria varies from forty-eight hours to fourteen days; but local lesions may prolong the illness to weeks or months; and when the disease is fatal within a week, it is so by extension of the exudative process to the larynx. Hence in the greatest number of cases the manner of death is by asphyxia; septicæmia, eclampsia, and cardiac paralysis, and, lastly, asthenia causing the next greatest number of deaths. Thus the disease may prove fatal very early simply by reason of the vital importance of the parts affected. The disease may be presumed to exist so long as there is elevation of temperature, with excess in the

excretion of urea, and of urates and albumen in the urine. Laryngeal symptoms rarely commence after the expiration of the first week of the disease, although they have been known to occur as late as the fourteenth day; and in more than half the fatal cases of diphtheria death results directly from disease of the larynx. When death occurs from asthenia the fatal result usually takes place about the second week of the disease. In the cases that are not fatal the specific disease terminates between the eighth and fourteenth day of the illness (JENNER).

There is great liability of other mucous membranes and of the skin when denuded of epithelium, as the surface of ulcers and wounds, to become covered with the specific diphtheritic exudation. Hence the duration of the disease may be greatly extended by the occurrence of such events, as when the lips, eyelids, ears, cheeks, vulva, and the like, may be affected. Like all other specific diseases of its class, the earlier period of an epidemic outbreak is generally attended with the largest proportion of fatal cases. In this disease, as in many others of the *miasmatic* kind, the *general* or the *local* symptoms may predominate, giving special features to each case; and the patient may die from the severity of the general disease—the specific fever—or from the severity of some one of the local lesions. It is one of those diseases in which there is a tendency to collapse in consequence of the poison. Hence sudden death is not uncommon, perhaps from some affection of the *vagus*, as shown in the loss of appetite and increase of pulse-rate which precede death (SENATOR).

However mild a case may appear to be, it is not unattended with danger. The great danger during the first week, and towards its end, is from extension of the exudative process to the larynx; and the least laryngeal quality in the respiration heard at the bedside is suggestive of danger. Much exudation in the nares and nasal passages is also unfavourable. Subsequently to the first week death is to be apprehended from exhaustion and loss of nervous energy. An extremely rapid and feeble pulse is of grave import; and a very infrequent pulse is of fatal significance. Vomiting and purging are also unfavourable symptoms, especially if they should recur many days in succession. Hæmorrhages and albumen in the urine indicate blood change of great severity; but the mere abundance of the albumen is not serious so long as the urine is normal in amount, without blood corpuscles or casts of tubes, and its specific gravity continues to be high (SQUIRE). All the cases in which Sir William Jenner has known delirium to occur have ended fatally. A sudden rise in the temperature of the body is also unfavourable—103° or 104° Fahr.—indicating a probable complication with some intercurrent lesion. To recognise the disease early, and its specific nature, and to manage it judiciously, are important elements towards a favourable prognosis. The danger in diphtheria seems to be in proportion to the youth of the patient. In the child, death is generally due to extension of the disease to the larynx; after puberty it more often occurs from the general affection.

Sequelæ.—Long after the termination of the disease, symptoms of a very peculiar and characteristic kind are apt to supervene. The phenomena are referable to deranged innervation; and although their frequency and intensity are by no means invariably proportional to the severity of the primary disease, yet the more severe the case is, the more likely is nervous

disorder to occur, and the more intense is it likely to prove. These consecutive phenomena do not appear at once. There is usually a period of convalescence between the disappearance of the primary and the appearance of the secondary phenomena of diphtheria. This period of temporary convalescence varies from a few days to a few weeks. By far the most common sequel is paralysis—(1.) of the pharynx, (2.) of the larynx, (3.) of the limbs, (4.) of the heart. The most alarming symptoms are referable to the heart. The frequency of its beats per minute begins to diminish, and a sense of languor supervenes, with tendency to vomiting. The heart's beats are found to be feeble and slow, and death may supervene from cessation of the heart's action (JENNER); or suddenly, from the deposition of fibrine within the heart, or in one of the great vessels (TANNER). In other cases the paralysis is more widely spread, and the nervous symptoms are more striking; although the nerve affections do not at once attain their maximum of intensity, but are progressive; and the progress of the paralysis, even in the same set of muscles, is seldom quite uniform. It is believed that the paralysis is due to a primary peripheral alteration of the nerves, which is propagated from the originally affected part to the spinal centre, much in the same way as in tetanus the irritation is transmitted from the wound (WEBER). The nerves of the soft palate and pharynx come for the most part from the pharyngeal plexus, in the formation of which the glosso-pharyngeal, the pneumogastric, and branches from the superior cervical ganglion of the sympathetic take part. Along these nerves in their connective tissue sheaths, and in the loose connective tissue which surrounds them, the morbid process gradually creeps on, along the terminal branch, upwards to the carotid plexus, and hence to the *ciliary ganglion*, to the *abducens*, *motor oculi*, and *trochlear nerves*, downwards to the *cardiac plexus* and the various branches of the *vagus*, to the *spinal accessory nerves*. In this way the morbid process advances to the *spinal ganglia* and the *spinal cord*. If several sets of muscles are attacked, the faucial or pharyngeal are usually the first set to suffer; and the impairment of function is very early betrayed by the condition of the voice, and especially by the difficulty of swallowing liquids, with loss of sensibility of the *velum pendulum palati*—i.e., with paralysis of the *velum* and return of drinks by the nose (TROUSSEAU). The sight is subsequently apt to become impaired; then the muscles of the tongue, the lips, and those of the upper and lower extremity, become affected in the order named. In Dr. Greenhow's experience anæsthesia has co-existed with the paralytic affection of the fauces in all the cases. The mucous membrane over that region, naturally so sensitive, becomes altogether insensible and callous, even to repeated and forcible pricks with the nib of a pen; and in rare cases speech becomes so inarticulate as to be almost unintelligible. It has also been observed that the paralysis and anæsthesia are sometimes more complete on that side of the fauces which was most severely affected in the early stage of the disease. When the pharyngeal muscles are paralysed, and anæsthesia prevails, there is alarming difficulty in swallowing. The impairment of vision is probably due to paralysis of the ciliary muscle. There is defective accommodation, and inability to perceive near objects distinctly. It generally comes on suddenly, and is preceded for a day or two by dilatation and sluggish action, or actual paralysis of the iris. The

tongue, lips, and cheeks may also become affected, both as regards motion and sensation. The parts then become numb or cold, or they experience a sense of formication or of scalding, and taste may be lost. The upper extremities are either first affected, or simultaneously with the lower. The power over the movements of the limbs becomes impaired, and anæsthesia is also more or less complete; or there may be tenderness with abnormal sensations, such as formication, and a perception of constriction or of tightness in the fleshy parts; or there may be convulsive movements of the limbs, resembling chorea. The earliest indications of these phenomena connected with the functions of the limbs are peripheral. Tingling is experienced at the tips of the fingers, accompanied with numbness, rendering the patient unable to pick up small objects. These phenomena then extend gradually to the wrist, and upwards to the elbows, and even to the shoulders; but the tingling and numbness are especially felt on the palmar surface of the hands. The limbs when so affected feel heavy, feeble, and cold. If the paralysis continues, the muscles concerned become flabby and emaciated, and strength becomes so much impaired that patients who can move the affected limbs freely in bed, often walk with much difficulty, or are unable to stand unless supported. Increased sensibility of particular parts of the affected limbs accompanies the loss of power and general numbness, causing great distress, such as tenderness of the soles of the feet, the calves of the legs, or fleshy parts of the arms. There is also pain on pressure along the course of the principal nerves, such as the median nerve of the arm and the sciatic nerve of the leg (*Edin. Med. Journal*, August, 1863). The duration of the paralysis varies greatly, but generally the cases eventually terminate favourably. The paralysis seems to be only dangerous when it affects the respiratory muscles and the heart, for there are now numerous cases in which the pulmonary and cardiac branches of the pneumogastric are implicated in the paralysis. In addition to the usual nervine symptoms observed after diphtheria in such cases, there is also great dyspnoea, with slow respiration and increased pulse-rate—phenomena seen in animals after section of the nerve (SENATOR, P. GUTTMANN). Another peculiarity which distinguishes the paralyzes of diphtheria from paralyzes of a peripheral kind is that their advance is not continuous, but by fits and starts; and do not usually attack one entire nerve trunk, but only one or more branches of it, and that only for a short time. These phenomena of impaired nervous power generally betray themselves within three weeks from the date of apparent convalescence.

Treatment.—There are especially three things it is desirable to bring about, namely:—(1.) Softening or suppuration of the exudation and its connections—to convert the process into a catarrhal one; (2.) to thoroughly disinfect the air passages; (3.) to overcome the marked asthenia by which all cases of this disease are marked (WAGENEN). To accomplish the first, vapour of water at a temperature of 120° to 130° Fahr. should be inhaled continuously for fifteen or twenty minutes every hour; and where the exudation is considerable and tough a very hot poultice should be applied externally, or compresses wrung out of hot water and covered with oil-silk should be used. Under such means the exudation may break down into a suppurating mass in from twelve to

twenty-four hours; but the deposit may return again, if the vapour inhalation is omitted too early. Even in those cases which die, this method of treatment accomplishes euthanasia. The temperature of the room in which the patient is confined to bed ought to be kept at 68° Fahr., and its atmosphere kept moist by the steam from a kettle with a long spout constantly boiling on the fire, and by large basins placed about the room, and kept constantly full of hot water. If the patient can be enveloped in a warm moist atmosphere, so much the better; and this may be done by making a tent with blankets over the bed, and, by the aid of a spirit lamp, a tin kettle of boiling water may be maintained at the boiling point, and its steam thus made to envelop the patient. The steam assists very much in dissolving the mucus and in loosening and breaking up the membranous deposit. To accomplish the second indication—namely, disinfection—*acetic*, *salicylic*, or *carbolic acid*, or *sulphurous acid* may be used. Neither is a specific. A wineglass of vinegar to a pint of water is a good proportion (Sir W. JENNER). The solutions ought to be made as strong as the patient will bear them; and should be used with the vapour of water as a spray in a steam-atomizer, as well as by gargling. The steam antiseptic spray is the best, as the steam carries the disinfectant to every part of the diseased surface; while the vapour which escapes into the room is also a carrier of the antiseptic, as it is of caloric, when a large room is heated by dripping water on a hot plate or stone.

Syringing the throat and *nares* with the *perchloride of iron* in solution is also very beneficial. The solution ought to consist of *tincture of perchloride of iron*, and of *glycerine*, each *half a drachm*, with two or three drachms of water. A stronger solution—the strength of the tincture—may be applied with a large camel's-hair brush to a patch of exudation, and the adjacent surface of the mucous membrane; or a preparation, twice the strength of the tincture, may be made by mixing equal parts of the *liquor ferri perchloride* and of *glycerine*, and may be used to brush over the patches if the exudation is very thick. But such an application, as it is powerfully styptic, should be confined to the surface of the exudation only. It readily penetrates it, and exerts its beneficial influence on the vessels below (SQUIRE). In short, the application of this remedy to the whole of the pharynx with food, or separately as a lotion, to be applied as a gargle, or a medicine to be swallowed, affords relief.

With regard to topical applications, Sir Wm. Jenner is of opinion that repeated applications to the throat of *caustic* solutions are injurious. He recommends one single but efficient application of a strong solution of nitrate of silver (℥i. to ℥i. of water), as a remedy which may stay the spread of the exudative inflammation; but that, on the whole, *hydrochloric acid* and water in equal parts will more frequently attain the object. It is especially the surface *round* the exudation, as well as the exudation itself, that should be painted well over with the solution, the brush being passed over the surface two or three times in quick succession. The white discoloration which results must not be confounded with the spread of the diphtheritic exudation. The discoloration from the acid passes away in about thirty-six hours, and that from the nitrate of silver somewhat quicker. *Medicinal carbolic acid* (as prepared by

Calvert, of Manchester) is valuable as a gargle, in the proportion of 1 of acid to 200 parts of water.

There are considerable differences of opinion regarding the usefulness of topical applications, and the best means of applying them. *The tincture of the perchloride of iron* is recommended by some to be gently painted over the fauces, as already directed. The throat should be washed out as often as possible with *permanganate of potash* (1 to 300), or with *lime water* in a solution of *chlorate of potash*. The local application of *lime water* by frequent gargling or gentle brushing, with the internal administration of *nitrate* and *carbonate of soda*, prove speedily curative in the milder cases, and alleviate distress in the more severe. Ice kept dissolving in the mouth is often also a great comfort, and its use should never be omitted where it can be had. Dr. Greenhow remonstrates against the application of the more severe topical remedies. The pellicle or false membrane ought never to be torn off. The bowels should be opened freely by a dose of *calomel* and *jalap*; or by *calomel* and *colocynth* pill, followed in the inflammatory or *sthenic* forms of the disease by a saline aperient—e. g., *sulphate of magnesia* in the *infusion of roses*. If feebleness of pulse supervene, if the redness of the throat assume a dusky hue, if the sense of general weakness become extreme, wine in large doses frequently repeated is required. Six or eight ounces of port or sherry during the day for an adult may be given from the first, with as good a diet as the stomach can digest. During the course of the disease, much larger quantities of wine and even brandy may be necessary; but the quantity of stimulants must be regulated by the habits and age of the patients. A child of three years of age may take with advantage one or two drachms of brandy every hour—i.e., from three to five ounces of brandy during the twenty-four hours (Sir WM. JENNER). So long as there is firmness of pulse the physician ought to abstain from alcoholic stimulants, and rest contented by giving such saline medicines as exert a slight action on the skin and on the kidneys, or on both. But a rapid pulse indicates the necessity of alcoholic stimulants, which ought to be freely given on the development of the earliest general symptoms. Under all circumstances efficient daily action of the bowels must be secured, and the urinary and intestinal secretions should be examined daily. If blood or albumen appear in the urine, diuretics are contraindicated. Mustard poultices, warm linseed-meal poultices, or the warm wet sheet, as recommended by Dr. Huss in typhoid fever, may be applied to the loins under these circumstances. The hot-air bath applied to the body, without removal from the bed, is also of great service. Diphtheria is most successfully treated by the administration of mineral acids, especially the *nitric* and *hydrochloric*; also by *guaiacum*, *chlorate of potash*, *quinine*, and *iron*. *Tincture of the perchloride of iron* is now fully recognised as having a beneficial local as well as general influence on the disease; and it may be advantageously combined with quinine in the following formula (TANNER):—

R. Quinæ Sulphatis, gr. ii.; Acidi Hydrochlorici diluti, ℥x.; Tincturæ Ferri Perchloridi, ℥xv.; Infusi Calumbæ, ℥i.; misce. Fiat haustus, omnibus sextis horis sumendus.

As soon as nourishment can be retained by the stomach *five to ten* grains of the *perchloride of iron*, equivalent to *twenty or forty minims* of the *tincture*, combined with *half a drachm* of *glycerine* and *half an ounce* of *water*, should be given, and repeated every three or four hours (SQUIRE). It should be commenced on the first day of the illness, or as soon as the nature of the disease is recognised. Evidence of its good effects are shown by a diminution of the secretions from the fauces and throat, and improvement in general symptoms. *Guaiacum* is of service in keeping the bowels open. It ought to be given in the form of the *rectified spirit tincture*, to the extent of one drachm combined with two drachms of *glycerine* for a dose, as recommended by Dr. Balmanno Squire. This makes a clear solution, to which no water must be added.

The third indication—namely, to overcome the marked asthenia—restoratives are demanded, and feeding is all-important in the treatment of diphtheria. The night should never be passed without either nourishment or stimulant being given; and the quantity of liquid nourishment and of stimulant given in the twenty-four hours must be equal to the estimated requirement of the patient (SQUIRE). A young child (one to two years) may require a teaspoonful of brandy every two hours; a child of three years, two teaspoonfuls, diluted, and given in small portions at a time. Milk, beef-tea thickened with arrow-root, milk punch, are most useful alteratives. By so averting death, time is gained for the general disease to run its course (JENNER).

Tracheotomy is of little avail in diphtheria, and the sole object contemplated by an opening in the windpipe is the prevention of death by suffocation. Nevertheless the operation is indicated in every case where the larynx is blocked up by membranous or pseudo-membranous formation; when we can scarcely ever anticipate that the disease will subside in a few days, and before fatal suffocation occurs. The condition of the patient is generally improved for the time by the actual operation, if it is not too long deferred, or done in the death-agony. It is also especially done to gain time; and it is also indicated whether the impediment to respiration is in the larynx only, or whether it extends farther down, or whether secondary changes have already taken place in the lungs. In the two latter cases the chances of recovery are generally very much less; but the operation of tracheotomy does not make the prospects worse for the patient; and it should be done when the existence of a more than merely catarrhal affection of the larynx is indicated by great difficulty of breathing, continuing more than two or three days; or by attacks of suffocation. The operation will not *cure* the disease. In this respect it differs greatly from *croup*; and it is chiefly in cases of *croup*, as distinguished from *diphtheria*, that tracheotomy is attended with such favourable results. (See under *Croup*, Vol. II.) Tracheotomy, if not too long delayed, saves many a life in *croup*.

In the consecutive paralyses tonics and local galvanism are the most important remedies, and the bowels should be kept open by a pill, taken morning and evening, containing from *a quarter to half a grain* or *a grain* of the *extract of nux vomica*, with a like quantity of *sulphate of iron*, combined with *two or three grains* of *compound rhubarb pill mass*. These may be varied with the administration of pills containing $\frac{1}{12}$ of a grain of *strychnia*, the

strychnia being triturated with *sugar of milk*, and made into pills with a sufficient quantity of extract of *gentian*. Syrup of the *phosphate of iron* in fluid drachm doses may be given twice a day (combined or not with liquor *strychnia*); and stimulants in the form of malt liquors, especially stout (if free of *cocculus indicus*), are beneficial if taken with or after meals, and the doses of iron may be taken at the same time. The syrup of the *phosphate of iron*, *quinine*, and *strychnia* I have also found of great use.

HOOPING-COUGH.

LATIN EQ., *Pertussis*; FRENCH EQ., *Coqueluche*; GERMAN EQ., *Keuchhusten*—Syn., *Stickhusten*; ITALIAN EQ., *Tosse Convulsa*—Syn., *T. Canina*.

Definition.—*An infectious and (sometimes epidemic) specific disease, preceded and accompanied by fever of variable intensity; attended in the first instance by catarrh, and subsequently by paroxysmal fits of convulsive coughing, which occur in numerous, short, rapid, spasmodic movements of forcible expiration, suddenly followed by a prolonged and deep inspiration, marked by a characteristic sound of a loud, sonorous kind, and variously named the "kink," "hoop," or "whoop." These paroxysms of expiratory and respiratory convulsive movements alternately recur several times, till the fit ends by a quantity of mucus being brought up from the lungs, or till the contents of the stomach are evacuated.*

Pathology.—(a.) *Causation and Modes of Propagation.*—That whooping-cough is induced by a specific poison there is little doubt; but in what manner this morbid agent is generated is not determined. But when the disease is once excited, the patient evolves a poison which is highly contagious, communicable from one human being to another—a fact which necessarily implies the idea of "specificity." The general public are so unanimously of this opinion, that no parent will permit his yet unaffected child to mingle with such as may be labouring under it. The profession are, with a few exceptions, of a similar opinion. It is probably most contagious at the period of its highest development. The infecting distance of the poison must be considerable, from the utter impossibility of isolating the patient at home, or of preventing the spread of the disease in schools. It is often propagated from patient to patient, from house to house, and from village to village, mainly through the atmosphere, but also by fomites. The predisposition to the disease is so strong that few persons pass the period of childhood without suffering from it; but it may occur at any subsequent age. The early age at which the large majority of patients pass through the disease is, however, a sufficient reason for our very slight acquaintance with the predisposing causes. It is said to be more common in spring and autumn; but it may be that it attracts more attention then from its specially high mortality at these seasons. This disease may co-exist with many others, and then often greatly influencing each other's course and duration. *Small-pox* and *whooping-cough* have often co-existed; and a very common and fatal combination is *measles* and *whooping-cough*. *Whooping-cough* and *cow-pox* are not unfrequently combined. Indeed, the lower classes erroneously look upon vaccination as in many instances a

cure for *hooping-cough*. The disease is a "specific pulmonary catarrh;" and very different opinions have at various times been entertained as to its nature. Its origin is not beyond 1510, when it was endemic in Paris; but its epidemic character was not determined till 1580 (SPRENGEL). It appears, as a rule, but once during life, and attacks chiefly infants and children. Dr. Watson gives an instance of a child born with hooping-cough. Dr. Johnson one at three months. There are instances, however, of its occurring not only late in life, but also a second time (HEBERDEN). Blache gives a remarkable instance of a grandfather and grandmother catching hooping-cough a second time from their grandchild, all of them labouring under the disease together. Some consider the disease to be a specific affection of the nervous system; others, that it is a specific catarrh; but both these pathological conditions co-exist in hooping-cough.

(b.) *Morbid Anatomy*.—Inflammation does not necessarily accompany the disease, although a state of the mucous membrane exists by which it is morbidly irritable, or highly susceptible to impressions. The results of nineteen *post-mortem* observations made by Dr. Graily Hewitt during a recent epidemic of this disease (1855), in children varying from one month old to four years, showed the chief lesion to be collapse of the lung-substance—a condition also known under the various names of *fœtal condition*, *carnification*, and *atelectasis*. The air-cells most distant from the roots of the lungs were most liable to this change, so that the margins of the lungs were chiefly affected; and there was generally emphysematous distension of the air-vesicles adjoining the collapsed portions of these organs. Congestion of the mucous membrane of the air-passages, lobular pneumonia, interlobular emphysema in children, and bronchial glands enlarged. But there are more traceable lesions due to complications of the disease. The pneumogastric nerve has sometimes been found red, with the medullary matter altered in colour, dense, and abnormally firm (KILIAN, AUTENRIETH). Other pathologists have ascribed the disease to cerebral irritation (WEBSTER, in *Medical Gazette*). But facts tend to show that the cerebral symptoms are effects, rather than the cause of the disease. In short, the formation and development of *hooping-cough* seem to follow as the result of a specific poison of an unknown kind, but which is communicated through the atmosphere, and seems more or less to affect directly the whole system, but having a special tendency to involve the pulmonary mucous membrane, producing in it a slight but specific inflammatory change, to the effect of which on the peripheric ends of the pneumogastric nerves the cough with its peculiar characteristics is due. This view is confirmed by the fact that it is from the implicated mucous surface that the contagium of the disease is chiefly, if not exclusively, emitted (BRISTOWE), like *influenza* and *measles*; and, like them, the disease sometimes becomes epidemic.

Symptoms.—In the majority of cases, hooping-cough commences like a simple catarrh, alike in children and adults; afterwards, however, the specific nervous element of the disease predominates, and is combined with the catarrhal element. The period of latency has not been definitely fixed; but, according to Bristowe, it is probably about a fortnight; according to Squire, usually about a week. The catarrhal symptoms may last for several days—three to fourteen—or even for several weeks—one to four

(WILLAN, TROUSSEAU); six to eight (LOMBARD)—before the specific convulsive cough supervenes; and in some instances, where all the other symptoms are present, the convulsive cough may be absent. In the first stage of the disease it is the incessant repetition of the cough which is most characteristic; in the succeeding stage, when the cough becomes convulsive, the incessant repetition subsides, the cough recurs less frequently, and is more convulsive. A sensation of tickling or prickling in larynx and trachea is the indication of a convulsive cough coming on. This is the warning which young children recognise and dread as the harbinger of a paroxysm, which suggests to them the necessity of seizing something for support during the fit of coughing, which almost immediately commences. The irritation is attempted to be got rid of by coughing; and in the expiratory efforts the air is expelled with great violence, and so repeatedly and irresistibly that the lungs are ultimately almost emptied of air. At the conclusion of these expiratory efforts the condition of the lungs resembles that produced by *asphyxia*. A sudden inspiration now necessarily and suddenly follows, the air being drawn through the glottis by the gasping patient, with a force and velocity which gives rise to a shrill, sonorous sound not unlike the crowing of a cock, which has been variously named a *kink*, a *hoop*, or *whoop*; and the disease has accordingly received various names; such as, *kink host*, *hooping-cough*, *whooping-cough*, *chincough*, *kink-cough*. The anxious and distressing inspirations are scarcely completed when the convulsive expirations of the cough are again renewed, and again followed by the gasping and crowing inspirations, till a quantity of peculiar viscid, transparent mucus is brought up from the lungs, or till the contents of the stomach are rejected by vomiting. The matters expectorated from the lungs are frequently thick, ropy, and viscid; or a colourless liquid. When vomited from the stomach, the patient throws up a glairy fluid of much tenacity, semi-transparent, and frequently amounting to the greater part of a pint; and should he have recently eaten, the food returns with it. It frequently happens, however, that the stomach retains the food and rejects the offending matter. If the fit be violent, the fluid rushes not only from the mouth, but also from the nostrils; and in some instances is mixed with blood, for blood occasionally bursts forth in considerable quantities from the congested vessels of the mouth, the nostrils, the ears, the eyes, and in some instances from the rectum. Such cases are most severe. Such are the phenomena of the fit of paroxysm of whooping-cough. After it is over, the patient in ordinary cases appears to be but little affected, and returns immediately to play, or to any other occupation which takes the attention at the time. Hooping-cough varies greatly in intensity, and most authors divide the group of symptoms into three stages. The first stage comprehends the period from the first symptoms of illness until the "*whoop*" confirms the convulsive nature of the cough. This is the period of development or evolution. The second stage commences as soon as the nature of the cough is determined, and lasts till the violence of the cough and the danger of secondary complications are passed. This is the period of spasmodic paroxysms characteristic of the disease. The third stage comprehends the convalescence of the patient, until the final and happy termination of the disease; or the occurrence of any event which may destroy the sufferer.

If the stethoscope be applied to the chest previous to the fit, the mucous rhonchus common to catarrh may exist, yet in most cases the respiration is natural. During the act of coughing the respiration appears completely suspended, and is not sensible to the ear in any part of the chest. On the "*whoop*," however, taking place, the air is heard to rush with remarkable violence into the trachea; but at this point it stops for one or more seconds till the bronchial tubes relax, and the air is then admitted into the lungs.

The fit having subsided, the eyes, which seemed to have started from their orbits, and inundated with tears, resume their natural position. The conjunctiva is more or less gorged with blood, but the natural expression and appearance of the countenance returns, and in a few minutes, in favourable cases, the good spirits of the little patient are renewed, and he eats with appetite. On the contrary, in severe or unfavourable cases, long-continued exhaustion, headache, and some fever, are the preludes to convulsions, inflammation, or the more severe complications of this disease. The paroxysm varies greatly in frequency and severity, but, in general, its frequency is as its severity. In ordinary cases it returns every two hours; but in severe cases, and especially during the second and third week, it returns every half or every quarter of an hour, or oftener. The disease commonly reaches its acmé at the end of the third, fourth, or fifth week; after which the paroxysms diminish in frequency, the intervals are prolonged, and the patient is to a certain degree convalescent. The duration of this second stage is from two to six or eight weeks. The third stage commences with the convalescence of the patient, when the paroxysms become milder, the intervals longer, the expectoration thicker and more opaque, greenish, or pus-like, and more like ordinary catarrh. The vomiting ceases, and the general health of the patient begins to improve. The duration of this stage, however, is often long and variable, and the cough may still harass the patient for many weeks, or even many months. It is to this stage that the term chronic is usually applied. Thus, the disease lasts from six, eight, to ten weeks before convalescence commences; but there are exceptional cases, which, on the one hand, get well in a week, and, on the other hand, may continue several months, or even a year. "The general duration of the disease is directly proportionate to the duration of the prodromata;" and the more quickly the convulsive cough makes its appearance, the more quickly does the disease subside (Trousseau). Hooping-cough may also pass through all its stages without the characteristic whoop; this is generally the case when it affects adults.

Many accidents may arise to complicate the symptoms of hooping-cough, and to increase the danger. Inflammation of the minute bronchia is the most usual complication—*capillary bronchitis*, or *peripneumonic catarrh*; but the secretions are in defect, so that the mucus is not only greatly diminished in quantity, but is thick and viscid, teasing the patient with fruitless efforts to free it from the air-tubes, thus causing a frequent recurrence of the paroxysm. In other cases it may assume the form of purulent inflammation, the pus secreted being formed into sputa, and moderate in quantity; or it may be thrown up pure, as from an abscess, and so enormous in quantity as to amount to one or two pints in the

twenty-four hours. The inflammation of the bronchial membrane may spread to the substance of the lungs, when the danger, as well as the symptoms, of some of the various forms of pneumonia will be added to the disease. Difficulty of breathing, lividity of face, crepitation over the chest, sibilant bronchus, increase of temperature and of pulse, with impairment of strength, are the phenomena which denote such complications. The mucous membrane of the stomach and intestines is often the seat of inflammation; and this is denoted by pain in the epigastrium, and by the suppression of the glairy fluid thrown up by vomiting, so that on the termination of the fit the patient often lies in a state of complete exhaustion, unable to discharge anything either from the stomach or lungs, or even to "*whoop*," and he is then said, in popular language, to labour under the "*dumb kink*." In mild cases the bowels are little affected in this disease, except that the patient sometimes passes fæces during the paroxysm. In severe forms the stools are often either black and offensive, or they consist of a colourless mucus, the latter evidently depending on an inflamed state of the mucous follicles. Headache is a symptom which usually attends the catarrhal stage, but generally ceases when the fever subsides. In some instances it continues throughout the disease, and is not unfrequently the forerunner of fatal convulsions, or epilepsy, or of inflammation of the membranes of the brain, terminating in delirium, coma, hydrocephalus, and death.

Diagnosis.—The fever of invasion is characteristic of whooping-cough. It is of greater intensity and of longer duration than the fever of an ordinary catarrh. It may last from seven to fourteen days; while the fever of a simple catarrh is rarely prolonged beyond two or three days. But generally the earliest recognition of whooping-cough is by the *obstinate coughing*. And the secretion of a viscid mucus from the bronchial mucous membrane, with repeated paroxysms of coughing—several times in a minute—and continuing for many days, or from one to two weeks, are sufficient to indicate a specific catarrh, as distinguished from an ordinary cold. In some cases, again, the nervous element of the disease is mostly developed; so that from the very commencement a kind of *hiccough* exists, or "spasms in the throat," from the efforts to inspire, causing a whistling through the larynx. The paroxysms of coughing are thus more frequent, obstinate, and severe in whooping-cough than is usually the case in a common cold; and the patient has a more troublesome sensation of tickling in the throat and inside the trachea—the commencement of the nervous element of the disease, which eventually becomes so characteristic. A cold giving rise to paroxysms of coughing, recurring fifteen, twenty, or thirty times in a minute, and which continues in this manner for four to ten days in succession, attended with high fever, is certainly a specific catarrh; and after a period varying from one to two weeks later, whooping-cough will manifest itself with its well-marked characteristics (TROUSSEAU). As soon, however, as the cough has been followed for two or three paroxysms by the "*whoop*," the diagnosis is perfect, no other disease being accompanied by this symptom.

Prognosis.—The proportionate number of deaths to recoveries in *whooping-cough* is not determined, but greatly varies in different years. In one year hardly a death will occur from the disease in a large city, while

in another year many children will die. In general, however, the milder forms of the disease are rarely fatal, while the more severe and protracted cases very commonly are so, according to the age and surroundings of the patient. Of ten fatal cases nine belong to the poorer classes (LOMBARD). The reports of the Registrar-General show that the mortality is greater from this disease in towns than in the country. The age of greatest mortality is between the first and second years of life (LOMBARD). Under one year of age, it is the most fatal of all the diseases of children; sixty-eight per cent. of all the deaths from it occur under two years of age, and only six per cent. above the age of five years (Dr. E. SMITH).

Danger from bronchial inflammation is to be dreaded rather towards the end than the beginning of the disease. Convulsions are apt to occur if dentition is going on at the time; and if they arise from congestion or effusion within the cranium, the case is generally fatal. The number of paroxysms which a child may have in twenty-four hours is the best basis of prognosis. Twenty fits in twenty-four hours denote a very mild case; when more violent, forty to fifty paroxysms may occur in that period; and when the number of paroxysms exceeds forty the case is a serious one, and prognosis grave. *Hooping-cough* may determine the development of *phthisis*.

Treatment.—On the invasion of the disease, beyond putting the patient on a low or very moderate diet, and attending to the daily action of the bowels, there is little occasion for medicine. It will run its course; and although no drug has any direct influence over this disease, one of two indications of treatment may be followed. The first is to prevent, if possible, cerebral irritation or convulsions, or any attack of inflammation, either of the lungs, the stomach, or of the membranes of the brain. The second indication, after the period of danger is past, is to prescribe such medicines as may diminish, mitigate, and control, as far as possible, the frequency of the paroxysms, to check those secretions which are in excess, and to excite those which are in defect. These objects are best obtained by mild sedatives, combined with gentle purgatives or laxatives. The choice of the sedative has been considered a matter of much importance. The Continental physicians have bestowed much praise on *belladonna*, others on *hemlock*, others on *henbane*, while some have contented themselves with *opium*. It must be admitted, however, that none of these narcotics possess any specific property in controlling this disease, so that the selection of the particular one must be left to the discretion of the practitioner. The mildest sedatives, such as *hyoscyamus*, or the *syrup of poppies*, are the safest and best. Should, however, *belladonna* be selected (as it seems to be the most efficient sedative), if the child be under four years of age, the dose ought not to exceed *one-eighth* or *one-tenth* of a grain of the extract, with the same quantity of the powder of the leaves in a pill, the pill being dissolved in syrup at the time when it is to be administered (TROUSSEAU). The dose should be given in the morning; and only once daily, commencing with one pill, and increasing to two or more. Or if *hyoscyamus* is chosen, *half a grain* to a grain every six or eight hours; while, if it be the *syrup of poppies*, this medicine should be given in such fractional doses of a drachm

as are suited to the age. *Powdered belladonna root* has been recently recommended by Vollaut. The dose is *one-fifth* of a grain, given at first *once*, then *twice*, then *four times a day*, and so on until the paroxysms begin to subside, when it is to be given at much longer intervals. Thus, he says, the spasmodic period may be positively arrested in three or four days. (*Syden. Society Year-Book*, 1862). *Cochineal* is an anodyne which sometimes affords relief. I have found it of marked benefit in cases where the child has been losing flesh from the constant vomiting following the paroxysms of cough. The tendency to vomit has always disappeared under its persevering use. It is usually prescribed in the form of a mixture, consisting of *cochineal*, 10 grains; *subcarbonate of potash*, 20 grains; *sugar*, $\frac{1}{4}$ oz.; *water*, 4 oz.; *rub together and strain* (*SQUIRE'S Companion to the Pharmacopœia*, p. 62). Of this mixture, a quarter of a teaspoonful four times a day is sufficient for a child one year old; half a teaspoonful for a child of two years; and a teaspoonful for a child of four years. Boiled apples in milk should be given for food. An opiate, in the early stage of the disease, ought not to be administered alone, as it is apt to check the mucous secretion. Some purgative or laxative ought, as a general rule in all cases, to be combined with it; such as the *confectio sennæ*, *rhubarb*, *castor-oil*, or *manna*. The neutral salts, however, sit easiest on the stomach, and (as the medicine must be continued) they are the most agreeable to the patient. If at the outset or afterwards the cough is very suffocative, an emetic is useful. Five to nine grains of *sulphate of copper* dissolved in three ounces of *distilled water*, and a dessert-spoonful every ten minutes, is the most efficient (TROUSSEAU). *Nitric acid*, in the following formula, has been found of service:—

Acid. Nitrici diluti, *f* ʒxii.; Tinct. Cardam. comp., *f* ʒiii.; Syrup, *f* ʒiiiss.; Aquæ, *f* ʒi.; *misce*. Of this mixture, one or two small teaspoonfuls may be given every two hours (GIBB).

If convulsions should come on suddenly, and without headache or other symptom of inflammatory action, small doses of any opiate, and *mustard poultices* to the feet, may relieve the patient; and should convulsions still continue, an *asafetida injection* may be administered. It often happens that convulsions are combined with a suppression of the vomiting, and of the usual glairy discharge. In these cases *leeches*, followed by a large *linseed poultice*, should be applied to the epigastrium. If the unfavourable symptoms should advance, and headache or other symptoms show an affection of the membranes of the brain, *leeches* should be applied to the temples and *cold* to the head. When the poison excites inflammation of the tissues or substance of the lungs, *bleeding* to a limited amount by leeches may be required; but the affection is not to be subdued by bleeding, as in simple inflammation. There is in severe *hooping-cough*, as in *typhus*, *cholera*, and many other affections, an unknown element which modifies and gives a specific character to all intercurrent inflammations. If the intestinal canal be affected, some purgative, combined with *calomel*, may be necessary to free the bowels from their contents. If the stools be white and muciform, and the patient not relieved, an enlarged state of the follicles may be suspected, and a *linseed poultice* should cover the abdomen for some hours,

preceded by an *enema of syrup of poppies and barley-water*, which should be administered night and morning.

The disease having passed into the third stage, it is desirable to attempt to abridge the duration of the cough, which often extends to a most distressing length; and for this purpose *tonics, antispasmodics, counter-irritants*, and other remedies, either externally or internally, have been recommended. For medicine there is, perhaps, nothing better than a few drops of *ipecacuanha wine* combined with a minute proportion of *laudanum* or *belladonna*, to be administered every two, three, or four hours. The more stimulant antispasmodics, as *asafetida*, *castor*, *musk*, *oil of amber*, *cantharides*, and *camphor*, are the remedies which have obtained the most suffrages in the cure of this stage of hooping-cough. The two first are in most esteem. *Asafetida* should be given in emulsion, in the dose of one or two grains to a child two years old, repeated three or four times a day, or even as often as every two or three hours. Cullen preferred *cinchona* to *asafetida*, and considered it "the most certain means of curing the disease." Many other remedies have been mentioned, as *alum*, *hydrocyanic acid*, *oxide of zinc*, *arsenic*, and many preparations of *iron*. All of these remedies have been found to a certain extent useful as tonics; but in estimating the results of remedies, we should be careful not to mistake temporary recovery for cure; and the fact of so very many remedies being highly spoken of suggests doubt as to the value of either.

When internal remedies have failed to make any impression, relief may be obtained by means of local treatment, or by derivatives, as by some liniment or embrocation, as the liniment of *camphor* or of *ammonia*, or with *asafetida*, *oil of amber*, *oil of turpentine*, or the *tincture of cantharides*. The general opinion, however, is, that these do little good unless they contain some opiate, whose absorption they facilitate, and this may be attended with danger. The application of strong solution of *nitrate of silver* to the skin over the larynx has been much recommended. Foot baths and the warm bath have also been used, and often with much efficacy. A change of air is a resource of great value. A change from the bad air of a town to the purer air of the country is at all times a great benefit; it has also been found that a change from the country to the town is not less beneficial, and that the patient is benefited even by the removal to a short distance from home.

Dietetic and General Treatment.—The patient should not be allowed animal food from the commencement almost to the termination of the disease *in its acute form*. It is desirable also that the temperature of his apartment should be regulated, and that he should not be exposed to any considerable or sudden change from heat to cold. In mild weather also, if no local symptom forbids, he should be permitted to take exercise in the open air. He should likewise, next his skin, wear flannel.

There are no known means of prevention, except an entire removal from every source of contagion.

MUMPS.

LATIN EQ., *Parotides*; FRENCH EQ., *Oreillon*; GERMAN EQ., *Ziegenpeter*—Syn., *Mumps*; ITALIAN EQ., *Parotitide*—Syn., *Orecchioni*.

Definition.—*A specific fever with inflammation of the parotid and other salivary glands, which is contagious and sometimes epidemic.*

Pathology.—(a.) *Causation.*—This disease is most common in male children; less frequent after than before puberty; and second attacks are rare. Nevertheless it is not confined to childhood or youth; unprotected adults and persons of advanced age may suffer from it. It is sometimes epidemic, and in certain localities it prevails more than in others; so that local causes may have to do with its propagation and maintenance. Epidemics of mumps usually occur in spring and autumn, when the weather is cold and damp. It is rare in the dry warm weather of summer. A *symptomatic parotitis* often occurs in the course of severe fevers (typhus and enteric); and it has been noticed in cases of cholera. In some epidemics of typhus it follows almost every case, and is sometimes a fatal complication, from the tendency to purulent infiltration of the gland, with suppuration of the interstitial tissue—all of which, rapidly extending, assume the fatal characters of diffuse phlegmonous inflammation. It is a result also of pytalism from iodine or from mercury. The *idiopathic parotitis* of mumps is quite different, and rarely proceeds to suppuration. The disease is extremely infectious, and is spread by contagion. Of direct propagation from person to person, Dr. John Harley gives the following example:—“A medical student had *mumps* in London, at a time when his mother was staying with him. They remained in town till the swelling disappeared, and then went—a hundred miles into the country—home. There was no *mumps* in that neighbourhood; but a fortnight after their arrival one of the children was taken with the disease, and it afterwards successively affected, at regular intervals of a fortnight, each member of a large family” (HOOPER’S *Physician’s Vade Mecum*, 7th edition, p. 558), thus also fixing a fortnight as the period of incubation.

(b.) *Morbid Anatomy.*—The disease commences as a catarrhal affection of the gland ducts of the parotid (VIRCHOW). The swelling that ensues is a soft swelling; and as it disappears rapidly when improvement takes place, the swelling is probably in great part composed of serous infiltration among the connective tissue of the ducts; and extends far beyond the borders of the gland. When suppuration does take place, as it may in patients of unhealthy constitution, the swelling, in place of beginning to subside about the fifth or sixth day, continues painful and hard; and when abscesses form, they either open on the cutaneous surface or into the external auditory meatus. If suppuration does not take place, still there often remains a circumscribed, hard, painless swelling in the region of the parotid, which exists for some time.

Symptoms.—Febrile phenomena generally precede the local symptoms, but sometimes they are associated with pain and uneasiness in the region of the parotid. The pain on moving the jaw soon becomes so great that mastication becomes impossible in severe cases. Considerable

fulness and soreness prevail at the angle of the jaw over the malar region and region of the parotid, generally on both sides. Beneath one or both ears redness may prevail, with pain on pressure. The pain becomes so great as to prevent sleep at night in severe cases; and then the swelling generally extends to the submaxillary glands, and to the tonsils, and neighbouring parts of the pharynx, so that to open the mouth is impossible, and swallowing becomes increasingly difficult and painful. The region of the swollen glands becomes tense and glossy; sometimes of a dusky rosy hue. When both sides are affected at the same time, the face is of an enormous size, especially as to fulness and breadth, and with a double chin. This condition continues for four or five days, after which it gradually subsides, and resolution ensues, the fever and pain gradually subsiding, and the swelling rapidly diminishing. The fever ceases about the fifth or sixth day; and after eight or ten days the face has generally assumed its natural dimensions. The temperature sometimes reaches as much as 103° or 104° Fahr. Occasionally during the course of the disease, but generally towards its subsidence, the testicles swell, or the mammæ in the female. Pain in the region of the ovaries, increased by pressure, shows that the ovaries in woman inflame similar to the testicles in men. In some severe cases the cerebral membranes become implicated, as also the gastro-enteric mucous membrane. These complications generally come on suddenly, often with symptoms of an alarming kind—high fever, delirium, and sometimes severe collapse.

Treatment.—Constant hot fomentations (after leeching in severe cases) should be applied to the swollen parts. The maintenance of a constant but gentle action on the bowels, by saline cathartics, must be attended to. Absolute rest ought to be enforced, a farinaceous diet enjoined, and no irritating applications ought to be applied to the swollen glands. The disease runs a definite course, and is not likely to terminate unfavourably.

INFLUENZA (EPIDEMIC CATARRH).

LATIN EQ., *Cattarrhus epidemicus*; FRENCH EQ., *Grippe*; GERMAN EQ., *Grippe*—Syn., *Influenza*; ITALIAN EQ., *Influenza*.

Definition.—*A specific catarrhal inflammation of the mucous membrane of the air passages, and generally also of the digestive organs, with severe constitutional disturbance. The disease is invariable in its essential characteristics, frequently prevailing as an epidemic, attended with lassitude and prostration to an extreme degree, with special and early implication of the naso-laryngo-bronchial mucous membrane. Frequent chills occur, and great sensibility to cold exists over the surface of the skin; the eyes become injected, and tend to fill with tears, the nostrils discharging an acrid fluid; attended with fixed and intense pain in the head, mostly frontal over the eyes, sometimes also attended with giddiness. The nights are sleepless, with delirium or lethargy; cough prevails, with yellow expectoration, most troublesome at night, and tending greatly to increase the headache. Fever attends the disorder, sometimes slight*

and sometimes severe, and of a type varying in different epidemics and localities. The duration of the fever is definite, of from four to eight days (PARKES). The sense of taste is generally greatly disordered, and there is great anxiety and depression over the region of the heart.

Pathology.—(a.) *Historical Notice.*—We have no credible accounts of the existence of influenza previous to the tenth century. In 1311 it was fatal throughout France. In 1403 the courts of law in Paris were closed on account of the deaths. Towards the end of the twelfth and thirteenth centuries it was observed that catarrh was not only endemic in particular districts, but that it occasionally spread over large portions of country; while still later, in the year 1557, it was found to prevail epidemically, not only over the whole of Europe, but even over the whole of the northern hemisphere, beginning in Asia and proceeding westward, till it terminated in America. In the eighteenth century, having advanced westward till it reached the Elbe, it passed over the intermediate countries and reached England, where the stream broke into two branches, the one crossing the Atlantic to America, while the other retrograded south-east through France, Spain, and Italy, till it was lost in the Mediterranean. Influenza has occasionally originated as far eastward as India; but more commonly it has broken out in the north of Europe, as Moscow, Warsaw, or Dresden. It seems probable that, like the poison of *Cholera Indica*, its spread may be limited to a small number of primary foci. In nearly every volume of the *Calcutta Transactions* we find accounts of some catarrhal fever spreading for a season along the banks of some principal river, and then subsiding; so that it is evidently only occasionally and at long intervals erratic, as in 1729, 1743, 1775, 1782, 1831, 1833, and 1837. The influenza, therefore, is both endemic and epidemic; and, in the latter case, we find it, at least in Europe, spreading from east to west, prevailing in the depths of winter as well as the heights of summer, lasting nearly the same space of time, from four to six weeks, in the different towns and cities it attacks, affecting contiguous places in different degrees and at different times. On looking to the habits of this poison, it is probable that its actions are not limited to man; for in most years, when influenza has been epidemic, a similar disease has been epizootic, especially among horses and dogs, as in the years 1728, 1732, and 1775. It is a disease of extraordinary rapidity of progress; and as its diffusibility is great, so are its periods of recurrence frequent—those cycles of its visitation which are as yet beyond our comprehension to explain.

(b.) *Causation and Modes of Propagation.*—Influenza is for the most part so universal a disease that large portions of the population of every country in which it has prevailed, without respect to age, sex, or condition, have been commonly infected. The air seems to be the chief medium; and the special agent of propagation seems to be one capable of indefinite increase, continual reproduction to a greater or less extent occurring, the virus being given off with the breath (PARKES). In general, women, from being less exposed to the weather, have suffered in a smaller proportion than men, and children less than either. In all epidemics the aged suffer most. The ages from ten to sixty furnish the most patients. The ages from thirty to forty furnish most male patients,

and from twenty to thirty most female patients. It has been remarked in several epidemics that the lower parts of towns have been more generally and more severely affected than the higher and more healthy districts. The nature of the "*epidemic influence*" which gives rise to *influenza* is quite unknown. Sudden changes of temperature appear to assist the development of the influenza poison; and exposure to cold predisposes the individual to the disease. A specific poison is believed to be absorbed, when, after a period of incubation varying from one to two or three days, or even to two or three weeks, it produces disordered functions of the great nervous centres, causing great general depression, extreme debility, together with slight or severe remittent fever. The specific actions of this poison are on the mucous membrane of the eyes, of the nose, and of the bronchi; in a smaller number of cases, on the mucous membrane of the fauces, causing sore throat; and in a still smaller ratio, on the substance of the lungs and on the pleura, causing inflammation of those organs. In most instances the disorder terminates in diarrhoea. These different pathological phenomena vary in frequency and complexity in different seasons and places. In most cases, when the poison is of sufficient intensity to produce fever, the type is remittent in this country, with exacerbations in the evening. Its usual duration is two, three, or four days, when it terminates in an abundant sweat, which not unfrequently leaves great debility behind it. At the same time, however (or, it may be, preceding or succeeding the fever), the patient has in general been seized with a slight inflammation of the ocular and nasal membranes, followed by coryza, or the serous discharge of a common cold or catarrh; and this inflammation generally extends to the larynx and trachea, or to the lungs. Few persons suffer more than one attack of influenza in the same epidemic, although many relapse; and one attack of this disease in no degree protects the constitution from a second attack in another epidemic.

(c.) *Morbid Anatomy*.—Patients die chiefly from pneumonia, in which the lesions are seen to occupy most commonly the middle and lower lobes, and only rarely the summits of the lungs. The forms of pneumonia are principally serous inflammation and red hepatisation, the latter occasionally interspersed with a few points of pus. In the fatal cases of pneumonia connected with influenza, Gluge found exudations in the bronchia, which he compared to the false membrane of croup. Such exudations were seen in the hepatised portions of the lung as white, elastic, firm cylinders filling the bronchia, from the fourth or fifth divisions of these tubes, into such as are not more than a quarter of a line in diameter. The inner membrane of the bronchia in such cases was extremely reddened, but not softened.

The Symptoms of influenza assume a variety of different forms, and the period of latent incubation is very short, alternating with flushes of heat and repetitions of chills, especially along the spine. Catarrh often exists without the fever, and, in a smaller number of cases, the fever without the catarrh. High fever, severe nervous depression, extreme prostration, anxiety, and præcordial oppression, are the most prominent symptoms; while in other instances the bronchial affection alone harasses the patient. The disease usually begins suddenly, with marked elevation

of temperature, and with chilliness and shivering, rapidly succeeded by an immediate and evident impression upon the mucous membranes of the nose, mouth, frontal sinuses, trachea, and bronchial tubes, to a greater or less extent. General soreness accompanies these symptoms, with severe, darting, neuralgic headaches, aching of the limbs, listlessness, great mental depression, complete anorexia, and an extraordinary weakness, which, in the experience of Dr. Parkes, bore a close ratio to the extent of the pulmonary affection, and consequently to the severity of the disease. The fever which accompanies these symptoms is slightly increased towards evening. Patients were usually seen about the third or fourth day, and then they were found complaining of cough, tightness of the chest, of pain in the epigastrium, and also of dyspnoea. The face was flushed, and sometimes swollen, the alæ of the nose red, the lips vesiculated, and the mucous membrane red, dry, and swollen. Sneezing is common with frontal headache, and pain in the eyeballs, and affection of the conjunctiva, the eyes streaming with coryza, and the voice altered as in a common cold. The tongue was moist, or coated with a yellow mucus, and taste was vitiated, the skin soft and without morbid heat, the pulse little augmented in frequency. But although each of the particular symptoms might be mild, there was a languor, debility, and dejection of spirits far beyond what might have been expected, and almost exceeding that of common continued fever. These symptoms were in many instances long in subsiding. The average duration of the cases in the epidemic of 1847 (so admirably described by Dr. Peacock) was from three to five days in the mild forms, and from seven to ten in the more severe. In mild cases such phenomena constituted the whole disease, and the patients recovered about the eighth or tenth day, after suffering for a few hours from sharp diarrhoea or profuse perspiration. In many instances, however, the patient, in addition, suffered from mild or severe sore throat; or cough came on, and continued for many weeks. In a few cases the symptoms were of a more aggravated character, the fever being more marked, the pulse accelerated, the skin hotter, and the cough more troublesome, with marked prostration; and these conditions have often been followed by inflammation of the lungs.

The pulmonary complications may be—(1.) Capillary bronchitis; (2.) Bronchitis supervening on tuberculous disease of the lungs; (3.) Bronchitis with disease of the heart or *aorta*; (4.) Pneumonia; (5.) Laryngeal inflammation. These are all apt to manifest themselves insidiously. The accession of capillary bronchitis is indicated by the chest symptoms becoming more severe and the cough paroxysmal, and the dyspnoea at first quite disproportionate to the cough and to the physical signs. The expectoration is scanty, and consists of small yellowish pellets, forming tenacious masses of a peculiarly nodulated form. The pulse becomes rapid (120 to 140), the tongue covered with a white-brown fur, and prostration is extreme. The only auscultatory signs are roughness of the inspiratory murmur, with occasional sibilus, and slight crepitation at the back. There is soreness and contraction of the chest, but no acute pain. Crepitation, unattended by dulness on percussion, soon extends over a greater or less extent of both lungs; and the dyspnoea speedily becomes so intense as to prevent the patient from lying down; the lividity of the

lips and face increases, and the eyes become prominent. The cough is now very frequent, the sputa very viscid, of a greenish-yellow colour, without air-bells, and often streaked with blood. The respirations are quickened; but there does not appear to be any uniform connection between the extent of the disease and the disturbed ratio of the pulse and respiration movements. The general rule is, that the respirations are relatively more quickened than the pulse (PEACOCK, PARKES). The physical signs soon become modified by rapidly developed emphysema of the lungs. Generally, it may be said that the *capillary bronchitis* of *influenza* is distinguished from *pneumonia* by the greater severity of the general symptoms; by the tendency of the fine crepitation of the early stage to pass into subcrepitant and mucous râles, rather than to give place to evidences of condensation; and by the peculiar characters of the cough, which is paroxysmal, and not attended by pain; and lastly, by the character of the expectoration, which consists of whitish viscid pellets, cohering into irregular masses, and destitute of the glairy adhesive character, russet colour, and small air-bubbles of pneumonia expectoration (PEACOCK). Inflammation of the substance of the lungs seldom occurs till the second or third day, and more commonly not till the fifth or sixth. In some years it has been characterised by well-marked symptoms, as pain in the side, dyspnoea, and by purulent or sanguineous expectoration; but in general the pneumonia has been adynamic in character, and presented a striking contrast to the usual symptoms, there being scarcely any local pain; the pulse, ordinarily so large and full, has been slow and small, and though sometimes counted between eighty and ninety, has ranged most commonly from sixty to seventy. The face also, instead of being full and red, has been sharp and pale, the lips blue, and the extremities cold. The patients also who generally preserve a good deal of power in the ordinary forms of pneumonia, were now so weak that they were obliged to be supported while auscultated. Even this mode of exploring the chest did not afford the usual indications; for crepitation was rare, the respiratory murmur was heard, except in a few points, all over the chest, and there was little or no bronchophony. The auscultatory signs are in general those simply of bronchitis, dry rhonchus in some parts, and harsh vesicular murmur in others. The expectoration likewise had not the characters observed in simple pneumonia; for instead of being purulent and mixed with blood, it was thin, transparent, and viscid, and, if fever prevailed, it was usually of an adynamic character, marked by a brown tongue, an accelerated pulse, and occasionally by delirium. Throughout the progress of this disease the symptoms of nervous derangement are much more prominent than in ordinary catarrh, and the muscular debility is great,—the most distinguishing feature of the disease. So great is this prostration, that in some instances the patient has fainted merely by attempting to sit up. The extreme debility often continues after all other symptoms have passed away. The disease generally terminates favourably by perspiration, or by a copious secretion of mucus from the bronchia, or a copious discharge of urine, which deposits a sediment on cooling. Towards the termination of the complaint, rheumatic affections, especially of the face and head, assume an intermittent type.

Prognosis.—The uncomplicated disease is rarely fatal. Children and

persons under forty die in a very small proportion, unless in a previous state of ill-health. The mortality, however, among the aged has in every country been great from influenza. It has been remarked, also, that the disease, if not fatal in itself, left the patient, of whatever age, greatly debilitated in body and depressed in spirits; and that those with tender lungs who suffered from it fell into phthisis, or continued to cough for several months afterwards, so that a complete recovery was often long and tedious. In the epidemic of 1847 the mortality was greatest amongst the adults and aged. In childhood the average mortality was raised 83 per cent.; in manhood, 104 per cent.; and in old age, 247 per cent. The epidemic of 1847 was much more fatal in the insalubrious parts of London than in those less unhealthy; and according to Dr. Peacock's experience, the mortality of influenza was owing more to the condition in which the disease found the patient, than to any inherent power of the poison itself—a result conformable to general experience and the returns of the Registrar-General (PARKES).

Treatment.—As a general rule, the great majority of cases in epidemics of influenza have scarcely required any medical treatment. In 1782 it was observed that “many, indeed, were so slightly indisposed as to require little or no medicine; nothing more was wanted to their cure than to abstain for two or three days from animal food and fermented liquors, and to use some soft, diluted, tepid drink. A gentle purgative at the beginning of the disease was useful in moderating the fever; and nature seemed to point out the repetition of it afterwards when there was pain in the stomach and bowels, and a tendency to diarrhoea.” The same was observed in 1762. Nothing, likewise, was observed so successful in mitigating the cough as to open the bowels, and afterwards giving a gentle opiate at night. In the year 1837 it was also remarked that, as long as the symptoms were limited to cough, hoarseness, headache, or other pains moderate in degree, the patients all recovered by putting them on a low diet, by attending to their bowels, and confining them for a few days to the house. It was quickly found that the disease ran a course scarcely influenced by medicine. A small number, however, required medical aid, either from the severity of bronchitis, the occurrence of pneumonia, of angina, or of severe dyspnoea, of the disordered state of the bowels, or more frequently from the debility induced by the disorder. Blood-letting, even by leeches, is always hurtful. It does not relieve the fever, and increases the nervous depression. But when pleurisy, bronchitis, or pneumonia may supervene, leeches to the chest, or cupping, may relieve symptoms. A mild purgative dose of calomel (one to three grains) should be given once at first, followed by a saline purgative. Dark-coloured motions are brought away, the spirits improve, and the fever abates. But mercury must not be given beyond an occasional purge. Emetics are also to be avoided. They increase depression, and are apt to produce irritability of the stomach difficult to subdue. Hot fomentations, bran, mustard, or linseed meal poultices to the chest, or flannel or cotton wool envelopes, are often of great comfort and usefulness. During convalescence *sulphate of zinc* is a useful tonic when the expectoration is thin and spumous; and alkalis are more useful when it is viscid and glairy (PEACOCK). *Nitrate of potash*, highly diluted and mixed with lemon

juice and sugar, is a most useful drink. From 60 to 120 grains in twenty-four hours may be taken. Stimulants are early called for on account of the intense prostration. *Opium* must be used with great caution in the severe pulmonary complications. Its use ought to be put off till the later stages, otherwise it may increase the tightness of the chest and the dyspnoea. It must not be given till all danger of lung congestion is past. When the fever and other immediately alarming symptoms of the influenza had ceased, there frequently remained a teasing cough, and the convalescents in general complained of languor, want of appetite, and that their sleep was broken and unrefreshing. For removing these complaints, change of air and riding on horseback were most effectual. To some they were absolutely necessary: and, in addition to these, mild tonics, or the natural chalybeate waters drank at the Spas, were of singular service. In slight cases it was sufficient to limit the patient to white fish and puddings; and in the more severe forms, to slops and light puddings. The night air was universally prejudicial. It does not appear that any precautionary treatment was of service in preventing the spread of this disease among the attendants on the sick.

GLANDERS.

LATIN EQ., *Equinia*; FRENCH EQ., *Morve*; GERMAN EQ., *Rotz*;
ITALIAN EQ., *Cimurro*.

Definition.—*A specific catarrhal inflammation of the nasal mucous membrane, produced by the contagion of matter from a glandered horse, and intercommunicated between men and horses. It is a febrile disease of a malignant type, characterised by vascular injection of the nasal mucous membrane, from which an aqueous, viscid, glutinous, or purulent discharge proceeds. Chancre-like sores are formed on the mucous surface of the mucous membrane, extending to the frontal sinus and neighbouring mucous surfaces. The lymphatic glands enlarge in the vicinity of these mucous membranes.*

Pathology.—(a.) *History.*—The horse, the ass, and the mule, animals possessing an undivided hoof, are liable to a specific disease termed glanders. Under the common name of *Farcinoma*, veterinists have considered *glanders*, *farcy*, and *grease* of the horse as one and the same disease; but the influence of the animal poison in man has been considered by the Royal College of Physicians to give rise to three distinct diseases—namely, *glanders*, *farcy*, and *equinia mitis*. The weight of evidence, however, is in favour of the view that *glanders* and *farcy* are varied expressions of the same disease, according to the locality in which the specific lesions induced by the virus develop themselves. It may be shortly stated, that in *glanders* the nasal passages especially suffer; while in *farcy* it is the lymphatic system which is affected. A number of severe accidents occurring to persons employed about glandered horses has shown that the poison producing glanders is capable of being transmitted from the horse to the human subject, and again from the human subject to the horse and to the ass; and there is reason to believe that it is capable of being transmitted

from one human being to another (ZIMMERMAN, in *Virch. Arch.*, Vol. XXIII., p. 209, and *Year-Book of New Syden. Society*, 1862). The attention of the profession was first called to this subject by Mr. Muscroft, in *The Edinburgh Medical and Surgical Journal*, in the year 1821, where he relates the case of the whipper-in of the Bradworth Hunt, who wounded himself in cutting up a glandered horse for the kennel, and died, at the end of a week, of confirmed glanders. Two similar cases appeared in the same work about two years afterwards. Simultaneously with Mr. Muscroft, Dr. Copland, in the course of a discussion at the Medico-Chirurgical Society of London, stated that the fact of the disease having been thus communicated had been proved by cases that had occurred in Germany, which were published in *Rust's Magazine* for 1821. The cases excited but little notice till Mr. Travers published his valuable work on *Constitutional Irritation*, in 1828, containing a letter from Professor Coleman on the transmission of glanders from the horse to man, and from man to the ass, together with some other cases which had fallen under his own observation. The subject was now followed up by Dr. Elliotson in two papers in the *Transactions of the Medico-Chirurgical Society*, narrating three cases which had occurred in his own, Dr. Roots's, and Dr. Williams's practice. At length all the then known facts were collected in an elaborate paper by Rayer, in the sixth volume of the *Mémoires de l'Académie de Médecine*.

(b.) *Causation and Mode of Propagation*.—The cause of glanders in the horse is but little understood. It is probably due to a specific miasmatic poison. Glanders, however, when it affects the human subject, has in all instances been distinctly traced to the glandered horse as the cause. No instance is known of the disease occurring primarily in man. In the horse certain predisposing causes greatly favour, and are perhaps necessary to, the spread of glanders, such as dirty, close, ill-ventilated stables, especially if the situation be low and damp. Horses when crowded on board transports are greatly liable to this affection. Bad food is a powerful predisposing cause in the horse, especially when these animals are picketed on military service, and exposed to the inclemency of the weather. At the close of a campaign the cavalry are often decimated by this disease; and towards the termination of the Peninsular War the losses from this cause are said to have been enormous. The cases occurring in the human subject are too few to allow of any inference being drawn as to the influence of the predisposing causes in the production of glanders; but the disease generally occurs in young men; and probably a close investigation would have shown that the habits of the patient were such as to fall within those laws which favour the production of the disease in the horse. There can be no doubt that the disease is contagious among horses, and if a glandered horse has been introduced into stables, the stock in these stables will become diseased. In this country the law is severe against offering for sale, or even working, a glandered horse; which shows that the opinion of our ancestors, time out of mind, has been that glanders is a contagious and a fatal disease. In Germany the belief of contagion is so general that it is said the law directs any horse that has been in contact with a glandered animal to be immediately killed. Direct inoculation from horse to horse has been effected (COLEMAN, PEAL, RENAULT); and Leblanc has repeated experiments till he has demonstrated that not only is glanders contagious, but

that farcy and glanders are mere varieties of the same disease,—the farcy matter producing glanders, and the matter of glanders producing farcy. Cases of the transmission of glanders from the horse to man are now numerous; and that the disease is actually glanders has been shown by the fact that two asses were inoculated with matter taken from the arm of a person labouring under the disease (consequent on a puncture received in dissecting a glandered animal), and both animals died of glanders. These experiments have been repeated with similar results, so that no doubt can exist of the fact. It seems proved, therefore, that glanders is transmissible from the horse to man, and again from man to the ass. It has been contended, also, that if glanders is transmissible from man to animals, the disease must be capable of being communicated from one human subject to another; and a case of this description appears actually to have occurred in St. Bartholomew's Hospital about twenty years ago, when the nurse, a healthy woman, contracted the disease from a patient in the ward; and, after a short illness, died with every symptom of glanders. The fact of repeated inoculation with glandered virus distinctly shows that fomites may be so infected as to produce the disease. The spread of the malady has been attributed to healthy horses having drunk out of the same pail or trough with a glandered horse, or to licking the neighbouring rack or partitions of the stalls in which a glandered horse had been placed; or by hay left by glandered horses being blown into the paddock where healthy horses were. Thus the specific poison of glanders has been introduced into the system both by the cutaneous and mucous tissues.

The result of all these observations shows that in cases of glanders a specific poison is implanted which infects the blood, and, after a given period of latency, produces, in slight cases, an abscess at the point of puncture, followed by some tumors in the course of the absorbents connected with the punctured part. In severe cases fever is previously set up, and after this has continued for some days, there follows either a diffuse or an eruptive inflammation of the mucous membrane of the nostrils and of the trachea, terminating in suppuration, ulceration, or gangrene; also some inflammatory affection of the lung, together with the usual farcy button or bud tumors in different parts of the body. Transmission of the virus from one animal to another, or from one individual to another, is the most frequent, and probably the only manner in which the malady is propagated. It is a specific disease, which is purely contagious; and the specific virus is contained alike in the discharges of the "*farcy buttons*," the flow from the nostrils, the blood, and in the excretions, saliva, urine, and sweat. It is known also that the disease has been transmitted at a distance of ten or twelve paces from the original source of infection, probably by particles of nasal secretion cast off by the horse—the snorting of the animal to get rid of the stuff in his nostrils carrying the secretion into the air, and so dispersing it about. Two cases have recently occurred in the occupants of a London mews, who slept in rooms over stables in which were glandered horses, and who had no other communication with the animals.

(c.) *Morbid Anatomy*.—In the cases collected by Rayer, either ecchymosis, ulceration, or gangrene of the mucous membrane of the *septum*

nasi, or of the sinuses, were found in the nose and nasal fossæ. The mucous membrane of the larynx, or trachea, has likewise been found studded either with the peculiar eruption, or diffusely inflamed or ulcerated, so much so that in one case the epiglottis was in part destroyed; and the lungs either gorged with blood, or the seat of lobular pneumonia, or of *vomicæ*. In Dr. Roots's case there was an encysted abscess of the lung, which contained about two ounces of pus. Besides these affections of the more vital organs, a number of small farcy tumors have been found in different parts of the trunk and extremities, remote from the point originally punctured. These tumors were in different states of inflammation, some being white and indurated, others soft and injected, and others in a state of suppuration. In Dr. Roots's case an abscess on the back of the hand communicated with the articulation of the metacarpal bones; and in another case an abscess had opened into the knee-joint. The absorbent vessels had likewise been found inflamed along the arm from the point of puncture, or site of primary inoculation, and the glands to which they lead have been found enlarged and indurated, or in a state of suppuration. In substance the nodules or tumors consist of germinating cells, young, small, and delicate, with numerous free nuclei. In the older nodules the cells are large, distinctly nucleated, lying closely together, forming and making up almost the entire mass of the tumor. As they progress, the older cells degenerate, and become partially filled with fat globules. They lose their sharply defined contour and break down, so that the nodule finally contains only a mass of detritus, with a few isolated elements. In the horse, ass, and similar beasts, these nodules are of the size of a hemp-seed or a pea; and the resulting ulcers are at first solitary or in groups; but as they coalesce they give a peculiar worm-eaten appearance to the surface. The ulceration penetrates to a great depth, laying bare portions of cartilage and bone, which die and are discharged. The catarrh is very intense around the ulcers, and the discharge is at first thin and transparent, which subsequently becomes thick, opaque, and purulent, discoloured by blood, acrid and fetid, and containing the *debris* of dead tissue (VIRCHOW, NIEMEYER).

(d.) *Period of Latency*.—The poison of glanders has a short period of latency, by inoculation about the second day. M. Gerard, an ex-veterinary surgeon of the French "Artillerie de la Garde," states that he introduced the matter of the discharge every day into the nostrils of certain horses, by means of a brush, and that the disease appeared in one on the seventh day, but in two others not till the thirty-second day. In the human subject the poison has in general been latent from two to eight days after the accident of inoculation. Where the virus has been implanted through a wound, the first symptoms generally appear within three or four days; but when there has been no breach of surface (as when the virus has been inhaled), the malady may not break out for months. The course and magnitude of the disease also differ in the two cases.

Symptoms.—When the poison acts on an abrasion, or is inoculated, the earliest symptoms are usually local. The wound inflames at the site of injury; the lymphatics proceeding from the part form knotted chains, and their glands form painful swellings. The cutaneous inflammation assumes an erysipelatous character, and is attended by intense

œdema. Blebs form, and pustules full of discoloured ichorous contents, and sometimes real gangrenous bullæ, arise upon the skin. Abscesses may also form, and diffuse destructive phlegmonous inflammation occur in parts about the inflamed lymphatics (NIEMEYER). Sometimes the disease remains thus a local one, so far as the lesions are concerned; but there is always fever of an intensity which is the measure of the severity of the case. The febrile signs of general implication of the system usher in the expression of the lesion.

Glanders may be either acute or chronic. Acute glanders is expressed by primary fever, followed by local inflammation; chronic glanders, when the local inflammations exist *per se*. The acute disease is ushered in by an attack of primary fever, with or without rigors. In some cases the rigors are repeated several times. The skin grows hot, thirst increases, the pulse quickens, and the patient feels depressed and languid, complains of pain in the head, sleeps badly, and has no appetite. Violent pains in the joints, especially of the greater articulations and the muscles, are constant and characteristic phenomena. The pains in the joints are augmented by motion and pressure, and sometimes the joint is swollen. Hence the disease may be mistaken for an attack of acute rheumatism. This stage of the disease may continue for three or four weeks or more, during which the symptoms either increase steadily or gradually grow milder, and subside into the second or eruptive stage. The parts become the seat of phlegmonous tumors, accompanied with much pain, redness, and tenderness; these more commonly terminate in abscess, sometimes discharging a laudable pus, but more usually a bloody sanies, and rapidly become gangrenous. Towards the close of the disease, in almost all cases there has been a discharge of matter more or less purulent, viscid, and mixed with blood, from the nostrils. This lesion begins by an erysipelatous inflammation upon the exterior of the nose and its vicinity. The nose, the eyelids, and the forehead swell, assuming a dusky redness. The skin becomes covered with blebs, which are the precursors of gangrene (NIEMEYER). The patient cannot breathe through his nostrils, and there flows from them a fluid which is at first scanty, thin, and mingled with streaks of blood, afterwards the discharge is foul and sanious. The period at which the symptom appears is not constant. It has been seen as early as the fourth, and as late as the sixteenth day. In the course of the disease the eyelids are generally tumefied, and discharge a thick viscid matter, like that from the nose. Enlargement of the sub-maxillary glands occurs. If the patient lies on his back, the discharge from the pharyngeal sinuses and nose flows back through the posterior nares into the pharynx, and causes much distress, with erosion of the soft palate and tonsils. The mucous membrane is then of a deep red hue, and is soon covered with ulcers and sloughs. One of the most remarkable symptoms of acute glanders in man is an eruption on the face, trunk, limbs, and genital organs. This eruption has been compared to *varicellæ*, to *small-pox*, and to *ecthyma*; but in fact it is an eruption *sui generis*, and cannot be compared to any other. It has been observed to occur about the twelfth day, and to be preceded and accompanied by profuse fetid sweats. It has been described as pustular; but Virchow has shown that in glanders there is no elevation of the epidermis by any exuda-

tion, but the skin beneath circumscribed spots of the cuticle becomes disintegrated. The affected portions of the skin are at first intensely red, at a small point, resembling a flea-bite. A papule then forms, which afterwards appears like a pustule, from the thick yellow liquid which is found to fill up the hole or loss of substance from the disintegrated corium. The contents of this glander-pustule sometimes grow bloody, and dry up into small blackish brown crusts. Besides this eruption, a number of black bullæ have been observed on the nose, forehead, below the ears, on the fingers, toes, and genital organs, and these have been followed by gangrene more or less extensive and deep. The pulse is full and quick in the early stages, but towards the close it becomes rapid, small, irregular, and even intermittent. The tongue varies, as in typhus, being first white and coated, and subsequently brown or black. Diarrhœa and meteorism often complicate the disease, and blood has been observed in the stools. Cerebral disturbance has come on as early as the second day, but more commonly not till towards the tenth; sometimes marked by a singular want of intelligence, at others by a sinister presentiment, followed by stupor and death. Acute glanders is rapid in its course, and two-thirds of the cases have terminated before the seventeenth day; some have died on the twenty-first day, a few on the twenty-eighth day, and only one has survived till the fifty-ninth day.

Chronic glanders, or farcy, differs from acute glanders in the circumstance of the local lesion preceding the general febrile derangement, the introduction of the poison being followed in a few hours by inflammation of the lymphatics proceeding from the wounded part, and extending sometimes to the elbow or axilla, and involving the axillary glands. These effects are followed by inflammation and extensive abscesses in the subcutaneous cellular tissue, often involving the whole limb. From this state the patient may recover; but should these abscesses be multiplied over various parts of the body, and be accompanied either by the pustular or gangrenous vesicular eruptions, or by both, the result is generally fatal; hectic symptoms supervene, and hasten the final catastrophe. The disease has terminated within a fortnight, but more commonly it has not proved fatal till the end of a month; and, in cases still more chronic, a twelvemonth has been known to elapse before the patient finally recovered or died.

Prognosis.—Of fifteen cases of acute glanders collected by Rayer only one recovered. Of fifteen cases of acute farcy only five recovered. Of seven cases of chronic farcy only one died. Of the three cases of chronic glanders two died. A favourable prognosis, consequently, is only warranted in the chronic form of the disease.

Treatment.—All the remedies hitherto tried in acute glanders have failed. The advent of typhoid symptoms has led to the administration of *quinia*, *valerian*, *serpentaria*, *ammonia*, and other stimulating medicines. In the more chronic forms of the disease the recovery of the patient has appeared to be owing to the excellence of his constitution; during the natural elimination of the poison, to good ventilation, and to generous diet, rather than to any effect produced either by general or local treatment.

Preventive Treatment.—The prophylactic treatment is the same as that

of all other contagious diseases—namely, being careful to avoid all contact with the morbid poison, especially when a finger or other part of the hand is abraded; and if by accident the veterinary surgeon should inoculate himself, he ought instantly to destroy the part with *potassa fusa*. It has been recommended, after the disease has been set up, to extirpate the enlarged glands; but according to the doctrines set forth in the text, this practice is as unwarrantable as hopeless.

FARCY.

LATIN EQ., *Farcinimum*; FRENCH EQ., *Farcin*; GERMAN EQ., *Wurm*; ITALIAN EQ., *Farmino*.

Definition.—*An inflammatory affection of the skin and of the absorbent system produced by the contagion of matter from a horse having farcy.*

Pathology.—This affection is to be regarded as a form of glanders; for there is evidence to show that the poison of glanders will produce the anatomical lesions of farcy, and of farcy glanders. While glanders is expressed especially by lesions of the mucous membranes, farcy is expressed mainly by a tubercular and pustular lesion appearing upon the skin, followed by suppurating, bloody, or gangrenous ulceration in various parts. A general inflammation of the lymphatics and of the glands may occur, giving rise to the small tumors known as “*farcy buds*,” or “*farcy buttons*.” These gradually suppurate, and the secretion from the open sores contain the specific virus of farcy and glanders. The *bud farcy* and the *button farcy* are regarded as different forms of the same disease. The “*bud farcy*” consists in the formation of a number of tumors on different parts of the body, as on the head, neck, and extremities, and particularly on the hinder ones of the horse. These tumors are formed not only by enlargement and inflammation of the glands, but of the areolar tissue; and at the end of four or five days the tumor softens and ulcerates. Similar bud-like tumors form in the substance of the pituitary membrane, and these follow a similar course of softening and ulceration. The form known as “*button farcy*” is an inflammation limited to the lymphatic glands and vessels, without involving, to any considerable extent, the neighbouring connective tissue. It usually commences in the hinder extremities of the horse, causing lameness and enlargement of the limb; and when the valves of the lymphatic vessels become thickened, it forms the peculiar tumor called “*farcy bud*.” When the lymphatics only are swollen, and show this peculiar enlargement throughout their course, the lesion is known as “*farcy pipe*.” The tubercles, buds, or tumors of farcy are larger than the tubercles of glanders. They contain also a greater quantity of caseous matter, and may be isolated or aggregated in clusters, chains, or wreaths, according as different parts of the lymphatics are specially involved. The open sores are round, with elevated and everted edges, a foul, irregular, sloughy base, which discharges profusely an ichorous fluid, gluing the surrounding hairs together, and drying up with them into a hard crust. The clusters lie deeper in the skin than the peculiar cutaneous eruption of glanders. They are in the form of large, flat, hard red tumors, from

which the cuticle is finally separated by an effusion of blood, so as to form livid or bluish bullæ. The upper portion of the cuticle covering these clusters is also infiltrated by a hæmorrhagic exudation, and the whole afterwards breaks down into a pulpy detritus (VIRCHOW). The "farcy buttons" of the subcutaneous tissue and muscles arise either with diffuse inflammatory infiltration of the adjacent parts, and all the symptoms of a severe phlegmon; or small, circumscribed, hard boggy tumors form, which are so painless that they may develop unobserved. The ulceration of these may extend even to the bones. Farcy is more common in man than glanders; and the peculiar eruptions on the skin of the farcy tubercles are larger and more numerous than those in brutes; and their contents, as well as those of the tubercles in the muscles and lungs, are purulent rather than caseous. The lymphatics and glands are similarly implicated in man as in brutes; and the skin becomes involved in a malignant erysipelas, tending to its great destruction by gangrene.

EQUINIA MITIS.

LATIN EQ., *Equinia Mitis*; FRENCH EQ., *Grease*; GERMAN EQ., *Equinia Mitis*—Syn., *Gutargiger Wurm*, *Druse*; ITALIAN EQ., *Equinia Mite*.

Definition.—*A pustular eruption, produced by the contagion of matter from a horse affected with the grease.*

Pathology.—Grease in horses is a specific inflammation of the sebaceous glands of the skin in or about the heels. There is at first a catarrh from these glands, the secretion which flows being greatly vitiated in quality, and it gives forth a very loathsome smell. The catarrhal inflammation of the glands passes into ulcers, which present the appearance of unhealthy sores, the discharge from which is very offensive. The ulceration assumes the form of deep, raw, and excessively tender cracks or fissures, from which exuberant granulations may arise, to which the name of "*grapes*" has been given. These may increase to an enormous size, accompanied with much general swelling of the leg of the animal. It is this earlier catarrhal discharge, which is at first a very thin acrid exudation, that is the most virulent; and when it escapes on to the hands of those who attend upon horses, dressing their heels when affected with the grease, gives rise to the specific pustular affection of *equinia mitis*. The term *equinia* was proposed for the disease known as glanders by Dr. Elliotson, and was intended to express its derivation from the horse: just as the term *vaccina* or *vaccinia* was before given to cow-pox;—subsequently, Dr. Shedel adopted the name of *equinia mitis* to the more mild affection derived from the grease of horses, now being described. The eruption is pustular, and the pustules are large, very similar to those of *ecthyma*, elevated above the skin, and with a red, purple tumid base. If there are abrasions, or much unsoundness of the skin of the hands, the eruption may be extensive in proportion, and very severe in man.

Symptoms.—The eruption is preceded and attended with febrile symptoms; and, in some cases I have seen, with very marked depression and tremor or shivering. The eruption takes about eight days to become

pustular; but the pus seems watery and abnormal; and in ten or twelve days the lesions begin to die away, scabs form, which fall off, leaving well-defined scars. There is generally considerable constitutional disturbance and much foulness of tongue; rapid pulse, with alternate heats and chills.

Treatment.—The greatest relief to the constitutional disturbance is obtained by frequent purgation with *aloes* combined with *ammonia*, and such moist local applications as may relieve pain.

ERYSIPELAS.

LATIN EQ., *Erysipelas*; FRENCH EQ., *Erysipèle*; GERMAN EQ., *Erysipelas*
—Syn., *Rothlauf*; ITALIAN EQ., *Risipola*.

Definition.—*An acute febrile disease, characterised by a peculiar inflammation of the integument, tending to spread indefinitely, and even to involve the areolar tissue beneath the skin. It is accompanied by severe general symptoms.*

Pathology.—(a.) *History.*—Erysipelas has occurred in a sporadic form at all times; and, with the exception of the tropics, in all regions of the earth's surface (HIRSCH). It has never prevailed in an epidemic form in an entire community, but has often prevailed as an epidemic in inclosed establishments, especially in large towns, as in surgical hospitals, military hospitals, prisons, and ships, sometimes associated with epidemics of puerperal fever. Destructive epidemics of erysipelas have now and then occurred in Europe; and several parts of America have of late years been the scene of similar ravages, especially in the New England States, the South-Western States, and the interior of Pennsylvania (Dr. WOOD). In Scotland the disease is known by the name of the *Rose*; in England it is sometimes called *St. Anthony's fire*.

(b.) *Causation and Modes of Propagation.*—As in other diseases of this section, it is believed that in erysipelas a specific poison is absorbed and infects the blood, and that after a period of latency it produces fever. The specific action of the poison, however, is mainly made manifest by a peculiar inflammation of the skin, characterised especially by "diffuseness," and originating from a focus of infection. It may also extend to the subcutaneous areolar tissue, and is especially apt to become complicated with inflammations of membranous structures covering the viscera, such as of the brain, the lungs, and bronchi, or of the gastro-intestinal mucous surface. The inflammation and the fever are of a peculiar nature, not yet clearly understood. It diffuses itself rapidly, mainly by continuity of surface; but it no less frequently tends to change its seat, and is not limited by any adhesive action. The lesion is not one simply of the skin; but it may affect any surface, external or internal, such as the mucous or serous membranes, the connective tissue, the orbit, the scalp, the lining membrane of arteries, the veins and lymphatics; but the essential morbid condition is the constitutional or general disease, often marked by much gastro-intestinal irritation (ERICHSEN). There may, indeed, be no local or cutaneous evidence of lesion, but merely the general febrile symptoms, with irritation of the stomach and bowels. Mr. Erichsen has seen such a form of disease prevail amongst patients in a ward where erysipelas prevailed

amongst other patients in the ordinary cutaneous form. The lymphatic vessels and glands are invariably implicated in the lesions of erysipelas (RICHTER, NIEMEYER, BUSK). It seems to belong to that class of diseases which result from blood-poisoning, of which pyæmia is the type (SAVORY). The inflammation generally appears at the seat of any injury to the skin as the infective focus of an erysipelas, such as a wound, excoriation, or other breach of surface, and is most intense there. By some, indeed (TROUSSEAU, for example), it is held that erysipelas always originates from some external injury or irritation, which may be very slight. "The fact, too, that erysipelas begins by preference at the orifices of the body, and at the point of junction between the skin and mucous membrane—*i.e.*, at the points which are most exposed to all kinds of irritation, speaks strongly in favour of this belief" (ZUELZER). But this character erysipelas has only in common with other eruptive diseases, as Sir James Paget has described in his admirable Address on Surgery, delivered to the British Medical Association at their meeting in London, in August, 1862. He noticed that, "having cut a boy for stone, the boy became very ill three days afterwards, and seemed in danger of his life; but soon a vivid red eruption appeared at and about the wound. This was measles, earliest and most intense at the seat of injury, just as erysipelas might have been. Thence it extended, and ran its ordinary course, and did no harm." Sir James Paget states that he has seen similar events with scarlet fever, the eruption commencing in an injured and inflamed knee. Dr. William Budd records similar events in a case of small-pox, in which the eruption first appeared and was most intense over a bruise on the nates. The argument from such facts is, "that the local determination of erysipelas, and of other allied diseases, after operations, is no proof of their local origin or local nature." Sometimes it happens that no traumatic cause can be discovered; but even here it is not the less evident, from the form and character which the patch of redness presents at its first appearance, that the spreading inflammatory process has started either from one centre of origin or from several neighbouring centres at the same time. In either case it is equally plain that a local infection has occurred—*i.e.*, that a contagium has been inserted, or otherwise came into existence, in the tissue of the affected part (BURDON SANDERSON, *Append. Report of Med. Officer of Privy Council, N.S.*, No. III., 1874, p. 20). The effects of age in predisposing to this disease are considerable. New-born children, for instance, are occasionally subject to it, but from that period to adult age it is seldom witnessed. The period of life most subject to acute attacks is from twenty to forty; and to frequent asthenic attacks, from forty to old age. Both sexes suffer in nearly equal proportions. The spread of erysipelas has been so frequently observed, both in the sick-room and in the wards of hospitals, that no doubt can exist of its being communicable by impalpable emanations. It is thus eminently contagious. Dr. J. C. Steele writes, in his excellent *Annual Report on Guy's Hospital* for 1863, that "for some years past it has been customary to place patients suffering from erysipelatous wounds in these (the medical) wards, in order to diminish as much as possible the risk of extending infection in surgical wards, as well as to promote recovery in the patients themselves. It happened, however, that in one of the wards of the new hospital into

which a patient suffering from erysipelas was placed in the course of the past year, *five* persons suffering from other complaints were attacked with the disease; and although none of the cases was attended with fatal consequences, the occurrence is sufficient to point out the danger which must be occasionally apprehended." Mr. Erichsen gives a remarkable proof of the contagious nature of erysipelas occurring in the winter of 1851, in one of his wards in University College Hospital. "The hospital," he states, "had been free from any cases of the kind for a considerable time, when, on the 15th of January, at about noon, a man was admitted under my care, and placed in Brundrett Ward. On my visit, two hours after his admission, I ordered him to be removed to a separate room, and directed the chlorides to be freely used in the ward from which he had been taken. Notwithstanding these precautions, however, two days after this a patient, from whom a necrosed portion of ilium had been removed a few weeks previously, and who was lying in the adjoining bed to that in which the patient with the erysipelas had been temporarily placed, was seized with erysipelas, of which he speedily died. The disease then spread to almost every case in the ward, and proved fatal to several patients who had recently been operated upon" (*Science and Art of Surgery*, Vol. I., p. 495). Another striking instance is given by Mr. De Morgan, which came under the observation of Dr. Rogers of Dean Street. A medical student went into the country, and was requested by his brother, a medical man, to visit an erysipelatous patient. Whilst leaning over her he became conscious of a nauseous odour, which almost caused him to be sick. A few days after he was suddenly seized with shivering, followed by fever. Erysipelas shortly came on in the head and face, and he died after a week's illness. Such cases are too numerous and too striking to admit of any reasonable interpretation save that of the presence of a specific poison, miasm, or virus, capable, by contagion or infection, of inducing the specific disease. That it is communicable by some palpable virus, was also long ago demonstrated by inoculation with the fluid contained in the phlyctenæ or vesicles of a genuine erysipelas, when a red, painful, diffused swelling and inflammation analogous to erysipelas was produced. The danger, however, attending this experiment has not allowed it to be repeated (WILLAN). Dr. Orth has also succeeded in the inoculation of rabbits from the liquid of an erysipelatous bulla, so as to produce a spreading inflammation, which corresponded in every important respect with erysipelas. The redness and swelling extended from the wound over the whole trunk and extremities, the temperature at the same time rising to 106° or 107° Fahr. Erysipelas also spreads by *fomites*. In hospitals, wards are occasionally obliged to be cleared out and thoroughly limewashed to stop the continued spread of this disease. In the navy its spread by *fomites* is well understood. It has spread extensively, and for long periods, in the Birmingham, Edinburgh, Glasgow, and London hospitals, and is only got rid of by emptying and limewashing the wards. The old "Dreadnought" hospital ship in the Thames was so impregnated with the *fomites* of erysipelas, that she had ultimately to be broken up, and a new vessel substituted.

A patient having passed through an attack of erysipelas has no security against future attacks of the disease; and many persons suffer repeatedly

from erysipelas—some periodically. There appears to be a constitutional predisposition to the disease in some people, and especially in those who have periodic attacks. Some women have attacks every month. Intemperance, and all influences which tend to depress the system, predispose to the disease, and hence partly the prevalence of the disease in hospitals. But there are unquestionably some unknown conditions of the atmosphere which seem to favour the dissemination of the disease. It has been observed that this predisposition to erysipelas exists in the ordinary wards of hospitals at the same time that puerperal fever prevails. Indeed, some forms of puerperal fever seem to be identical with erysipelas. Each may co-exist or seem so interchangeable, that either seems capable of exciting the other by contagion. It is also within the experience of surgeons that patients will perish a few days after operations when erysipelas prevails in an hospital without any external sign of even erythema. They die of an internal form of erysipelas (WARREN, CAMPBELL DE MORGAN). It is also within the experience of physicians that malignant forms of pneumonia will result from communication with erysipelatos cases; and that such forms of pneumonia may convey erysipelas to others having open sores or wounds (GIBSON, CAMPBELL DE MORGAN).

(c.) *Morbid Anatomy*.—The cutis, when examined after death, whatever may have been the form of the disease, is always found greatly thickened and infiltrated; but the redness, except in cases of gangrene, has entirely disappeared. The appearances found after death from erysipelas are similar in many respects to those found in cases of typhus fever. The visceral organs do not present any special characters beyond congestion of spleen, and kidneys, and liver, and mucous surface of the stomach and intestines. As in typhus fever, so in erysipelas—the blood is obviously altered. It may be very fluid and thin, or pitchy and dark, staining by decomposition of its colouring matter the inner surface of the large blood-vessels; and showing the veins distinctly through the skin by transudation of the coloured fluid through their coats. It has no disposition to separate into its fluid part and coagulum; and any coagulum that may form is loose and thin. It is also mentioned by Mr. Busk, that in all the fatal cases he has examined the lungs were highly congested; and that, on close inspection, the smaller pulmonary vessels were always found to contain pus. In fact, a minor degree of pyæmia was always present; and, when the head was the seat of erysipelas, pus was similarly found in the small veins of the head (CAMPBELL DE MORGAN). Inflammation of the mucous membrane of the throat; hypostatic congestion with croupous pneumonia; enteritis in the lower portion of the jejunum, are lesions which have been found after death. The skin is affected in its whole thickness, and also the subcutaneous areolar tissue. These structures are cedematous, swollen, and penetrated by large, finely granulated white blood-corpuscles. The blood-vessels are enlarged and filled with corpuscles; but their walls are unchanged. An extensive occurrence of globular (punctiform) bacteria (without spontaneous movement) has also been found everywhere in the inflamed tissues, especially abundant in the cedematous parts contained in lymphatic canaliculi; and, much less so, in the blood (BIESIADECKI).

(*d.*) *Period of Latency.*—Erysipelas has occasionally followed a few hours after exposure to the infection. Dr. Elliotson thinks five days elapsed in his own case, and Dr. Watson has given three cases in which the interval was a week. It has been observed in hospitals that a fortnight has elapsed after its subsiding in one case and appearing in another in the same ward. It is probable, therefore, that the period varies from two to fourteen days.

Symptoms.—The attack may be sudden, or ushered in by rigors, irregular flushings, muscular pains, accelerated pulse, white tongue, nausea, vomiting, and deranged bowels. Sore throat is an early and constant accompaniment. These symptoms last for some hours—perhaps till the end of the second night or beginning of the third day—when the fever becomes continued, the tongue brown and dry, and shortly afterwards the cutaneous inflammation appears, but without any remission of the fever. Idiopathic erysipelas is very constantly preceded by fever—eighteen times out of twenty; and although it may be supposed that the fever is consecutive to the inflammation of the skin, yet, before the redness of the skin is seen, the temperature, if measured by a thermometer, will be found above 98.6° or 99° Fahr., and attended with general malaise. The course of the fever in erysipelas is very similar to that of measles, and the maximum temperature may be higher than in that disease; but the advance of the fever to its height continues longer—seven to eight days—and the epoch for the commencement of the defervescence vacillates between the *fourth* and the *eighth* days. Temperature rises rapidly, and may reach 104° or 105° Fahr. on the first evening of the cutaneous inflammation. It may so remain for a variable number of days, with slight but decided morning remissions, or may continue rising to 106° or 108° Fahr., as long as inflammation of the skin continues, every extension of the inflammation being indicated by a rise of temperature. Thus, high evening exacerbations may be maintained for from one to two weeks or more. In this respect it differs greatly from measles. The defervescence, as a rule, is rapid, the normal heat being attained, or nearly so, in from twelve to thirty-six hours. Frequently, however, the case is not terminated therewith. New relapses may take place, and the disease may be prolonged through two or even three weeks. These relapses are severally of short duration; but they come on again and again, and are ushered in by increase of heat, connected with a renewed spread of the cutaneous affection. It is only after the eruption has ceased that complete and definite defervescence ensues. In favourable cases the temperature generally falls to the normal standard on the sixth or seventh day of the eruption. Very sudden changes of temperature are characteristic—4° or 5° Fahr. in twenty-four hours, or a fall of 7° or 8°, commencing immediately on the appearance of the characteristic redness (COMPTON). This erratic and protracted form of erysipelas is most frequently met with in the aged, associated with gouty or rheumatic states of the system, as well as with albuminuria or renal disease. The *local* symptoms vary according to the part affected, the mode of termination of the inflammation, and also according to the character and duration of the fever. When erysipelatous inflammation affects the face, it may begin either in the skin or in the subjacent areolar tissue. If the areolar

tissue be primarily affected, the face at the inflamed part becomes swollen; but the skin suffers no discoloration for some hours, so that it is impossible to distinguish it from an ordinary attack of swelled face. At length, however, the skin inflames, and the part is red, hot, and painful, as well as swollen. At the commencement of erysipelas of the face the attack is usually partial, and perhaps limited to the bridge of the nose, to one ear, to the lower eyelids, or to one cheek; but in severe cases it gradually extends, often involving the whole of the integuments of the face, head, and neck; so that at the end of three or four days those parts

TYPICAL RANGE OF TEMPERATURE IN A CASE OF ERYSIPELAS AFFECTING THE FACE. THE RECORDS INDICATE MORNING (A.M.) AND EVENING (P.M.) OBSERVATIONS (C. L. Fox).

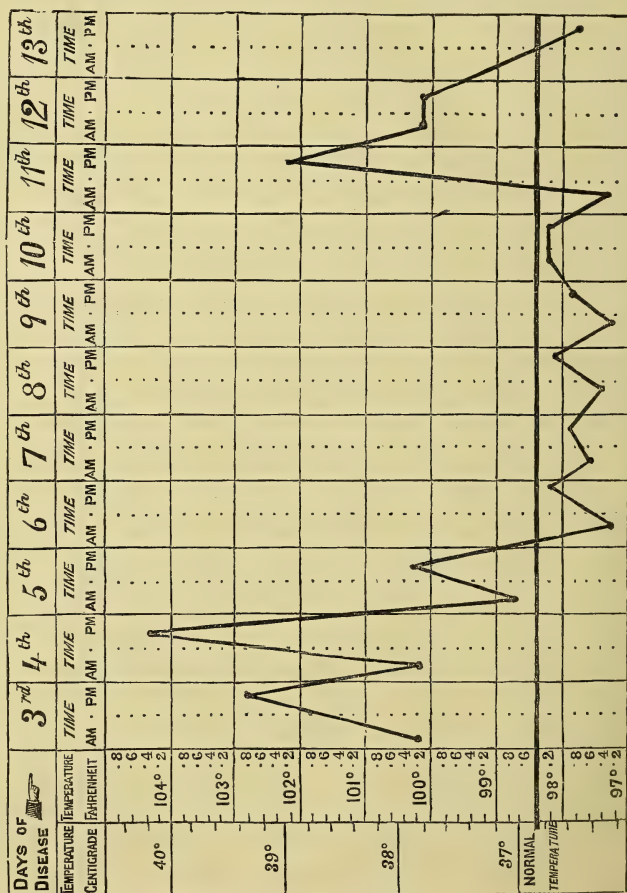


Fig. 92.

present a strangely swollen, disfigured, and even, in some instances, hideous appearance, scarcely a feature being discernible. The nostril, moreover, is imperforate from internal swelling, so that the patient is obliged to breathe with his mouth open, while the inflammation may extend to the auditory passages, and render him completely deaf. Extension of inflammation to the membranes of the brain sometimes takes place, while the external inflammation continues. This untoward event is followed by

delirium and coma. But delirium frequently supervenes in the course of erysipelatous attacks, independently of any extension of the disease to the membranes of the brain. It commences with wandering of the mind at night, similar to that which is observed in fever. Utterance is given for the most part to low, muttering, and rambling expressions, which rarely assume a noisy character, but which in fatal cases terminate by coma. When the patient has been of dissipated habits, or is otherwise of a dilapidated constitution, then the delirium resembles that of *delirium tremens*, not due to inflammation of the brain, but in consequence of an altered condition of the blood and of the nervous system (BARCLAY).

On the fourth, sixth, eighth, or some later day, the bright red colour of the skin changes to a deeper hue; the serum effused is absorbed, desquamation takes place, and the skin gradually returns to its natural colour. It is not unusual, however, for abscesses to form, particularly on the eyelids or cheeks, which, being opened quickly, heal, and hardly retard the convalescence of the patient. In some cases the disease becomes *erratic*, and extends over the chest or down the back, and desquamation is seen going on in one part while the erysipelas is spreading in another. This is characteristic of cases associated with gouty or rheumatic constitutions. The extremities are more commonly the seat of erysipelatous inflammation than the trunk, and the lower extremities are more frequently affected than the upper. When these parts are affected, the fever is less severe than in erysipelas of the head; but the local symptoms are generally more formidable, for the degree of heat is greater, and the pain so severe, that the weight of a sheet can hardly be borne. The inflammation likewise may obviously involve the lymphatic vessels, when they can be traced by white or red lines for many inches, as from the knee or elbow to the inguinal or axillary glands; and these sometimes enlarge and suppurate.

If the erysipelatous inflammation ends in suppuration, the pus is always diffused, *infiltrated* through the areolar tissue, and the swollen limb gives a peculiar sensation to the hand, like passing over a quagmire. The dark, black, discoloured appearances of gangrene are too obvious to render any description of the parts so affected necessary.

Numerous varieties of erysipelas are referred to in practical works, especially surgical (to which the student is referred), most of which are modifications of the disease as above described. Besides the external forms of erysipelas, the disease also expresses itself by internal lesions, such as diffuse inflammation, spreading over *mucous* or *serous surfaces*, the *lining membrane of arteries, veins, and lymphatics*. Of *mucous surfaces* the most liable are the *fauces*, the *pharynx*, and *larynx*. These parts are liable to be affected by the continuous spreading of the inflammation from the scalp or skin of the face. This form of the disease in the throat is eminently contagious. When the *larynx* is implicated, the danger is extreme from the oedematous infiltration of sero-plastic fluid, which occupies the *rima glottidis*, and extending into the interior of the larynx, obliterates its cavity by the extent of the swelling. Such swelling, however, never spreads below the true vocal chords (ERICHSEN). *Erysipelas of serous membranes* is chiefly seen in the *arachnoid* and *peritoneum* after surgical operations, or, as regards the *arachnoid*, after injuries of the head or erysipelas of the scalp. As regards the *peritoneum* it is met with after

operations for hernia, or as the result of disease or injury of the pelvic or abdominal organs, or of puerperal fever.

Diagnosis.—The diagnosis of erysipelas is in general easy. For a few hours, perhaps, if a joint be attacked, it may be mistaken for acute rheumatism; or if a surface be attacked, it may be confounded for a short time with erythema; but the intumescence and spread of the disease quickly enable us to rectify the error. Frank has pointed out a symptom which he considers diagnostic—namely, that whenever a patient has exhibited, for twenty-four or forty-eight hours, an intense febrile movement, attended with *pain, swelling, and tenderness of the lymphatic glands of the neck*, he does not hesitate to announce the approaching development of erysipelas; and in no case has the diagnosis been invalidated by the result. Chomel expresses a similar belief; and Mr. Campbell de Morgan's experience confirms the statement that the neighbouring lymphatic glands are generally, if not always, tender *before* erysipelas of a part sets in; and Mr. Busk concurs with this statement of Mr. De Morgan's experience. These records go far to confirm the view that the specific inflammation of erysipelas commences in the lymphatics.

Prognosis.—The most experienced physicians consider erysipelas, at all periods of life, "a dangerous and deceitful disease;" and when it fixes on the face or scalp it is one of the most serious diseases to which an aged person especially is liable; for when all appears to be going on well, the membranes of the brain may become involved, or the powers of life may give way, the patient sinking suddenly under the depressing influence of the poison. The disease may extend to the fauces or the glottis, and the patient then dies suddenly from the oedema of the glottis which supervenes. The disease is peculiarly fatal to drunkards and to patients of broken-down habits; and frequently recurring attacks show such a bad state of health as indicates a speedy break-up of the constitution (MAC-LACHLAN). It is five times more fatal to people above sixty than to people between fifteen and sixty years of age (*Registrar-General's Fifth Report*, p. 456).

Treatment.—"Experience has proved that general bleeding has no other effect than to blanch the eruption, without notably abridging its duration;" and from the nature of the disease blood-letting in any form is not to be thought of. Rest, saline laxatives, cooling drinks, and low diet, are the elements of treatment in mild and simple cases. An emetic is useful at the commencement; and I have seen, in the practice of an eminent surgeon (Mr. Syme) that frequent resolution of an erysipelatous attack has followed an antacid laxative, such as forty grains of *rhubarb* to sixty of *bicarbonate of soda*, divided into twelve powders, one to be taken every six hours, together with the counter-irritation of a mustard poultice over the stomach. Laxative and cathartic remedies are to be selected and apportioned according to the violence of the attack and its nature, as tending to the unfavourable results of the specific inflammation already noticed. *Calomel* is a most valuable purgative, as a sedative in febrile disturbance, especially when followed by *castor-oil*, or the common *black draught*. The indications to the use of certain remedies, as given in the treatment of scarlet fever, are equally applicable here. If the febrile state is not subdued, antimonials are of great service; and so far as they are

diaphoretic in their action they tend to subdue the vascular excitement. If symptoms of nervous depression ensue, *opium*, or *opium* and *ipëcacuanha* are indicated, also *wine* and *quinine*, *ammonia* and *camphor*, in asthenic cases with a tendency to a typhoid state. The *tincture of the perchloride of iron*, in doses of ten to thirty minims, *three, four, or five* times a day, in water, infusion of *quassia*, or *calumba*, is now also a remedy much in use, and it may be alternated with the *syrup of the phosphate of iron*, in doses of a teaspoonful three or four times a day. In cases where the system is obviously gouty or rheumatic, and where the joints are affected, *colchicum* with *saline diaphoretics* are the most efficient remedial agents. Local applications are potent for good or evil, and must therefore be used with great caution. The effects seen on the skin do not constitute the whole disease; and if the development of these processes on the cutaneous tissue is imprudently interfered with, there is imminent danger to internal organs. To check the advance and prevent the encroachment upon new territory, rather than to subdue it, if already in possession, ought to be the sole aim of local treatment, and to mitigate the local pain and uneasiness. Bland *mucilage*, such as that of viscid *linseed tea*, from which light muslin cloths have been steeped and spread over the inflamed surface, sometimes affords relief. Dry *flour*, or *rye-meal*, frequently dusted from a dredge-box over the erysipelatous patches, are soothing applications. A thick layer of cotton-wool dusted with tutty powder is also a soothing application to the part. A lotion of *nitrate of silver* painted daily over the affected parts sometimes gives great relief (one scruple of the nitrate to an ounce of water, to which ten drops of *dilute nitric acid* are added). *Dilute nitric acid* should, at the same time, be given internally, if typhoid symptoms predominate, as in the following formula :

R. *Acid. Nitrici* dil., ʒi.; *Syrup. Zingib.*, ʒss.; *Aqua*, ʒvss.; *misce*. A tablespoonful every four hours.

To arrest the spread of the process over sound skin, *nitrate of silver* in very strong solution, or *tincture of iodine* have been considered efficient agents. A line of circumvallation is to be painted round the erysipelatous part, so as completely to inclose it. The *nitrate of silver* should either be employed in the solid stick; or as proposed by Higginbotham, in solution of eight scruples of the nitrate with twelve drops of nitric acid in a fluid ounce of water. Dr. Wood has practised with success and recommends the use of *tincture of iodine*. But in this country the boundary line of *nitrate of silver* has not been attended with success; and our best surgeons are now discontinuing the practice as a useless source of irritation (ERICHSEN, CAMPBELL DE MORGAN). *Sulphite of soda*, in solution of ten grains to one ounce of water, is recommended by Dr. Addinell Hewson of Philadelphia. He has never seen it fail to arrest the progress of the disease, if it has not advanced to the deeper parts below the skin. Lint soaked in the solution is to be applied to the affected part, and over a considerable distance beyond any visible appearance of redness. The lint is to be covered with oil-silk, to prevent evaporation. The application of *sulphate of iron* as a lotion or ointment tends to shorten the disease. In lotion, *one drachm of the salt to*

a pint of water; in ointment, a scruple to an ounce of lard (VELPEAU, LUKE, DE MORGAN). It has the inconvenience, however, of soiling linen with which it comes in contact. None of these lotions should be applied cold, but tepid or warm. *Collodion* and *iodised collodion* are also worthy of commendation. Long and deep incisions into the inflamed textures are sometimes demanded. This is more especially the case if there be tension of fibrous tissue, such as the subcutaneous fasciæ; and erysipelas of the head is frequently greatly alleviated by repeated innumerable minute punctures, made by the point of a lancet all over the parts of the face and scalp which are affected.

PYÆMIA.

LATIN EQ., *Pyæmia*; FRENCH EQ., *Pyohémie*; GERMAN EQ., *Pyæmie*; ITALIAN EQ., *Piemia*—Syn., *Piemasia*.

Definition.—*A febrile affection associated with a morbid state of the system generally, the invasion being marked by frequently repeated severe rigors alternating with profuse sweatings, and usually sequent on some inflammatory and infective condition of wounds, suppurative inflammation of bone, the puerperal state, or surgical operations, which results in the formation of secondary lesions in the internal visceral organs (most frequently in the lungs, the liver, kidneys, spleen, and brain), and also in the joints and connective tissue. Sometimes, but not necessarily, it is associated with phlebitis or embolism. The blood becomes poisoned, and contains an excess of white corpuscles and of fibrine.*

Pathology.—(a.) *History.*—Multiplicity of views and great uncertainty still pertain to many points regarding the pathology of *pyæmia*. It has been clearly shown that the word is itself a misnomer, which implies a theoretical origin of the disease now known to be incorrect. Nevertheless it is better to retain the name, provided that the constant and characteristic phenomena peculiar to the morbid state are comprehended by the designation.

A number of morbid processes, having many elements in common, of great practical importance, of frequent occurrence, and dangerous to life, have hitherto been described under the various names of *Pyogenic fever* (Sir WILLIAM JENNER); *Acute purulent diathesis* (TESSIER); *Purulent infection* (SEDILLOT); *Septic or putrid infection of the blood* (PANUM); *Septicæmia* (VOGEL); *Systemic infection* (the *ichoræmia* of VIRCHOW); *Putrid fever*; *Pyæmia* (*Pyohémie* of PIORRY); *Phlebitis*; *Surgical fever* (Sir J. Y. SIMPSON); *Purulent absorption* (SOLLY); *Multiple abscesses* (LARREY; POTR); *Suppurative fever* (BRAIDWOOD). The first two—namely, the *pyogenic fever* of Jenner, and the *purulent diathesis* of Tessier—are characterised by the occurrence of *multiple abscesses*, with true purulent contents. The remaining six of the morbid states which have been enumerated have been classed by Virchow, Bilroth, and others, under the common name of “*metastatical dyscrasiæ*.” In them there is evidence of the occurrence of secondary or metastatical inflammations, which seem like the translations of matter from one part of the body to another—a tendency to multiple centres or foci of inflammation, to “multiple

abscesses," and to suppuration in various parts of the body. Associated also with these lesions is the occasional formation of *clots*, *thrombi*, *plugs*, or *embolia*; and the occurrence of *thrombosis*, or of *embolism*, or of *phlebitis*, with the accumulation of and *softening* of infarcts in the minute tissue of visceral organs, such as the *brain*, the *lungs*, the *heart*, the *liver*, or the *kidney*, or of *gangrene* of the extremities. Thus there are several febrile conditions in which the blood is materially disturbed in various ways, tending to complex forms of lesions in many parts, especially to multiple centres or foci of inflammation, with a great tendency to suppuration, or liquefaction in them, or to death of limited areas of tissue. In the Gulstonian lectures delivered at the Royal College of Physicians in London, for 1853, Dr. Jenner directed particular attention to some lesions which are apt to follow immediately after the termination of acute specific diseases, such as *scarlet fever* and the like. The simplest form of these lesions consists in the formation of several (or multiple) abscesses of small size in the subcutaneous connective tissue, especially of the scalp, chest, loins, legs, or arms, accompanied by more or less febrile disturbance. Such multiple abscesses are (on circumstantial evidence only) presumed to owe their origin to a diseased condition of the blood—to represent what is popularly known as "the dregs of the fever"—as the media by which something unwholesome is ultimately evacuated—or as a *crisis* in which the specific affection terminates. Sometimes, on the other hand, these abscesses are accompanied by severe constitutional disturbance, and instead of being superficial, are deeply seated, either in the connective tissue or into the cavities of the joints, and in rare cases even into the serous cavities of the cranium, abdomen, or thorax. Cases of all these varying degrees of severity are observed, which seem to differ only in the more or less wide diffusion of the local affections. Such cases are found to be in close alliance with those diseases in which purulent discharges issue at the same time from the mucous passages, and to that chronic cachexia in which the least scratch or abrasion of the skin tends to "fester" and not to heal. This condition of the blood and system was first recognised and described by Tessier, in 1838, under the name of the "purulent diathesis," or "a tendency to suppuration in the solids and coagulable fluids."

In cases of this nature the febrile disturbance seems to be established before any local lesion is set up; and the morbid condition of the blood thus came to be looked upon as "a primary substantive affection," because it seemed to lead to a "fever" followed by these lesions (*Med. Times and Gazette*, May 7, 1853). The subcutaneous tissue and the joints are the most frequent seats of these abscesses; much less frequently the lungs or other viscera, which in the following class of cases are the most frequent seat of the multiple abscesses. Here we have an important distinguishing character between the two classes of diseases—namely, that the abscesses of pyogenic fever seem to be developed out of a *constitutional* state of general ill-health, whereas those which are about to be considered may be regarded from the beginning as due to a blood-poison, or other causes of irritation set up from external sources. In the former case the cause of the disease seems to be *constitutional*; in the latter, *specific*. Hence the London College of Physicians has placed

pyæmia among the General Diseases, Class A. Again, the class of lesions associated with pyogenic fevers is still further distinguished as of one kind, by the following characters:—(1.) The abscesses are not consequent upon the pre-existence of any wound, abscess, ulcer, clot, or plug in the blood-vessels; (2.) There is no evidence of the formation of pus or of fibrinous *debris* in the veins, nor of the passage of that fluid into the blood, and the establishment of *pyæmia* in the literal sense of the term; (3.) The symptoms, and still more the *situation*, of the disseminated abscesses differ in cases of *pyogenic* fever from those which occur in cases of true *pyæmia* or *phlebitis*. It is with *typhus* or *typhoid fever* only that *pyogenic fever* is apt to be confounded; but from this fever it is distinguished by the activity of the febrile symptoms at the outset, the early delirium, the absence of eruption, and the rapid formation of the numerous centres of suppurative action. The closest alliance, pathologically, of *pyogenic fever* seems to be with *phlegmonous erysipelas*; but from true *pyæmia*, either in the form of *septicæmia*, or in that combined with the tendency to multiple abscesses, it ought to be distinguished.

Thus under the generic name of *pyæmia*, *metastatical dyscrasie* have attracted considerable attention from several points of view:—(1.) In connection with various conditions of the veins, to which the name of “phlebitis” has been given; (2.) in connection with various changes in the blood itself, such as are indicated by the names of *septic* or *ichorous* infection, and more especially such as are described by Virchow and Bennett under the name of *leucocytosis* and *leucocythæmia*; (3.) in connection with the inoculation experiments made by Dr. Burdon Sanderson—experiments which have largely contributed to our knowledge of pyæmia. Several important questions associated with each of these views are still the source of controversy. *Pyæmia* literally means a condition in which there are pus-cells in the blood; but the expression has now come to imply that the blood is altered throughout the whole system by the poisonous action of putrid animal matter, which may act in the form of gases, fluids, or solid particles, and which so disturb its relations with the living tissues as to induce coagulation of the fibrine of the blood in some part, during life, within the blood-vessels. These changes are associated with fever and the formation of local infarcts in one or more of the viscera and other parts, usually accompanied by *phlebitis*. These secondary lesions appear first as minute spots of congestion; serum or lymph is then found, and subsequently what looks like pus. This later signification of pyæmia has been in some measure forced upon us, because it is found impracticable to say when any given specimen of blood is full of white blood-discs or of pus-corpuscles. It was once supposed that pus could be absorbed *as pus*, and be conveyed away in substance; and that two results might follow such an event—either, (1.) That the pus would be passed off by the urine or the fæces as an excretion, but still in the form of the original pus; or, (2.) That the phenomena of pyæmia would be the other alternative. Now we know that neither of these results ever takes place. Pus, *as pus*, is never taken up or absorbed into the system. Its fluid part only may be absorbed by veins or lymphatics; but the solid portion remains as a thick, inspissated, or concrete mass. Again, pus may be completely absorbed; but only after the cells have been reduced to a state of milki-

ness by fatty degeneration, and have become converted into an emulsive mass—a kind of milk—composed of water, albuminous matter, fat, mucus, cholesterine, sulphates, lactates, and the like, and in which also sugar may be present. Thus far the history of pyæmia shows that it has been mixed up with other distinct processes of disease.

Much, however, has been recently done to clear up this confusion. On Jan. 23, 1874, Mr. Prescott Hewett delivered an address before the Clinical Society of London on "*Pyæmia in Private Practice.*" He gave an account of twenty-three cases, in six of which only had an operation been performed. He considered that the cause of pyæmia had still to be worked out; and a valuable discussion followed his address, which was maintained at several subsequent meetings of the Society. Three years later (May 16, 1877) the then President of the Pathological Society (the late Dr. Murchison) named a committee to carry out an investigation in pyæmia and the allied diseases; and also made the gratifying announcement that a sum of £350 had been granted to the Society by the Local Government Board for this purpose.*

The committee reported to the Society on Tuesday, 20th May, 1879, generally to the following effect:—That *septicæmia* comprehends—(1.) *septic intoxication*, (2.) *septic infection*; and that (1.)—*septic intoxication*—includes those cases where the dose of the septic poison is so great as to kill within a few hours of its absorption—not a common occurrence in man. For such an occurrence to take place there must be a large quantity of septic material in a wound; and this material must be rapidly absorbed;—that (2.)—*septic infection*—includes cases of septic poisoning *without* metastatic inflammation—common *septicæmia*. In this disease there is a true infective process (see pp. 364-366, *ante*).

Mr. Jonathan Hutchinson has also given a clear expression to the meaning of the term *septicæmia*. It has, he says, at least two meanings, namely:—(1.) That it is due to some poison which is introduced into the patient's blood from without; that the poison then grows, spreads, germinates in the blood, and produces the symptoms. He would suggest—(2.) That the term be applied to the results of poisoning of the blood induced by the inflammation of the patient's own tissues (other than the veins), whether

* In a memorial addressed to Mr. Selater-Booth, along with an application for a grant of money, the attention of the Board was invited to several considerations, showing the importance of an inquiry into the nature, causes, and prevention of the infective diseases known as *pyæmia*, *septicæmia*, *purulent infection*, and *puerperal fever*. It was pointed out that the lives that are daily sacrificed to these affections are not only numerous, but in many instances the lives of persons whose existence is of the greatest value to the community; that, although the causes are unquestionably of such a nature as to admit of investigation, the actually existing knowledge of those causes is extremely imperfect; that, the field of research afforded by the Hospitals of London is of unrivalled extent; that this field could only be rendered available with the co-operation of the Surgeons and Physicians of these institutions, all of whom had already declared their willingness to give their most cordial support and assistance to the proposed scheme;—and finally, that, such an investigation is calculated to throw light upon the intimate nature of the whole class of infective diseases. The working committee to carry on the investigation consisted of Mr. Marcus Beck, Dr. Greenfield, Mr. McCarthy, and Dr. Ralfe. The other members consisted of representatives of the Metropolitan Hospitals, co-operating with the working members in organising and carrying out the research—namely, Dr. Broadbent, Mr. Callender, Dr. Green, Mr. Prescott Hewett, Mr. Hilton, Mr. Holthouse, Mr. Hulke, Mr. Hutchinson, Dr. Murchison, Dr. Bardou Sanderson, and Mr. Wood.

that inflammation arose spontaneously, or was caused by an irritant from without. It seems now to be clearly established that *septicæmia*, *pyæmia*, and *phlebitis*, are distinct pathological conditions—all of which may be coincident in the same patient (*Brit. Med. Journal*, April 17, 1875, and January 8, 1876);—that the stage of gangrenous inflammation of a part is an essential stage of *septicæmia*; and that it is quite possible for *pyæmia* to occur without any local poison introduced from without.

(b.) *Causation*.—As to the cause of *septicæmia*, the committee state;—(1.) That for the occurrence of *septicæmia* in the form of *septic intoxication*, there must be a large wound which from its situation or shape is difficult to drain. The most favourable condition for its production is the presence of septic material in a serous cavity, as in the peritoneum after ovariectomy, or in wounds of large joints;—(2.) That in *septic infection* the poison when absorbed multiplies in the organism; and the blood of the animal so infected is able, by inoculation, to start a similar condition in another animal. The effects of the poison are not proportionate to the dose; and the development of the disease is slower, and the duration longer, than in *septic intoxication*.

There is nothing more irritating or acrid than the fluid of a wound immediately after operation (BENJAMIN GOOCH). The fluids in such wounds are in some state of decomposition; the pus especially undergoes change, and acquires a toxic action in various degrees of putridity. It is, therefore, of importance to note that the composition of pus varies considerably; and the conditions under which it varies have not yet received the attention which its importance demands. It seems to vary with the locality whence it comes, and with the circumstances under which it is formed. The pus of a *pulmonary vomica* differs from that of a *psaos abscess*, and that again from the pus of a *mammary* or *hepatic abscess*. So also the pus of *syphilis* differs from that of *small-pox*. The very large and variable amount of organic elements which pus contains renders it also extremely liable to change; and the products of decomposition of the elements of which pus is composed are extremely various and diffusible—e.g., ammonia, gas products, and salts, leucine and tyrosine; the acids also of the butyric group, as well as formic acid. Another fact to bear in mind is the “spontaneity” with which these elements undergo the process of putrefaction, without any *apparent* co-operation of other matters, and solely by the influence of atmospheric agents. As constant products of such putrefaction there are always to be found *carbonate* and *butyrate*, and *valerianate* of *ammonia*, *sulphide* of *ammonium*, *sulphuretted hydrogen*, *leucine*, and *tyrosine*. By putrefaction, and under the influence of impure air especially, pus may thus undergo an acid or an alkaline fermentation. The former is rare; but when it does occur, there are developed volatile and fixed fatty acids, such as *butyric acid* and *margaric acid*. Unhealthy pus more commonly tends to become alkaline without becoming acid. It evolves *ammonia* and *hydrosulphate* of *ammonia*. The large compound granular corpuscles which attend inflammatory lesions soften and run together, nuclei disappear, and the whole becomes a mass of granules which are probably capable of reabsorption. This is the condition in *hospital gangrene*. Pus essentially consisting, as it does, of cells in a pus-serum, varying in size from $\frac{1}{2000}$ to $\frac{1}{3000}$ of an inch, has only one mode in which its

cells can find their way into the blood—namely, by the perforation of a blood-vessel by an ulcer or a puncture. An abscess close to a vessel may open into it; but the result is harmless if the pus is fresh and healthy. The passage of pus into lymphatic vessels is still more easy when such vessels run into open abscesses. But then lymphatics do not empty their lymph into blood-vessels (with one exception) till they have elaborated such lymph in the lymphatic glands; and from the nature of gland-structures it is known that no pus-corpuscle, as such, can pass a lymph-gland. Such glands not only *filter* mechanically, but are living absorbents of *some constituents* only with which they come in contact, and amongst others, no doubt, the constituents of the pus-fluid without the actual pus-corpuscles, whose *debris* would simply be retained within the glands. Irritation of these glands leads to continuous germination of the gland-cells, and subsequently to the passage into the blood, by the left jugular vein, of cells which cannot be distinguished from pus—the colourless blood-cells. Under such circumstances nothing is easier than to demonstrate what seems to be pus. The presence of white or pus-like cells in the blood, by what we know of blood formation, can be explained. We know that the blood gets these blood-cells—(1.) After every meal we take; (2.) by irritation of lymphatic and other glands from cachexia; (3.) with the advance of pregnancy and splenic enlargement. In scrofula, typhoid malaria, and cancer they also abound. The existence of pus in the blood cannot therefore be demonstrated as the term “pyæmia” has hitherto been understood to mean; and now, therefore, when the term is used, it is meant to imply that an unknown matter, derived apparently from the spontaneous decomposition of some kinds of purulent or albuminoid substances, has mingled with the blood—has poisoned that circulating fluid, or has so altered it that it tends to coagulate in the vessels during life, or gives rise to various secondary phenomena about to be noticed.

To define pyæmia completely, Dr. Burdon Sanderson writes that “we must take into account its mode of origin, its symptoms, and the anatomical changes which it produces; not confining our attention to either of these to the exclusion of the rest;” and he comprehends in his definition the following propositions:—

“1. Pyæmia originates by the introduction into the living tissues, and eventually into the blood, of a *poison* which is itself a *product of inflammation*.

“2. The action of this poison manifests itself in an alteration of the blood, and in disorder of the vital functions. The former of these is characterised by the presence of bacteria, and by change in the optical characters of the blood, which often becomes obviously more transparent and darker by reflected light than it is naturally. Of the latter—viz., the general disorder of the vital functions—the most prominent phenomenon is fever, which, in the more intense forms of the affection, is followed by collapse which culminates in death.

“3. More remotely, the disease manifests itself in secondary suppurations—i.e., in the formation of metastatic abscesses, which may occur either in the internal organs or underneath the skin. The special characters of these metastatic infective abscesses are those which are well known both to surgeons and physicians. They have the additional less known character, that the pus they contain is full of bacteria.”

As to the nature of the poison he writes:—

"I wish to show, (1.) that every pyæmic abscess contains a poison, which, when introduced either into the circulation or into a serous cavity, produces the symptoms of pyæmia; and, (2.) that we have this poison so entirely in our possession, and so far under control, that, beginning with an agent so mild in its action that it produces no marked symptoms, we can convert it into an agent of such intensity that it kills in two or three hours with formidable symptoms. This intensification is effected by a process which may be called cultivation. Dr. Klein made the important discovery that, if a pyæmic liquid were transferred to the peritoneum of a guinea-pig, and allowed to remain there for a couple of days, although it did not at first produce any intense symptoms in the animal itself, its toxic intensity increased in such a degree that, when the transudation-liquid produced in this was injected into another animal, it had acquired the most deadly activity; and that all such extremely active liquids were crowded with bacteria of a particular character, the increased number of which seemed to be in proportion to their toxic properties. With regard particularly to the question of bacteria, our observations lead us to conclude, first, that they *afford a characteristic by which we may distinguish the products of infective inflammations from those which are not infective*, and that their number affords an *indication of the degree of infectiveness*; and, secondly, that their *presence in the blood is an indication of that constitutional disturbance which accompanies infective inflammation*; not merely when that disturbance assumes the greatest degree of intensity, but in the slighter form of irritative fever. If these facts prove to be true, not only in the lower animals but in man, their importance is quite unaffected by any theory which we may entertain as to the origin of bacteria" (see also p. 370, *ante*).

But that the active principle of the putrid blood, pus, or fluids must be some other morphological or chemical constituent than bacteria is proven by the experiments of Wolff and Popoff; and as a result of Dr. Sanderson's own observations and experiments, it may be stated that although these living particles are probably the carriers of infection, there is yet no sufficient ground for supposing that their existence is the essential condition of infectiveness of certain products of inflammation. In septicæmia the blood is, indeed, profoundly altered—tending to putrescence (induced, it has been believed, by bacteria); and unless septicæmia goes along with pyæmia, bacteria are not present in pyæmia—an important distinction between the two diseases, if such a fact can be substantiated and maintained (see p. 370, *ante*).

"Pyæmia" as a specific original entity—the result of absorption of pus, as such—is not now believed in. It is now rather to be regarded as a collective name for different disease-processes, just as the essential phenomena of Bright's disease are brought about by several different morbid conditions of the kidney. It is especially necessary to distinguish its characteristics from the following phenomena—namely, (1.) Leucocythæmia; (2.) Embolism, with resolution of clots, putrid decomposition, or gangrene; (3.) *Phlebitis*. To these phenomena (excluding those connected with leucocythæmia) Virchow has given the general term of "metastatical dyscrasiæ." The phenomena presented by individual cases capable of

being classed under the head of *metastatical dyscrasie*, are sometimes so extremely varied that names have been given to diseases as well as to lesions which do not always have the same anatomical limit or range of significance. For example, suppurative fever caused by *embolism* may occur (according to Virchow) when a considerable fragment of a *clot* or *thrombus* becomes arrested at a certain point in the course of the circulation. It may then crumble down and be carried away by the pressure onwards of the blood, the minute particles being conveyed into still smaller vessels, and so limited deposits occur which may break down or suppurate. Thus far certain local lesions having the appearance of multiple abscesses may be accounted for, but the specific suppurative fever is not always present in such cases. Neither *phlebitis* nor *thrombosis*, with the phenomena of embolism, are constant in relation to pyæmia (SAVORY). *Pyæmia* is certainly not always a result of phlebitis. "The immediate cause of pyæmia in any given case is, that some diseased part (which need not be an external wound) so affects the blood circulating through it, that this blood afterwards excites destructive suppuration in parts to which the circulation carries it—namely, commonly first in the lungs, or (in certain cases) liver and lungs, and later generally about the body" (SIMON, *Public Health Report* for 1863, p. 60). In a case of which Mr. Simon was cognisant, and which is described by Mr. Bowman, fatal pyæmia in a young man was apparently produced by an ulcerated state of the mitral valve of the heart. In another case Mr. Simon records fatal pyæmia produced by the penetration of pus from a small mesenteric abscess into the thoracic duct; and in the thirteenth volume of the *Pathological Society's Transactions* Dr. Bristowe records several instructive cases, in which pyæmia complicated, *ab initio*, cases of *idiopathic necrosis unattended by external wound*. I believe, on careful analysis of the subject, and from what I have seen of patients during life in the course of *pyæmia*, and after their death, that two classes of cases may be distinguished—namely, (1.) Cases which came on very soon after injury, or operation, or the puerperal state, and in which the fever temperature is extremely high from the first, and which die leaving little or no evidence of secondary lesion. (2.) Cases which are prolonged and essentially chronic, in which fever does not reach a high degree of temperature, but in which suppurative tendency is necessarily expressed in many organs, and in different parts of the same organ. There is in reality a *septicæmia*, or blood-poisoning, from the action of a specific poison proving fatal, as other specific fevers do; and another class of cases in which there is superadded to the *septicæmia* the suppurative tendency which expresses itself in multiple abscesses—true *pyæmia*. True pyæmia is generally preceded by suppuration somewhere, especially when that process has its seat in bone or tissues freely supplied with veins. One of the most frequent antecedents of pyæmia is osteo-myelitis. In this affection the pus accumulates in the interstices of the bone and the small venules, which, on account of their intimate connection with the osseous canals, are unable to contract, are constantly bathed with pus, and are thus, as it were, always ready to receive the poisonous material which may be developed by the decomposition of pus (*Lancet*, March 28, 1874). Mr. Hutchinson, on the other hand, is of opinion that those are the most definite forms of pyæmia, which are due to inflammation of the veins, and furnish the most impor-

tant group of cases. He considers the greatest heresy at present prevailing respecting pyæmia is the belief that it is possible for it to be caused by the introduction of a specific poison from without into the blood. Like septicæmia, he holds that pyæmia is produced by an inflammation of the patient's own tissues; and that what we call pyæmia in the more typical cases is due to *phlebitis*. It is a poisoning of blood by inflammation of veins—a *suppurative* and *gangrenous phlebitis* in which the veins become full of pus—in which *pyrolymph* adheres to the ulcerated lining membrane of the veins (*Brit. Med. Journ.*, April 17, 1875).

From experience, and by analogy, there are grounds for belief that a specific poison is the cause of pyæmia. The individual poison (as with other disease-poisons) has not yet been isolated; but by analogy its existence is inferred, because measles and other diseases have been communicated by means of inoculating the blood taken from persons affected with them, although the specific poison of measles has not itself been isolated. So the suppurative fever of pyæmia has been communicated in a similar manner, and is therefore believed to be developed from a specific cause. Professor Panum, and, after him, Dr. Richardson, have analysed the blood of patients dying from pyæmia. After boiling and subsequent evaporation to dryness, there remains a fixed and *non-volatile* substance, soluble in water and insoluble in alcohol, which can be compared in the intensity of its action to such poisons only as that of *serpents*, *curare*, and the vegetable alkaloids. Dr. Richardson has named the substance "septine," and with it he has caused the death of animals by inoculation or injection into veins. It seems clear that the poison forms in the body in connection with the wound or injury; that it is reproduced, multiplied, and intensified during the progress of the disease; that it passes into the blood; and that the blood and the juices of the patient can communicate by inoculation a similar disease, which operates with variable degrees of virulence in different instances. The results of *post-mortem* examinations of such cases have too often painfully demonstrated the virulence of the poison. In the examination of patients dying after ovariotomy of true pyæmia, I have found the fluid in the cavity of the peritoneum so acrid or poisonous as to irritate, to a painful extent, the skin of my hands. The evil effects also which follow the use of sponges in hospital practice, and the readiness with which the poison is thus carried from patient to patient, is a strong argument in favour of the local absorption of some poisonous material (ERICHSEN). "Pyæmia in a surgical hospital is a dreadful scourge. It may be conveyed by the surgeon, by dressers, by nurses, by anybody; and beyond a doubt it may be conveyed, as Professor Lister has shown, through the atmosphere" (LEISHMAN, *Brit. Med. Journ.*, April 17, 1875).

(c.) *Morbid Anatomy*.—The committee of the Pathological Society of London state that evidence to prove the existence of septic infection in the human subject must be looked for in the following directions:—(1.) The existence of a wound of just such a size as to make a fatal dose impossible. (2.) Evidence of infection from one patient to another. (3.) The presence in the blood of living organisms during life or immediately after death. The committee analysed 127 cases, and these were divided into eight groups, viz.:—(1.) Cases where there was no evidence that the secondary inflammations were due in any way to

embolism; and these again were subdivided into—(a.) Cases where visceral disease was absent; and, (b.) Cases where visceral abscesses were present, but where there was no evidence of thrombosis or of softening clot to be found: (2.) Cases where thrombosis had occurred, but where it was apparently due to the condition of the blood: (3.) Cases where there were thrombosis, softening of the thrombus, and secondary abscesses in other viscera, but not in the lungs: (4.) Cases where there were venous thrombosis, and softening of the thrombus, with secondary abscesses in the lungs, and in other viscera or tissues: (5.) Cases in which there were venous thrombosis and softening of clots, with embolic abscesses in the lungs only: (6.) Cases where there were thrombosis and softening of clots without any embolism: (7.) Cases of pyæmia secondary to acute necrosis in children: (8.) Cases of pyæmia secondary to endo- or myo-carditis. The blood had been examined in eighteen cases of pyæmia and septicæmia; and bacteria had been found in most, though not in all of the cases. Six forms of bacteria were met with,—(1.) Simple jointed filaments; (2.) ovoid bodies; (3.) large spherical bodies; (4.) dumb-bell bacteria; (5.) rod-shaped bacteria; (6.) protoplasmic masses, corresponding to large micrococci in size and appearance. This latter variety was met with in one case only; the dumb-bell variety was also only seen in one case. In five cases, the bacteria presented a great variety of forms; in three cases, the form of the bacteria was not noted; in the rest, there were globular forms occurring in pairs and chains, associated with very minute or ordinary rod-shaped bacteria. Thus so far as observations went, *there was no one form of bacterium peculiar to any one form of septic disease.* The white blood-corpuscles were found to be increased both relatively and absolutely, but in very varying degrees in different cases, and at different periods of the disease; the variations were not found to coincide with any change in the temperature, or with any definite symptoms. In one case, the lowest proportion followed the occurrence of repeated rigors. In a great number of cases micrococci were found in most of the organs, always situated in the blood-vessels, and chiefly in the capillaries, where they completely blocked the vessel, which in some instances appeared to be ruptured or partly absorbed, so that the micrococci were seen to be extruded for some distance into the contiguous tissues. In the large vessels they formed a layer of uniform thickness, lining the walls. Their distribution in the different organs was very unequal. Bacteria were found in the rod-shaped form in two cases only. In one case, they occurred in a glomerulus of the kidney; but in this case the epithelium of the kidney was much decomposed. In the other case, rod-shaped bodies were met with in the heart of a patient with myositis; in this case, the *post-mortem* examination was made very soon after death, and hence the presence of bacteria could not be due to decomposition. It was possible that some change in the chemical composition of the tissues of an organ might be necessary for the growth and multiplication of these different organisms, and that this change was induced by the pyæmic poison. If this were so, the presence of these organisms must be looked upon as an evidence of the existence rather than as a cause of pyæmia. In almost all cases changes were found in the blood-vessels. As a rule, the epithelioid

membrane was detached to a greater or less extent. In some places the detachment had taken place *en masse*. In a good many cases, the deeper layers of the intima were hypertrophied, producing a diminution in the lumen of the vessels, apparently due to obliterative endarteritis. Sometimes this hypertrophy was followed by degeneration, a condition which was especially seen in the glomeruli of the kidney. In some cases the external coat was hypertrophied, and this, again, was frequently degenerated like the intima. These changes were not found in the venules, although thrombi were very common in them. No special changes were found in the walls of the larger veins, except such as were associated with inflammation in the surrounding tissues. The thrombi in the veins and venules were mainly composed of leucocytes and fibrine, sometimes the entire channel being thus blocked; but in some cases, a passage was left patent on one side. Occasionally, minute abscesses were met with connected with, and apparently dependent upon, the thrombi. In the examination of the urine, no special chemical characteristics were noted, except such as are observed in other acute forms of pyrexia. The organic solids were slightly increased, the inorganic solids somewhat diminished, chiefly as regards the earthy phosphates; it was possible that the diminution in the inorganic constituents was due to the diminished ingestion of food, or even to the retention of some of the waste material in the blood to serve for the nutrition of the organisms. The potash, which, it had been suggested, might be increased in consequence of the destruction of the red blood-corpuscles, was found to be below the normal average.

The remains of the secondary or metastatic abscesses, as they have been called, are some of the most remarkable characteristics of *pyæmia*. They differ from ordinary purulent collections,—(1.) In the peculiar character of the pus they contain, which is usually a thin oily-looking pus, containing an immense number of granular cells, while “conspicuous by its absence” is the true nucleated pus-corpuscle. The fluid also sometimes forms a fine fibrinous coagulum after removal from the abscess. They contain bacteria. These collections form with great rapidity; and a few days commonly suffices for their attaining a large size. Decomposition of the body sets in very rapidly after death, and is indicated by the livid mottling of the skin, the change of colour, or dark red lines along the course of superficial veins or lymphatics, and the green hue of the lower part of the abdominal region. In fact it has already set in as a putrefactive process before death. The purulent deposits are generally isolated in the lungs and liver, but diffuse when they occur in the connective tissue. They generally show evidences of congestion of capillaries in a limited portion of tissue, as in a lobule of the liver or the lung. The chief seat of these secondary abscesses is in the thoracic viscera. The *pleuræ* are generally inflamed—sometimes adherent by deposit of recent loose yellow lymph, while serum, mixed with pus, or a deeply coloured turbid fluid, exists in the cavity. The lungs are generally much congested at the base posteriorly, and the tissue is generally friable in the congested parts. Sometimes they are completely consolidated in their lower halves, and emphysematous or œdematous in the remainder of their extent. The secondary abscesses

may be seen studded over their surface, or they may only be seen on section imbedded in their substance. They vary in size from that of a pin's head or hemp seed to that of a hen's egg. They are generally surrounded by a zone of condensed lung tissue, with hæmorrhagic infarction; and when the abscess is of considerable size, it may present a distinct lining membrane. The contents are generally pus-corpuscles more or less changed, fatty granules, disintegrated globules, and amorphous *debris*. In the smaller lesions the central part generally consists of a grey slough, and sometimes a grumous semi-fluid matter; and where hæmorrhages have existed, they may present the usual characters of lung apoplexy, appearing on section like a slice through damson cheese, or of a tawny yellow colour, from absorption of colouring matter. These deposits are most frequently found on the posterior surface of the lower lobe, or in the interlobular fissures. In the *liver* the abscesses are generally of a larger size than those in the lungs; but they have the same general characters as to form and position, with a zone of hæmorrhage and congestion similar to those in the lungs. But even in severe cases, where the lungs suffer most markedly, there may be no abscesses in the liver; and occasionally abscesses are met with in the liver when none exist in the lungs. When such is the case they are regarded as primary abscesses, and sometimes appear as a simple collection of pus, having a more or less branched arrangement. The *cortical substance of the kidneys* stands next in order of frequency as the site of secondary abscesses in pyæmia, and the abscesses present similar varieties to those in the lungs and liver. In the *spleen*, owing to its pulpy and diffuent condition in pyæmia, abscesses are not so readily recognised after death; and they are not so common as in other parts. Secondary abscesses of pyæmia are also frequent in the connective tissue, and in muscles. There they have no well-defined zone of congestion or limitation of fibrine, and hence the pus tends to infiltrate the limbs and trunk, forming immense diffuse collections of thin serous fluid, mixed with shreds of lymph and of dead areolar tissue. These collections are most common in the axillæ, the loins, the back, the iliac fossæ, the thigh, and the calf of the leg. The joints, especially the knee and shoulder joints, are also sometimes the site of thin, yellow, purulent-like liquid. *Phlebitis* may coexist, and so may *embolia*; but as they are not now considered essential features in the pathology of *pyæmia*, these lesions will be considered in their proper place among local diseases.

"Pyæmia thus occurs in several forms, which are characterised each by more or less peculiar symptoms, but most distinctly by the pathological appearances discovered *post-mortem*. There is that most widely known when you have septic embola, and scattered abscesses caused by them, and perhaps otherwise also. There is that where you have inflammation of the peritoneum and other serous cavities, including the synovial and endocardial. There is that where the mucous membranes are chiefly affected—the muco-enteritic. And, lastly, there is that where the only results found after death are—alteration of the blood, enlargement of the spleen, the liver, and degenerations of their most important tissues, with similar degenerations in other organs. It is this last which, often rapidly fatal, was described by Helm, and is now often called acute septicæmia" (MATTHEWS DUNCAN, *Brit. Med. Journ.*, Aug. 15, 1874, p. 215).

Symptoms.—The suppurative fever of pyæmia supervenes most frequently after injuries or operations in which bones are involved, or in which veins are especially implicated. The thin-walled veins in bones, the large distended intracranial sinuses in close proximity to the eyes and ear, the large hæmorrhoidal and prostatic vein plexuses, the open state of the uterine veins after delivery, are all favourable local conditions for promoting the development of *pyæmic* fever poison, while suppuration affects those parts. The specific fever of pyæmia—*septic intoxication*—generally commences suddenly with rigors and pyrexia, from the third to the fifth day after injury or operation. This is the acute form. Generally, in surgical cases the phenomena commence to be expressed between the second and fourth day after an operation or injury. The tissue round the source of the mischief becomes red and œdematous towards the third day; and *ichor* may exude from the injured part, mixed with bubbles of gas. Disintegration spreads with great rapidity from the seat of injury; and where the part is to-day simply *œdematous*, to-morrow it will be an *ichorous* infiltration; and so it proceeds till death occurs. In some cases it may be looked upon as a progressive gangrene of areolar tissue (the acute purulent œdema of Perigoff). If thrombi form in the veins, and the phenomena of embolism occur, the danger is increased, and the case is more complex; but the *septic* phenomena, in the first instance, are quite independent of embolism. A very high temperature and a more continued fever are characteristic of septicæmia. The pulse rises (generally above 120), and the intense burning heat communicated by the skin of the patient is well known to the hand of the experienced surgeon. On the evening of the fourth day after an injury, Bilroth records the temperature at $37.3^{\circ}\text{C.} = 99.5^{\circ}\text{Fahr.}$ In the night the œdematous infiltration commenced, and on the ensuing morning the temperature was $40.1^{\circ}\text{C.} = 105.8^{\circ}\text{Fahr.}$ On the eighth day the patient died, up to which time the temperature remained high, its lowest point being $39^{\circ}\text{C.} = 102.2^{\circ}\text{Fahr.}$ A rapid and considerable increase of temperature on the *third* or *fourth* day after a wound or operation, with delirium and a somnolent state, with an ichorous appearance of a wound, and œdematous infiltration around it, are certain signs of septicæmia. Albumen occurs in the urine in some cases, and sweats are common (BILROTH); the skin acquires a faint yellow colour, and the breath a hay-like smell. The highest temperature recorded by Professor S. Ringer is $107.4^{\circ}\text{Fahr.}$ (*MS. Notes*). But pyæmia may be chronic, and its symptoms may not commence for weeks after such diseases as *typhus*, *enteric fever*, *scarlet fever*, *rheumatism*, or *dysentery*. When pyæmia commences, the expression of the countenance is anxious, and the face pallid or flushed alternately; mental depression prevails to the extent of expecting or foreboding a fatal issue. The eyes lose their lustre, the features become pinched and careworn. The conjunctivæ and skin assume a "dusky," "sallow," "leadens," "icteric," or "yellowish" tinge, but much less bright than the golden hue of true jaundice. It is like yellow mixed with a dull leaden or ash-grey colour. A peculiar odour has been noticed as exhaled from the body during the progress of pyæmia (SAVORY), and the breath gives forth a peculiar, "sweetish," "heavy," "purulent," hay-like odour (BERARD). Respirations become more frequent—40, 50, or more in the minute (BRISTOWE). Bronchitis or pneumonia generally supervenes.

In this character it resembles measles, small-pox, and typhus fever. The *nervous phenomena* are essentially *typhoid* or *adynamic*; sleeplessness and restlessness night and day prevail; rigors, with depression succeed each other; and muttering delirium marks the severity of the attack. The pulse is generally always above 90; and if it rise high at the commencement, it so continues to the end—140, 160, and even 200 in the minute (BRISTOWE). Profuse perspirations occur, and mark the commencement of secondary abscesses. No period similar to a crisis can be defined, and no incubative stage can be fixed. The commencement is generally dated from the occurrence of the first rigor, or *increased temperature of skin*, thirst, and perspiration. A close observation of temperature *after* operations is all important. A nurse experienced in the use of the thermometer can very early detect the accession of pyæmia. Death generally ensues from exhaustion; and when the multiple centres are in process of evolution, the febrile phenomena are always increased—a distinct attack, with rigor, being excited and associated with each centre of development. Thermometrically, the commencement of *pyæmia* is very sharply defined, either by a sudden fall of temperature or a sudden and rapid rise—the first effect of

DIAGRAM OF TEMPERATURE IN A CASE OF PYÆMIA (Ringer).

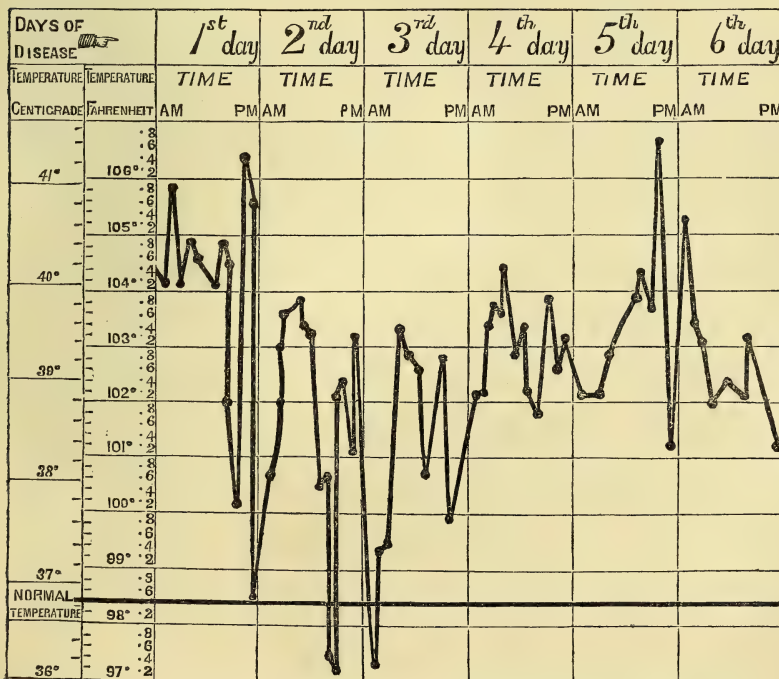


Fig 93.

the virulence of the poison. The elevation of temperature is, as a rule, rapid—complete in a few hours, or in half a day (4·5° or 6·3° Fahr.), or in the course of a day, and seldom lasting more than a day and a half. The temperature always rises above 104° Fahr., and generally exceeds

105·8° Fahr., and commonly reaches 107·6° Fahr. The mode of rising of temperature is as follows:—During the first twelve or fifteen hours, from morning till near midnight, the temperature rises about 1·8° to 2·7° Fahr.; but after midnight a more rapid rise takes place, so that in the morning the temperature is considerably increased; and if in the course of a febrile state already existing, the fever of pyæmia is at least 2·7° to 4·5° Fahr. higher than that febrile state would warrant, or than the daily maximum of the days which preceded the commencement of pyæmia. A further rise may persist on subsequent days; and generally rigors occur during the ascent of the temperature (WUNDERLICH). Dr. Ringer believes that the rise of temperature precedes the occurrence of the rigors; so that he has been able to predict the approach of rigor by noticing a commencing rise in the thermometer. Le Gros Clark also records that the temperature rises always shortly *before* the commencement of the rigor, and remains high for a varying length of time—generally about half an hour after the termination of the shivering (*Diagnosis of Visceral Lesions*, p. 74). As the rigors subside, the profuse sweating commences, and so they repeat themselves at intervals of every eight or twelve hours.

On reference to the diagram, it will be seen that the first sudden paroxysm of fever, as Wunderlich shows, takes an acuminate form; and after the temperature has reached the highest summit or maximum, it begins immediately, and just as rapidly (or even more rapidly than it rose), to fall again, so much so that in a few hours it may sink from 3·6° to 7·2° Fahr. Thus the temperature comes to be lower after the first paroxysm of fever in pyæmia than it was before, even in cases where fever existed before pyæmia set in. After the first paroxysm, the temperature seldom falls quite to normal, although, indeed, it comes very close to it. It generally falls as low as 100·4° or 101·3° Fahr. The low temperature which immediately succeeds the first rise rarely lasts longer or scarcely so long as half a day. It begins to rise again immediately, and this fresh rise, whether a rigor is associated with it or not, is generally just about as rapid as the first, but may not reach to so great a height. The temperature in pyæmia reaches a greater height in a shorter time than it does in any other disease; and the first pyæmic paroxysm may be distinguished from all forms of continued fever by the temperature quickly turning back and falling rapidly as soon as it has reached its maximum (RINGER, WUNDERLICH). “Brusque,” sudden rises of temperature, with corresponding falls, repeat themselves more or less frequently (twice or thrice daily) without any regular rhythm. This is well shown in Dr. Ringer’s diagram. Rapidity of movement downwards after the summit is reached is characteristic. Evening exacerbations of fever are distinctly marked, so that the ranges of temperature during the course of the disease are similar to those of extensive suppuration attended with fever. The temperature, however, in some of these cases is not very high; but in both forms of pyæmia—namely, those with multiple abscesses and those without—the type of the fever is intermittent during the day (RINGER, BILROTH)—*i.e.*, the temperature between the hours of greatest elevation varies considerably, and may even become normal, or below; and Professor S. Ringer writes that he has never met with any disease in which this occurs except *pyæmia*. It is

no doubt due to this circumstance being hitherto overlooked that such varied statements are current regarding the heat of the skin in pyæmia. In some cases, indeed, the temperature is never very high, nor the intermittent elevations very numerous, yet these are the kind of cases in which the internal organs are apt to be studded with secondary abscesses. The secondary abscess formation is among the most characteristic phenomena of the suppurative fever of *pyæmia*.

In the class of cases of pyæmia in which intense febrile phenomena occur, or of multiple abscesses, the temperature is very high, the intermittent elevations are numerous, and frequently there are no multiple centres of inflammation in visceral parts (RINGER). Though the exact chemical change in the composition of the blood has not been made out, yet, as lesions have been produced experimentally by the injection of filtered putrid fluids into the veins or intestinal canal, it may be fairly concluded that such cases are due to the absorption of putrid poisons in a gaseous or other fluid form, especially resulting from the decomposition of tissues. Tissue-change or metamorphoses in the febrile state may also establish sources of blood-poisoning (e.g., "Rheumatism"). Dr Parkes states how the amount of urea in a case of pyæmia, after amputation, rose from 3·1 for every lb. of body-weight to 4·26 grains. The weight of the body was 131 lbs. It fell to 110 lbs. in eleven days, and the temperature rose from 101° to 105·6° Fahr. (*On the Urine*, p. 178). Rapid wasting of the body is characteristic of the fever of pyæmia.

Diagnosis.—It is necessary to distinguish the suppurative fever of pyæmia from the following diseases; namely,—*meningitis*, *bronchitis*, *pneumonia*, *typhus*, *icterus*, *intermittent fever*, *gonorrhœal or urethral rheumatism*, *hectic fever*. It is the combination of symptoms presented by the suppurative fever of pyæmia which must distinguish it and prevent error. There are the occurrence of fever four or five days after operation or delivery; the dusky and icteric hue of the conjunctivæ and skin; the "heavy," "sweet," "purulent," "hay-like" odour of the breath; the inflammation, with effusion into and suppuration of joints; the formation of secondary abscesses in various parts of the body—following each other in natural sequence with great prostration. At the same time these symptoms are often so mixed up with those of the condition out of which pyæmia may arise, that the specific suppurative fever of *pyæmia* may be recognised with difficulty (BRISTOWE). It is to be distinguished from *erysipelas* by the fact that *erysipelas* is an eight-day fever, the poison of which acts most virulently on the skin and serous membranes; whereas the suppurative fever of pyæmia alters the blood, inducing secondary changes in the vascular pulp of many viscera, without affecting their serous coverings (BRAIDWOOD). Its difference from *pyogenic fever* has been noticed at page 797, *ante*.

Prognosis.—The disease is one of the most fatal; and the premonitory symptoms are therefore the ground for the gravest alarm, because recovery from a fully developed and unmistakable attack of pyæmia is almost quite hopeless (BRISTOWE). Some cases, however, undoubtedly get well, although a very large proportion die; and the chances of getting well are greater in proportion to the freedom from embolism, and from intense febrile disturbance, as indicated by temperature, and from the formation

of secondary abscesses. On the other hand, the prognosis is unfavourable in proportion to the rapidity with which the effects of the disease are developed. In acute and well-marked pyæmia we can hardly venture to admit a ray of hope; but, as a general rule, the longer the patient can struggle against the mischief, the more chance is there of his being ultimately rescued from the perils through which he has to pass (SAVORY). Youth is favourable to recovery; and as the fever varies in degree, just as other fevers do, the prognosis must not be always hopeless. As in *cholera*, so in *pyæmia*, "as long as there is life there is hope," if the patient's strength can be supported. One or two *pyæmic* rigors may be recovered from; but it is the repetition of the rigors, followed by profuse sweating, and attended by such extreme exhaustion, that is so fatal. The danger and rapidity of fatal termination in any given case will, *cæteris paribus*, depend on the rapidity of the recurrence of the rigors and their severity (ERICHSEN). Dr. Gillespie and Mr. Spence, Professor of Surgery in the University of Edinburgh, have recorded undoubted instances of recovery from cases of this kind; and Mr. Prescott Hewett recounts ten recoveries within his experience.

Treatment.—The aim should be to prevent pyæmia by dressing wounds and injuries by Lister's antiseptic methods. Wherever the antiseptic treatment of Lister is properly carried out, there is complete absence of pyæmia. The indications are,—(1.) To remove all those conditions which favour the degeneration, metamorphosis, or putrefaction of blood-clots or tissue in connection with wounds; (2.) To subdue the force of the circulating current, so as to prevent excitement during the long and slow metamorphosis of a clot when it has formed; (3.) To sustain the strength and allay the nervous irritability. Wine, brandy, rum, milk, strong animal soups, and nourishing diet, should be freely given from time to time, in small quantities. Opium should be given freely. Exposure of the patients to currents of fresh air—treatment in the open air, in fact—has been shown by Sir James Paget to be the most promising of success.

The use of disinfectants, and attention to ventilation, give support to the belief that the poison is one generated among cases of accumulated severe wounds and open sores. The patient ought to be isolated. *Condy's fluid*, *carbolic acid wash*, and *chloralum*, are each useful disinfectants. Preparations of *iron* are the most suitable tonics.

PUERPERAL FEVER.

LATIN EQ., *Febris Puerperarum*; FRENCH EQ., *Fèvre Puerpérale*; GERMAN EQ., *Puerperalfieber*—Syn., *Kindbettfieber*; ITALIAN EQ., *Febbre Puerperale*.

Definition.—"A continued fever, communicable by contagion, occurring in connection with childbirth, and often associated with extensive local lesions, especially of the uterine system."

Pathology.—(a.) *History.*—As a continued fever, this disease is at present classed, by the College of Physicians, with *typhus*, *enteric*, and other continued fevers; and certain important local lesions are specified as

attending, or following, it—namely, *peritonitis*, *effusions into serous and synovial cavities*, *phlebitis*, *diffuse suppuration*, as well as *exudations*, *congestions*, *purulent secretions*, and other changes of structure of the mucous lining of the alimentary canal and respiratory organs. It was not, however, intended by the College that this definition should explain the phenomena of the disease or indicate its nature. It is only for the purpose of identification, and includes all the forms of infective continued fevers which may attack the lying-in woman, whether they are peculiar to the puerperal state or not. Puerperal fever (like pyæmia and Bright's disease) has hitherto been a comprehensive term including a great variety of puerperal febrile affections; and it does not express any theory or doctrine as to their pathological nature.

The history of puerperal fevers can be traced in the literature of medical science back to the remote periods of antiquity; and though comparatively a modern introduction into nosological classification, it has retained a degree of exactness in its nature through all ages which can be claimed by few other diseases, which enables us to recognise it without difficulty in the descriptive works of many ancient authors. Thus, in the classical periods of antiquity we recognise its existence very much in the same forms as in the present day. The nosological distinctions were then based on symptoms only. Hippocrates clearly describes the disease, and characterises its early stage by the suppression of the *lochial discharge*, with fever and abdominal swelling. He also notes that erysipelas in a malignant and non-malignant form was prevalent—a statement of fact which leaves the impression that he had noticed a connection between atmospheric conditions and the occurrence of certain diseases which he could not explain. He recognised also several causes as capable of producing the series of symptoms which we call *puerperal fever*; and, that amongst these causes there is a clearly conceived notion of infective agency. For example, he recognised phlebitis, or inflammation of the cellular tissue of the limb; also, abortion; inflammation and ulceration of the uterus, and retention of placental structures—the symptoms following such retention being referred to the effects of decomposition—the *septic agency* (“*σήψις*”) of modern pathology. In 1681 Francois Mauriceau mentions the symptoms of puerperal phlebitis, which he attributes to suppression of the lochia; and gives in detail the most minute as well as the leading symptoms of nearly all the forms of puerperal fever with which we are acquainted. Cullen recognised the great mortality that characterised its occurrence in public institutions and hospitals. Willis, whose book was published 200 years ago, regarded puerperal fever as having some connection with a uterine wound; but he did not know the nature nor the anatomy of the uterine wound by the separation of the placenta; overlooking all other bruises, wounds, or injuries, such as contusions or lacerations of any part of the genital canal, of the lying-in woman as of any consequence in the results. Van Swieten, Willis, and many old authors on puerperal fever, regarded it as a wound fever, as Cruveilhier and Simpson have subsequently done; while Eisenmann (in 1837), like Kirtland, similarly named the disease “wound fever and childbed fever.” In 1788 Tenon described the state of the Hotel Dieu from so-called epidemics of puerperal fever, and for a

century before that time (1652-1783) there were constant notices of its prevalence. In 1750, at Paris, none who were seized with it recovered; and in one London hospital, in the space of two months thirty-two patients were affected with the disease, and all except one fell victims to it (GORDON'S *Treatise on the Puerperal Fever*, quoted by Dr. ROBERT LEE, *Brit. Med. Journ.*, March 6, 1875). In 1750, Plouteau described the disease as "*epidemic erysipelas of the peritoneum*." Dr. Alexander Gordon's treatise (1795) on the subject contains evidence that the fever depends on a particular poison which may be communicated from one person to another; that the occurrence of a number of cases of the disease is to be explained in that way, rather than by atmospheric or other conditions; and that the disease does not occur epidemically. He, too, considers it a disease which principally affects the peritoneum and the ovary. About the same time, at the end of last century, are the works of Denman, Lees, Hull, and Kirtland. The latter commences his essay on "*Childbed Fever*," by the question "What is properly *puerperal fever*?" for the writers of his time "include under this name—a thousand disorders." Since that period two opinions have been mainly held regarding the pathology of puerperal fever. Puzos, Levret, Hamilton, and White regard it as an idiopathic putrid fever. Armstrong, Macintosh, and Campbell maintained that it was essentially a local inflammation, of which the fever symptoms were merely a sympathetic consequence. Rayer, Le Gallois, and Cruveilhier endeavoured to show the analogy between the condition of a woman recently delivered and any other patient who was the subject of surgical injury; and to point out the similarity of the morbid affections to which they were both liable. Cruveilhier compared the uterus after delivery to the condition of an amputated stump, for its muscular walls were laid bare, and at the placental spot were the gaping orifices of divided vessels, through which deleterious matters may gain ready access to the general circulation. Sir J. Y. Simpson, in 1850, carried out still further the ideas of Cruveilhier in the memoir he wrote on the analogy between *puerperal* and *surgical fever*. But one of the best essays on puerperal fever during the present generation is that by the late Dr. Robert Ferguson, Professor of Midwifery at King's College, London; and still the weight of evidence as to the pathology of puerperal fever is in favour of the opinion originally expressed by him, namely:—that "The phenomena of puerperal fever originate in a vitiation of the fluids; and the various forms of puerperal fever depend on this one cause of vitiated blood, and are readily deducible from it." He pointed out that none of the local lesions are constant; and so none can be assumed as the essence of the malady; and as all may exist, they may each and all be regarded as the results of a cause capable of producing them singly or conjointly. Whether, therefore, the fever takes the form of *peritoneal*, *gastro-enteric*, *nervous*, or *complicated*, where several organs or tissues are attacked at once, a poison in the blood is the one source or cause.

But now the whole subject of puerperal fever must be read in the light of what is known regarding the pathology of *pyæmia*, as described in the previous pages; and from the records of that admirable discussion in the Obstetrical Society of London in the session of 1875, from which the following account is mainly taken. Mr. Spencer Wells

took the initiative in the debate, by an address on "The Relation of Puerperal Fever to the Infective Diseases." He drew up and illustrated certain propositions in the form of exhaustive questions, in order to elicit the opinions of the Fellows of the Society. These questions were as follow:—

(1.) Is there any form of continued fever, communicated by contagion or infection, and occurring in connection with childbirth, which is as distinctly caused by a special morbid poison, and as definite in its progress and the local lesions associated with it, as typhus or typhoid, scarlet fever, measles, or small-pox? (2.) May all forms of puerperal fever be referred to attacks of some infective continued fever—as scarlet fever or measles—occurring in connection with childbirth, on the one hand; or, on the other, to some form of surgical fever, or to erysipelas, caused by, or associated with, the changes in the uterus and neighbouring parts, following the process of childbirth? (3.) If all cases of contagious and infectious diseases which occur under other conditions than that of childbirth are set aside, does there remain any such disease as puerperal fever? (4.) Assuming that a form of continued fever—communicable by inoculation, contagion, or infection—does frequently occur in connection with childbirth, how can its spread in private and in hospital practice be most certainly prevented or checked? (5.) What relation have bacteria and allied organic forms to the pyæmic process in the puerperal state? (6.) What is the value of antiseptics in the prevention and curative treatment of puerperal fever?

Mr. Wells fixed attention on the facts; (1.) that, while the infective diseases were among the most numerous class of cases treated by the physician and general practitioner, the varied forms of traumatic fever (which, under the head of pyæmia) fell more frequently under the care of the operating surgeon than the obstetrician; and, (2.) that *puerperal fever* has its analogies in general surgical practice—placing puerperal fever almost exactly in the position in which surgeons place the several kinds of maladies from which their patients suffer after operations.

Thus the history of puerperal fever shows that many theories have been propounded as to its pathology—some setting forth that it is an essential or specific disease, *sui generis*; others, that it is simply a local inflammation of a sthenic type; or, that it is a phlegmasia of an asthenic type, of the nature of typhoid fever. Some maintain that it is in close alliance with hospital gangrene; others, that it is of the nature of erysipelas, or of uterine phlebitis. Clinical investigations, however, and recent pathological research, now teach that puerperal fever or fevers are but forms of blood-poisoning—pyæmia (*puerperal pyæmia* or *septicæmia*), similar to what is seen in others than puerperal patients.

(b.) *Causation and Propagation.*—The exceptional *constitutional* condition of the woman after delivery must be taken first into consideration. This condition is most graphically described by Dr. B. W. Richardson, as a peculiar one physiologically. The blood is in a peculiar condition. That colloidal fluid which, separated in a solid form, we call fibrine, is sometimes in excess—from 3 to 6, 7, or 8 parts in 1000. The blood at that time is thus in a trembling equilibrium, ready on the slightest

possible disturbance to precipitate it. There is also a diminution of salts in the blood—a condition also favourable to the precipitation of the colloid fibrine. The woman has also been supplying to the child a mass of blood from her own body, which has now ceased to be supplied; so that practically she is in the condition of a person who has lost a limb, a considerable portion of the body. She is also in a peculiar nervous condition. She has been supplying from her own potential energy, that element which has been shown in the movements of the fœtus; and since that has ceased with the birth of the child, she suffers from that nervous reaction which comes on when that motion is suddenly arrested. She is therefore in the exact condition for a series of changes necessarily febrile in character; and which renders her specially liable to impression about which we know very little (LEISHMAN); but with a peculiarity of constitution prone to and ready to receive poison, which intensifies in her system, and goes on to a disastrous issue (HUTCHINSON). The system, too, becomes loaded with effete material, during the process of involution of the uterus, the discharge of blood, the milk-process coming on—a state just treading on the verge of fever (*milk fever*), when at any moment the slightest excitement or the slightest noxious matter carried into the blood is ready to set up fever (BARNES). When these natural processes are interrupted, there is an arrest of that eliminative action which the involution process implies; and that in consequence of this arrest there is some accumulation in the body of those effete matters which ought to be expelled from the system (FARRE). It is a state in which the fluids are laden with old, effete, and useless materials; laden also with materials out of which the old uterus is removed, and a new one is to be built up. They are laden also with those elements out of which eventually the new secretion of milk is to be established. There is a *something* altering her blood (Dr. CHARLES WEST). The woman, too, is in some degree weakened by her confinement.

The causation of puerperal fever has also to do with the *local* condition present after delivery. The uterus and surrounding parts are bruised—perhaps lacerated, so that a wound or breach of surface exists, so that if a woman is sometimes poisoned by others, she not unfrequently poisons herself. For, if once the womb contains any fetid fluid, there can be no better arrangement for poisoning a human body, than that which exists in the womb after delivery. It is a large bag, a spongy bag, partly bruised, sometimes torn, with abundant lymphatics enlarged by the gravid process, and whose duty it is to absorb more speedily—with greater avidity—than at any other time. Thus if putrid fluid exist in the uterus it is absorbed by the lymphatics and so into the peritoneum, so that peritonitis is set up (TILT). The existence of a *specific* puerperal fever is not now entertained; and the term puerperal fever should have been “puerperal fevers”—a group of fevers connected with the act of childbirth. But some of these fevers are also to be recognised as occurring in other than puerperal patients, which are modified and rendered more formidable by the local and systemic peculiarities associated with parturition. Dr. Fordyce Barker, an eminent obstetric physician of New York, entertains a different view, and asserts the doctrine of a distinct and

specific puerperal fever (*On Puerperal Diseases*; also, *Brit. Med. Journ.*, July 24, 1875, p. 97).

The identity of symptoms between puerperal fever patients and surgical patients affected with pyæmia, and also the corresponding varieties in each case as to origin, severity, and *post-mortem* results, at once suggest identity of causation and propagation. The peculiar condition of the interior of the uterus at the placental spot affords a ready mode of ingress for what may be deleterious, as pointed out by Dr. Ferguson long ago; who held that all wounds and bruises in the genital canal might create diseased secretions from a traumatic surface, and by absorption might give rise to the phenomena of puerperal fever. In the able and exhaustive summary of the discussion at the Obstetric Society, given by Dr. W. O. Priestley, he gives the following as the outcome relative to causation:—

It may be stated that puerperal fevers, or *fevers which are attended by high temperatures* and are commonly associated with extensive local lesions, are due to the inception of a morbid poison which vitiates the blood, and which produces a great variety of symptoms in accordance with the nature and intensity of the virus, the amount of the dose absorbed, the state of the patient when attacked, and a diversity of other conditions. The ultimate nature and chemical composition of such poison are undetermined; but it has two sources:—(1.) The virus is formed in the patient's own body as the result of initial morbid changes in some special part, which infects eventually the rest of the system (*auto-genetic, auto-infective*); (2.) The virus is imported into the body and system of the patient by contamination from without (*hetero-genetic, hetero-infective*). In (1.) the septic matter is spontaneously formed within the system of the patient, as the result of disorganisation and death of tissue (necrobiosis of Virchow). Thus the retention of a portion of placenta, or of membranes, or of blood clots, may become a source of septic poisoning; or a contusion or laceration of the cervix, vagina, or perinæum may become the seat of unhealthy action, when organic substances in the process of decomposition are formed, which, on being absorbed, vitiate the blood. As to the mode of absorption obstetricians are not "of one mind in a house." That fluids do pass from the uterine cavity into the uterine sinuses is abundantly proved; and, as further evidence, Dr. Snow Beck states "that after the uterus has been injected by *perchloride of iron* to arrest *post-partum* hæmorrhage, the uterine sinuses have been found gorged with grumous blood containing an abundance of iron." But the lymphatics of the uterus are the chief agents in absorption, and not the veins (SAVAGE, TILT). An undue relaxation of the uterine walls after delivery is another local condition which favours the absorption of septic fluids (GRAILY HEWITT, SNOW BECK, and others). Hence the importance of thoroughly promoting the contraction of the uterus (and of keeping it contracted) upon the expulsion of the placenta. But even with empty vessels, Majendie proved absorption to be rapid in other parts of the body as well as in the uterus. To fetid lochiæ and their absorption much importance is to be attached, as factors producing the febrile affections of the puerperal state (Dr. TILT). Erysipelas, scarlatina, or other zymotic disease in a puerperal woman, may cause the lochiæ to become putrescent and virulent. The absorption of such putrid lochiæ then adds fresh peril to the case, and so

accounts for the serious character of the complication. And again, if absorption of decomposing or putrescent materials formed in the patient's own body is to be feared when there is no breach of continuity other than that caused by the separation of the placenta, how much greater may become the danger when any laceration has taken place in the genital canal which exposes a wounded surface to external agencies (see page 800, *ante*)? If this be pent up it may become a source of auto-infection. Medical men are not all equally alive to the greatness of this danger. The decomposition of an intra-uterine clot or portion of placenta is a frequent source of much mischief, particularly if there are any abrasions of the parturient passages. Local *metritis*, *cellulitis*, *peritonitis* of the worst type, tend to aggravate the general infection of the system, caused by the absorption of the decomposing material. In the practice of ovariectomy, Mr. Spencer Wells has become thoroughly conversant with this fact, and so he drains off from the cavity of the peritoneum the acrid fluid, which otherwise would inevitably poison the patient. Also, in surgical practice, a patient may be tolerant enough of decomposition which may be set up by a wound of his own body; but will be intolerant of poison conveyed to him or her from any other wound (Mr. CALLENDER). If this be true in surgery—as it is—*à fortiori*, it is true in obstetrics, and demonstrates how essential it is to the safety of the patient that the accoucheur should keep his fingers clean and free from all contamination. With regard to the hetero-genetic or hetero-infective forms of puerperal fever in which the virus is imported from without, the inquiry at once suggests itself as to,—(1.) Whence is the poison derived, which, introduced into the system, the patient is so intolerant of, and which has such potent results? (2.) Is it always one and the same poison, or are there several poisons capable of producing different diseases in non-puerperal patients, but provoking generally the same results when introduced into the body of a woman recently delivered? This latter inquiry is the most difficult of solution; and there are many conflicting views to be reconciled, especially regarding the relation of puerperal fever to the specific contagious fevers (already considered in this section). There can be no doubt that lying-in women are prone to be affected by every febrile disease to which they may be exposed. It matters not whether it be scarlet fever, measles, small-pox, or other of these contagious specific fevers, the affection is apt to break out with more than ordinary severity, and with graver complications, frequently ending in death. During pregnancy the accession of one of these fevers will often bring on abortion or premature labour; and even the protection usually afforded by a previous attack of scarlet fever breaks down under the trial of childbirth (Dr. BARNES). Nevertheless there is abundant evidence to show that a woman recently delivered may go safely through scarlet fever, with the disease so little modified as to retain its distinctive characters and be easily recognisable (SQUIRE, BRUNTON, BROWN). Cases have also been occasionally recorded in which scarlet fever passed through the system of the mother, who was herself protected by a previous attack, and infected her child *in utero* without serious consequences to the mother (CORDES of Geneva, and Dr. JACKSON, Notting Hill, London). On the other hand, a most valuable contribution to our knowledge of puerperal diseases was made (1871) by

Dr. J. Braxton Hicks, F.R.S., Physician Accoucheur and Lecturer on Midwifery, at Guy's Hospital. He came to the conclusion that there are "*states of the system engendered in some way or other in the puerperal woman by poisons (disease-poisons) which, in other conditions or states of the system, manifest their presence in a different mode.*" Trousseau expressed something similar, when he wrote, "*The lying-in female exhibits a peculiar morbid opportunity, and presents a remarkable pathological aptitude, for the malady.*" Evidence is also brought forward which shows that while puerperal fever exists in the mother, her children were affected with *scarlet fever*, either before or immediately after. One case is quoted where *erysipelas* succeeded in the nurse and infant, at least a week after the occurrence of puerperal fever in the mother; and another where the husband and nurse were severely attacked by sore throats a day or two after the puerperal woman was attacked by fatal puerperal fever. Such cases give support to the belief that a woman having puerperal fever derived from scarlet fever, or from *erysipelas*, can give to a non-puerperal person the original disease. But the difficulty exists, that a common origin may explain them all. Certain diseases, in entering the puerperal woman, may act upon her system in such a manner as to give evidence of its presence in a mode widely different from that which it presents ordinarily in the non-puerperal state. Dr. Hicks then shows,—(1.) That of those who are severely afflicted after delivery, in private practice, a large number owe their troubles to the poison of *scarlet fever*. (2.) That half of this number have most of the usual symptoms of ordinary scarlet fever—namely, the rash and sore throat. (3.) That with this ordinary appearance, secondary troubles are frequent, both local and general,—*e.g.*, *metritis*, *cellulitis*, *peritonitis*, *pycemia*. (4.) That the sore throat is slight in all cases, and the rash appears more simultaneously over the body than in ordinary cases of scarlet fever. (5.) That the influence of scarlet fever is shown in the puerperal female without necessarily the rash or sore throat; the patient having symptoms of blood-poisoning in a greater or less degree, producing, if the patient survive a sufficient time, secondary local and general lesions, such as are to be observed when the ordinary symptoms are present. (6.) That the disease almost always commences from the third to the fifth day after delivery. (7.) That though the woman may be exposed continuously for months before labour, still the disease does not generally manifest itself till the third to the fifth day after. The temperature in such cases has been noted at about 105·6° Fahr.

But if the evidence be true in reference to the exanthemata, it is even more forcible, and borne out by a greater concurrence of opinion, concerning the serious peril encountered by the puerperal woman when exposed to the poisons of *erysipelas*, *diphtheria*, and *typhus*. The most intimate alliance seems to be between puerperal fever and *erysipelas*. The diseases seem to be mutually interchangeable; so that if *erysipelas* prevail in a district, a third party may communicate, from a patient affected with *erysipelas*, puerperal fever to a woman recently delivered. There are also instances on record where husbands and nurses in attendance on women dead of puerperal fever, have been themselves attacked with *erysipelas*; and during an epidemic in the General Lying-in Hospital, London, the child born of every female in whom

puerperal fever proved fatal, died of erysipelas in a few hours (RIGBY'S *Midwifery*, p. 392). Instances also have not been rare where the medical man attending a puerperal fever patient has from her contracted erysipelas and died. Mr. Spencer Wells and others, in the debate on the subject at the Obstetrical Society in 1875, mentioned instances in which there could be no doubt that puerperal fever was produced by direct or indirect communication with erysipelas. The connection between the two has long been noticed; and observations have accumulated with such force and frequency as to constitute absolute proof of close affinity (W. O. PRIESTLEY, l. c.). Virchow also speaks of puerperal fever as *malignant puerperal erysipelas*. A connection has also been remarked between puerperal fever and diphtheria (Drs. PLAYFAIR, FORDYCE BARKER, LUSK, and others in America). As to typhus it is observed that if a woman contracts this fever after delivery, it may set up a virulent and fatal form of a so-called puerperal fever (Drs. WEST, BECK, and Sir HENRY MARSH). In private practice *erysipelas* causes fewer cases of puerperal fever than in hospital practice, where it is a most frequent cause of puerperal disturbance—so frequent that some pathologists regard *puerperal fever* as *erysipelas of the peritoneum*. By some also, *scarlet fever* and *erysipelas* have been considered as slight modifications of the same poison, and apparently interchangeable, and that certainly they have much affinity.

There is undoubtedly an intimate connection between the specific disease-poisons of the General Diseases comprehended in this section (A), and forms of puerperal fever; and hence it is easy to understand, as Dr. Hicks points out, how imperfect drainage, close rooms, and bad dwellings are sufficient, by continued action, to induce such a state of the constitution as tends to *effusions*, *suppurations*, *pyemia*, and *peritonitis*. Dr. Hicks has also noticed that the forms of puerperal fever are more frequent in newly built than in older houses; and the inmates of such houses are also more liable to attacks of *scarlet fever*, *enteric fever*, and *diphtheria*, probably from the drains of new houses being more liable to be deficient. Hence the broad conclusion must be drawn, that while the special poison of puerperal fever may originate in the person of the parturient woman, and be conveyed into her blood through the absorption of putrid coagula, portions of decomposing placenta, and decomposition of discharges, there are other modes, especially by the specific disease-poisons, through which the fever may be induced. Thus, also, the fever may be propagated through the intervention of a third party; or by the hand of the accoucheur carrying a putrid poison into the system during vaginal explorations. That it has thus resulted from poisonous material carried from the dissection of the dead body was abundantly and disastrously proven in the midwifery clinic of Vienna in 1847 and 1848. Any fluid matter in a state of putrefaction, communicated by linen, by a catheter, by a sponge, by small particles of placenta, or even by the ambient atmosphere impregnated with foul substances, may produce puerperal fever. Depressing mental conditions are also powerful predisposing causes. They tend to lower vitality, so that even the occurrence of a chill may develop febrile conditions; and the greater proclivity of unmarried women to puerperal fever ought now to be fully recognised.

Generally, thereafter, as to the causes of puerperal fever, they are

summed up as follows by Dr. W. O. Priestley, as President of the Obstetric Society :—

“There seems to be literal truth in the remark made by Dr. West, that ‘there is not one single solitary cause to which we can refer the symptoms of puerperal fever; that it occurs now from one cause, now from another.’ With equal truth, also, it may probably be asserted that there is not one puerperal fever, but several, arising from a diversity of causes. The term puerperal fever has, indeed, been somewhat loosely but habitually applied to all diseases of lying-in women which were attended by febrile symptoms and tended to a fatal termination. In attempting a more accurate pathology, it is well to bear in mind that fever is only a symptom of disease; and that we have to find out what is the underlying disease, of which the fever is but the indication. Perhaps the best practical classification of puerperal fevers which has been suggested, in accordance with our present knowledge, is:—(1.) The ephemeral forms, which depend on some transient cause; and the graver class, divided into, (2.) the auto-genetic or auto-infective form, and (3.) the hetero-genetic or hetero-infective, in which the poison is imported from some zymotic or other infective source, and the origin of which can be traced. Notwithstanding this classification, there will always be cases the origin of which it is difficult or almost impossible to detect. These must necessarily form a class by themselves, until further knowledge enables us to assign to them their proper place.

“A warm discussion has also taken place as to whether puerperal fever is ever really epidemic; that is, dependent on general atmospheric or other influences which pervade a community, as distinctive from the miasms of unhealthy hospitals, or the propagation of the disease from one patient to another by inoculation. But the annals of medicine afford undoubted evidence of the epidemic character of puerperal fever at definite epochs, and, if we turn to the histories by Hirsch, Dr. Robert Ferguson, and others, besides the speech of Dr. Fordyce Barker in this Society, we shall find that it occurred extensively in certain years among all classes of the community, in hospital and out of it, in town and country. In 1821, for example, it occurred as an epidemic in France, in Holland, in Scotland, and in London at the same time; while, further, it has been noticed that, when puerperal fever prevails epidemically in the human subject, the lower animals die in large numbers of diseases connected with parturition. Erysipelas is so undoubtedly and frequently epidemic in its character, has so commonly a spontaneous or idiopathic origin, apart from its association with traumatic injuries, that, if the mutual interchange between puerperal fever and erysipelas be admitted, the epidemic character of both follows as a matter of course. The influence of a vitiated atmosphere in overcrowded hospitals in producing an endemic form of the disease is only too well confirmed. Whenever a number of lying-in women are aggregated together, there is danger that a miasm may be generated which will develop puerperal fever; and it is by no means easy to define the amount of ventilation and isolation which are necessary to prevent these untoward consequences. Hence a feeling has gradually been growing up that, notwithstanding the economic and educational advantages of lying-in hospitals, poor women are more safely confined at their own homes. In the recent Congress on Maternity Hospitals at Brussels, M. Léfort stated that, in Paris outside the hospitals, the mortality had been one in 212, while in the Maternity Hospital it

had been one in twenty-nine. Fortunately, the statistics of maternity hospitals in this country are not so unfavourable; but the mortality in them even is much greater than when patients are delivered at their own homes. At King's College Hospital, we had a notable instance of the very serious results provoked in puerperal women by their being confined under the same roof with patients in general medical and surgical wards. The mortality in the lying-in wards gradually increased year by year, until at length it reached the fearful maximum of nearly one in every thirteen women delivered; and then it was determined to close the midwifery department altogether.

"As to the contagiousness of puerperal fever, there is no question that the disease may be propagated from one patient to another, although there may be some difference of opinion as to the way or means by which it is communicated, and some forms are regarded as less likely to be infectious than others. For a long time, the contagiousness of puerperal fever was disputed, and in Paris writers and practitioners remained unconvinced of its contagiousness long after it had been conceded elsewhere. In our day, the direct proofs of its contagious quality have accumulated to such an extent, that to disregard them in practice would amount, on the part of a medical practitioner, to a plain dereliction of duty. So far as some of the forms are concerned (however the attack may have originated), if it be but a single case, it may become the focus and centre of propagation to other women. And, of the several ways in which the subtle poison may be conveyed to the lying-in woman, none is more certainly proved than that it may be conveyed by the hands of the practitioner in making vaginal examinations. But this is not the only way. It may probably be communicated through the clothes, instruments, respiration, or even the secretions of the medical practitioner, midwife, or nurse. That it may be so spread, we have ample evidence, if we look back to the records of Gordon, Armstrong, Gooch, Ramsbotham, and others. Dr. Robertson, of Manchester, gives one of the strongest instances possible. In the space of one calendar month, a midwife attended twenty cases belonging to a lying-in charity; of these sixteen died of puerperal fever. Other midwives of the same charity attended 380 cases in the same district, and at the same time, and none of these were affected by the disease. For further evidence on this subject, I may refer to the works of Dr. Tyler Smith, Barnes, Routh, and Semmelweiss; and I can particularly recommend the perusal of the book published by Professor Wendell Holmes, one of the most charming and original of writers on general as well as on medical subjects. There is very cogent collateral evidence towards proof that the blood of a puerperal patient may be infected by other channels than by the genital canal. It is certain that puerperal fever may begin in a patient before her delivery. Dr. Barnes's statement that he has been conscious of absorbing poisonous and offensive emanations from patients with putrid discharges, and that he has suffered from shivering, diarrhea, and foul breath as the result, affords a very strong presumption that an attendant may go about charged with an infectious disease, and convey an atmosphere about him which shall be most deleterious to susceptible patients. A diseased child *in utero* may also infect the mother with a form of pyæmia, which may be transmitted on to other lying-in women."

But are all forms of puerperal disease attended by febrile disturbance equally infectious? Opinions seem to coincide pretty generally that some

forms are highly contagious and infectious, others are less so; and it is probable that those arising from extraneous sources are most contagious; those from poison generated in the patient's own tissues least so. Or, as Dr. Priestley puts it in another and more practical form,—“The most acute forms, running to a rapid termination, are most likely to be highly contagious; those which run a more chronic course, and are attended by secondary complications, are least so.” Nevertheless, as we do not yet know that even the chronic cases may not, in a minor degree, be infectious, it behoves us to exercise caution in attending these cases, to purify nurses who have been in charge; and, above all, to be careful not to convey any of the discharges to other lying-in patients.

As to the relation of *bacteria* to puerperal fever, it is exactly in the same position as that of *bacteria* to pyæmia (see pages 802 and 805, *ante*).

(c.) *Morbid Anatomy*.—In the lesions which follow puerperal fever, nothing can be set down as constant. Evidence of peritoneal inflammation is perhaps the most frequent; and hence sometimes the fever is described under the name of “*puerperal peritonitis*.” In such cases the inflammation is general over the peritoneum, and there is more or less sero-purulent effusion in the peritoneal sac, and any lymph is soft and bloody. It is particularly abundant in the pelvis, with more or less hyperæmia of the peritoneum and omentum. The uterus is large. Its inner surface covered with a muco-purulent, somewhat reddish secretion. In the uterine sinuses more or less pus-like fluid is found. There is no tendency to plastic forms of lymph. *Uterine phlebitis* is another frequent lesion. In such cases multiple abscesses are apt to be formed in the lungs; so that such cases may be regarded as cases of puerperal pyæmia. Such cases have generally thrombi or clots in the pelvic vessels; and are to be distinguished from other cases of *puerperal fever*, where the *uterus* is softened as to its tissue, as well as the *ovaries*, and where the *lymphatics* only are inflamed. In such cases there are no multiple abscesses.

Symptoms.—Increase of body-temperature, followed by rigors from the third to the fifth day after natural delivery (with or without hæmorrhage), are the earliest indications. Consequent upon these phenomena there is generally pain and tenderness on pressure over the whole surface of the abdomen, or limited to the lower half. The pulse at the same time increases in rapidity, ranging from 120 to 160, or more; at the same time it is an extremely feeble pulse. Respiration is rapid and short. Thirst is distressing; and not unfrequently nausea and vomiting occur. As a rule, the lochial discharge is suppressed or altered as to its character, and sometimes also the flow of milk ceases. Symptoms of great prostration and sinking are constant, as in pyæmia; and the countenance expresses great anguish, with sunken features and circumscribed lividness round the eyes. Diarrhœa is apt to occur, and the abdomen becomes gradually distended, while there is tympanitic resonance. Gradually the patient gets weaker and weaker; shortness of breath increases, with slight cough and expectoration; and she sinks after two or three weeks without more marked symptoms. In some cases the symptoms are those of acute mania.

Prognosis.—Puerperal fever cases so frequently end in death that Dr. William Hunter said—“Treat them in whatever way you will, at least three out of every four will die.” Dr. Farr, of the Registrar-General's

Office, estimates the mortality after childbirth in this country as 1 in 190 women delivered. Dr. J. Matthews Duncan, in his address at Norwich in 1874, regards this as much too low a rate, and, after careful computation, states the mortality, within four weeks after delivery, to be 1 in 120; while if the time be extended beyond four weeks, he believes that the number of puerperal deaths reaches as high as 1 in 100. This last calculation is so appalling, that we may vainly hope it is in some way exaggerated; but, taking the lowest estimate, 1 in 190, we find ample scope for grave reflection, and more especially as a large proportion of the deaths so computed arise from *puerperal fever*, or the group of *post-partum* diseases which have been somewhat vaguely designated by the name of "puerperal fever." Moreover, the number of ascertained deaths does not express all the mischief done. Many of those who survive the attack are greatly hindered in recovery, are exposed to much suffering, and possibly remain permanently enfeebled or maimed for life. The acute forms of illness which occur after delivery, and often end fatally, are especially cruel affections; attacking women, as they commonly do, during the very flower of their age, just at the dawn of fresh hopes, and at the time of their greatest usefulness in life. The anxiety of mind caused to the medical man when such cases occur in his practice, and the damage which, rightly or wrongly, results to his professional reputation, can only be appreciated by those who have had the painful experience. The mental anguish so engendered is reported, indeed, sometimes to have culminated in mental aberration. If reason were required, therefore, for the interest manifested in the late discussion on puerperal fever, it is sufficiently forthcoming in the above statistics, and more especially as in and out of the profession there is a growing impression that these deaths are in a great measure preventible.

Treatment.—Having regard to the varied sources whence *puerperal fever* may take its origin, prophylactic treatment ought to be the first consideration; and especially *the burning question* as to what ought to be the conduct of the medical practitioner in the face of perils of this kind (PRIESTLEY). Preventive measures are of vital importance, for such measures have been proven sufficient to arrest the development of the disease.

Dr. W. O. Priestley concludes his admirable address as follows:—

"The method, therefore, to be pursued for guarding lying-in women from noxious influences, consists of all those measures which prevent the formation of poisonous materials in her own system, and which secure her isolation from all contagion from without. In following out the first indication, it is necessary to provide, as much as may be practicable, for a woman encountering her confinement in the best possible condition of health, by impressing upon her the necessity of obedience to natural laws during her pregnancy. If complications occur during pregnancy, they must be combated as the circumstances will permit. Dr. Barnes has informed us that albuminuria at the end of pregnancy is extremely apt to go into puerperal fever. On these cases, therefore, a sedulous watch must be kept, the bowels must be carefully attended to, and other suitable treatment adopted. The process of parturition should be conducted with the view to the genital canal of the woman being exposed as little as may be possible to the effects of irritation, continued pressure, and laceration.

After the birth of the child, a full and perfect contraction of the uterus should be secured, by seeing that the organ is not emptied too suddenly, and that the hand follows the fundus down from above as its contents are expelled. Subsequently to delivery, the various known methods should be practised which promote the contraction of the walls of the womb and the diminution of the uterine cavity; and any clots forming, should be removed from time to time during the hour after removal of the placenta. It is superfluous to say that the placenta should be extracted in its entirety when possible; and great care should be taken not to leave any detached portions adherent to the uterine walls. The same precautions should be observed, when practicable, in cases of abortion. If any considerable laceration of the preinæum have taken place, the edges of the wound should immediately be united by sutures, not only for the purpose of restoring the perinæum, but also to prevent contamination of the wound by putrid discharges. In the case of other wounds in the vagina or cervix uteri, especial care should be taken to keep them clean by repeated injection, and to leave as small a raw surface exposed as may be practicable. In all cases where the lochia are in the least degree offensive, the vagina should be well injected with Condyl's fluid and water, or other innocuous disinfectant, twice in twenty-four hours, or oftener if necessary; and the injection may be carried into the uterine cavity, if it be much distended, and there is a suspicion that it harbours fœtid contents. The injection of the wound-cavity, however, should be conducted slowly, carefully, and without force. These uterine injections were practised long ago by William Harvey, and there is concurrent testimony in this and other countries of their marked utility in abating the symptoms, even when puerperal fever has apparently set in. Next in importance is to take care that there is no fœcal accumulation in the bowels, and to recollect that the existence of previous diarrhœa may be the indication that retained masses are lying in the intestines and producing irritation there. It must be within the experience of many how acute febrile symptoms, accompanied, it may be, by delirium or incipient mania, have passed away with a proper evacuation of the bowels. I may put it, in short, that, to promote recovery after parturition, every direct or reflex source of irritation should be removed, pure air and water ensured, absolute cleanliness observed; and perfect quietude, mental and physical, should be enjoined. Nursing the child should also be recommended when the mother's condition permits, as puerperal ailments have been observed more frequent in women who have not suckled their children. To secure the isolation of a lying-in woman from noxious influences which may be communicated to her from without may be difficult, and in some cases perhaps impossible; but as Mr. Callender has informed us how surgeons have learned to banish, for all practical purposes, those affections which may be termed septicæmic from surgical wards of hospitals, so I cannot help thinking that the accoucheur will in time succeed in preventing the like affections in puerperal women, so far, at least, as the hetero-genetic cases are concerned. We are all agreed as to the absolute necessity of preventing, directly or indirectly, any communication between cases of erysipelas and puerperal patients. We are agreed also as to the necessity of the midwifery practitioner avoiding all *post-mortem* examinations. His hands should avoid all contact with specific poisons or septic materials; and if perchance his fingers have touched anything suspicious, he should at once carefully and thoroughly disinfect them. The necropsies which seem most baneful are of those bodies which have recently died of erysipelas, peritonitis, zymotic disease, or any other inflammatory and febrile

affections; and in such instances the accoucheur should not even be present in the room when the dissection is made, as, though he decline to touch, yet his person and clothes may become infected by the poison. There may possibly be a doubt whether one who dissects bodies altered by decomposition or antiseptic injections, as they are commonly found in the dissecting-room of medical schools, is so liable to contaminate a puerperal patient as he who makes a necropsy soon after death. Semmelweis, however, believed that animal matter in a state of putrefaction would cause puerperal fever, and immediately lessened the mortality, during the fearful epidemic of puerperal fever which raged in the Vienna hospital in 1846 and 1847, by obliging all dissecting students to wash their hands in chlorine or chloride of lime before making vaginal examinations. Impressed by Semmelweis's views and opinions, I endeavoured, during my occupancy of the Chair of Obstetric Medicine at King's College, to prevent students frequenting the dissecting-room and *post-mortem* theatre from attending midwifery cases, and the result was a very small mortality in the outdoor maternity charity. This care about absolute cleanliness of the hands in attending labours should extend even to occasions when a medical man has had digitally to examine patients suffering from offensive discharges either proceeding from the surface or from the interior of the body. We have heard in this discussion that the discharges from cancer may produce fever in a lying-in woman; and this is probably true of other putrescent discharges. I have had grave anxiety, extending over some weeks, about a patient after her delivery; because I had gone straight from an instrumental case where labour had been so protracted that febrile symptoms had set in, and the passages had become inflamed, so as to pour out an irritating muco-purulent discharge. When a medical man has a bad case of puerperal fever in his own practice, or is required to see one in consultation, he should certainly not go direct to another midwifery patient without first changing his clothes, besides careful ablution of his hands; and he will be all the safer in his ministrations if he adopt some of those precautions mentioned by Dr. Swayne and others, such as using a Turkish bath, or the more radical measures for disinfection recommended by Dr. Wynn Williams.*

* No method, however, has been pointed out, or even alluded to, in the course of the discussion by which the septic poison could be got rid of should it have attached itself in any way to the body or dress of the accoucheur or nurse. We have more than one substance—one in particular—that will not tolerate the presence of putrid animal matter or septic poison: this drug is *iodine*, which, as soon as it is brought in contact with septic matter, is converted into two harmless substances—acid and carbon. Let any one who has been in attendance on any case, whether a parturient female or any other, where there is any putrid emanation, wash his hands in water into which he has poured *tincture of iodine*, and I will answer for it he has no septic matter under his finger-nails. Again, if he fancy that his clothes, hair, and skin are saturated with it, let him go into the water-closet (I mention that as the smallest room in the house), place a few scales of *iodine* on a plate and put a spirit-lamp under it, and he will soon find himself surrounded by a violet vapour, which will fall upon him in a shower of minute scales, from which he has only to protect his eyes. If he then carry with him any of the puerperal poison, my whole theory of the disease must be wrong. One thing I can assert, that in my own practice I have never had a case of fatal puerperal septicæmia since I have used *iodine* as an antiseptic, now more than twenty years ago. *Iodine* is equally efficacious in warding off septicæmia in other surgical diseases. I have injected solutions of septic poison under the skin of guinea-pigs, and produced death by septicæmia. I have also injected some of the same solutions, into which I had dropped a few drops of the *tincture of iodine*, without producing any ill effects. It is needless to observe that the prevention and the cure of this disease go hand in hand together. Should any septic poison be present in the puerperal woman, wash her out again and again with *solution of iodine* until the solution

"Many chemical substances have been recommended to be added to water for purifying the hands; *iodine, chlorine* and its compounds, *sulphurous acid, cyanide of potassium, carbolic acid*, and the *permanganates*. It matters not which agent is employed, so long as it is used carefully and efficiently; and if it be necessary to disinfect clothing, this is readily done by exposing it in an oven to a high temperature, for which many upholsterers have a suitable arrangement. With all deference to Dr. Matthews Duncan, I have no doubt that every medical practitioner who earnestly desires to promote the welfare of his patients and to keep himself blameless, should at once abstain from attending fresh cases of labour when one or more of those recently delivered by him have died with acute febrile symptoms, or are still so ill as to require his constant visits. When, indeed, there is the remotest suspicion that he may convey the poison in his own person, he should not incur the risk of disseminating it. How long he ought to absent himself from midwifery practice, is yet a moot point. Dr. Swayne thinks a medical man should seclude himself for a week only; others think a month hardly sufficient. The remarkable experience of Dr. Huntly of Yarrow-on-Tyne (*Brit. Med. Journ.*, Feb. 27, 1875, p. 271), led him to believe that he actually generated poison in his own body, as the result of some faulty process, which he communicated to his patients. Taking all the circumstances into consideration, I am disposed to recommend a week's seclusion after regular attendance has ceased on a single puerperal fever case. When a series of cases have occurred in the practice of any one medical man, he should absent himself from midwifery practice for a month at least. During either the short or the longer interval of seclusion, the means for disinfection should be fully carried out.

"Finally, I have to say a word or two as to the propriety of attending patients suffering from scarlet fever or other zymotic disease, and lying-in women at the same time. After what has been said in this debate, I cannot expect the same accord of opinion as on other parts of the ground I have gone over; but I would earnestly beg those who have as yet had so favourable an experience, when attending the two sets of patients conjointly, to ponder well what has been said by others on the reverse side of the question. It cannot be expected that men in general practice, who may be in charge of a scarlet fever or small-pox patient, shall at once relinquish all midwifery practice for the time being, because zymotic diseases are so prevalent that this would practically preclude their attending confinements altogether, or make their attendance on midwifery patients so irregular as to be unreliable. Nevertheless, in view of the dangers which have been indicated by various authorities, albeit their experience may seem to point to different conclusions, they are bound to exercise vigilance, lest perchance they slip into a pitfall unsuspected by them. In seeing an ordinary case of illness, a medical man rarely stays sufficiently long in the sick room to concentrate any considerable dose of contagium about him, and the after-exposure to fresh air in passing from house to house no doubt usefully dissipates any smaller quantity. I would suggest, however, to those treating infectious cases, and liable at the same time to be summoned to cases of midwifery, or who may be in attendance on puerperal women, that they should not stay long in the sick chamber; not undertake the duties of nurses, as Dr. Duncan puts it;

comes back the same colour as it was thrown up. The temperature of the patient will probably be 103° or 104°, and it will go down in a very short time to 98°. This I have witnessed since the present discussion began. (Dr. WYNN WILLIAMS, l.c.)

that they should see puerperal patients before seeing infectious cases, and never go from the infectious case direct to the lying-in room, without changing clothes and the most careful ablutions. Some doctors change their clothes and wash with a disinfectant whenever they have seen infectious cases. Whatever either will, or may, conduce to the wellbeing of patients, will, I am sure, not be regarded as either too irksome or troublesome by any member of our profession" (*Brit. Med. Journ.*, Jan. 8, 1876).

If *scarlet fever* can be prevented, the number of *puerperal fever* cases would be diminished one-half (HICKS); and every possible step ought to be taken to remove the pregnant female alike from the influence of *scarlet fever* and from *erysipelas*. Dr. Hicks observes, also, "with regard to the medical attendant, although I have found some fully aware of the risk of carrying *scarlet fever* and *erysipelas* to the woman in labour, yet I have met with not a few who either have not taken care, or have not been fully alive to the danger in which they are placing their patients." He considers it well to have three suits of clothes in use when attending such cases, keeping those out of use before a very warm fire, or hung out in the open air; the hands to be frequently washed and the nails short; and a good walk in the air should be taken before going to any other patient. Where there are partners, it is advisable for one to attend contagious cases, leaving the other to manage the midwifery. With regard to the nurse, the tendency of women's dress to retain the *fomites* of contagion must suggest their frequent purification by disinfectants, and the use of material capable of being boiled in the process of washing, and of otherwise being subjected to the action of disinfectants. With regard to the prevalence of the disease in hospitals, the wards ought to be closed entirely against the admission of patients, at regular intervals, during which the process of purification should go on. The ward to be purified should then be filled with *chlorine gas*, in a very condensed form, for forty-eight hours at least; the windows, doors, and fireplaces being kept shut. The floors and woodwork should be covered with chloride of lime, mixed with water, to the consistence of cream. The woodwork should then be painted, and the walls and ceilings washed with fresh lime; the blankets and sheets should also be washed and heated in a stove to a temperature of 120° to 130° Fahr. The hospital beds should be stuffed with straw; and as soon as the case is concluded, the straw ought to be burned, and the covering of the bed washed and heated like the blankets and sheets (COLLINS'S *Midwifery*, p. 388). Patients threatened with puerperal fever ought to be isolated.

With regard to medical treatment, *pure air*, *nutriment*, and *stimulants* are the agents best able to obviate the great depression which marks the commencement of puerperal fever. *Quinine* in large doses has been found useful after the bowels have been fully evacuated. It ought to be given till the physiological action of the drug is manifest, from the deafness and vertigo it produces. A form of *quinine* is now made soluble in warm water, which may be injected *hypodermically*. *Opium* is of great value. It has been given, to a large amount in the twenty-four hours, by Drs. Graves, Stokes, and others, since its great value became known. It must be given till incipient *narcotism* is produced; and the best evidence

of its good effects is seen when the respirations become sensibly diminished from the great frequency they attain in puerperal fever. If they can be reduced to fourteen or to twelve, with a pulse below 100, a subsidence of tenderness, and of tympanitis, the amount of opium may be gradually lessened and finally discontinued (CLARK). *Warburg's tincture* (suggested by Professor W. C. Maclean) has been found successful by Dr. Playfair in the treatment of puerperal fever.

CHAPTER VIII.

ON THE NATURE OF THE GENERAL DISEASES COMPREHENDED IN SECTION B.

THE diseases belonging to this section comprise, "*for the most part, disorders which are apt to invade different parts of the same body simultaneously or in succession.*" They are sometimes spoken of as constitutional diseases, and they often manifest a tendency to transmission by inheritance. They are typically represented by *rheumatism* and *gout*; also by those affections in which an alteration in the proportions or qualities of the elements of the blood is the obvious and prominent fact, such as *anæmia*, *chlorosis*, *general dropsy*; and, lastly, by those diseased states of the constitution which manifest themselves by such diseases as *syphilis*, *cancer*, *lupus*, *scrofula*, *rickets*, *cretinism*, *purpura*, *scurvy*, and *beriberi* (see Chap. II., p. 335, also p. 355, *ante*).

While the General Diseases included in Section A, which have been described, become developed in the human body under the direct influence of agents acting from *without*, the General Diseases which Section B includes may be said, by contrast, to become developed under the influence of agents generated *within* the body itself, through the continuous exercise of its functions in the daily course of nutrition, development, and growth. The original organisation of the human body may be of such a kind that the continuous exercise of its functions, in place of preserving the system in a healthy state, ultimately brings about diseases of various kinds, which (from this mode of origin and development) have received the name of CONSTITUTIONAL DISEASES. All of them are eventually attended with more or less local manifestations of disease, some of so fixed a character, and so strongly expressed, that the local lesions are sometimes looked upon (although erroneously) as constituting distinct diseases; such, for instance, as the joint or heart affection in *rheumatism*, or the bone affection in *rickets*. The local manifestations of those diseases which belong to the *constitutional* class are invariably the expression and result of a pre-existing unhealthy state of the system, without which no outward, extraneous, or accidental condition could call them into existence; although such conditions might determine (as a stimulus would) the local development, external manifestation, or expression of the constitutional disease. With reference to the term "*diathesis*" (a word often used in connection with descriptions of

these diseases), it is to be explained that there are certain states of the human body which physicians have called "morbid dispositions" (*diathesis morbosa*). The term *diathesis*, however, has been used extensively, and sometimes vaguely. By it pathologists have meant to designate an unknown, impalpable, undefined state of the human constitution, the existence of which is sometimes assumed, or is sometimes legitimately inferred by inductive reasoning; and which then is made to explain the characteristic tendency of some morbid constitutional state, which betrays itself by repeated local manifestations of morbid action (CRAIGIE). A *rheumatic diathesis*, a *gouty diathesis*, a *cancerous diathesis*, a *lithic acid diathesis*, are all thus spoken of. It is now generally understood to imply—(1.) The existence of *latent* conditions in the constitution of the body itself, necessary for the development of peculiar diseases; (2.) A tendency to the development of special and peculiar diseases during the course of the nutrition and other morphological changes between the solids and fluids of the body, and which are only influenced by the operations of agents from without, acting as stimuli or excitants to the morbid development. The CONSTITUTIONAL DISEASES are therefore all associated with what is termed a *cachectic* state (*cachexia*), or, in common language, "a bad habit of body." They are sporadic diseases, and are sometimes discovered to be hereditary. The lesions which attend them are observed to be rarely limited to one part or organ; and, before death ensues, several organs, tissues, or apparatus, not necessarily contiguous to each other, become diseased, and new materials of a heterologous nature may grow up in their substance.

In the local lesions are to be found those elements which mark the "anatomical characters" or "anatomical signs" of the several constitutional diseases. We do not yet know why one organ or texture should be either earlier or more constantly affected than another. It cannot be explained why *tubercle* selects at one time the *bronchial glands* for its main *nidus*, and the *lungs* at another; nor why *cancer* infests by preference the *mamma* and the *uterus*; nor why *rheumatism* affects the *white fibrous tissues*; nor why *rickets* affects the *bones*. The predilections of constitutional diseases to express themselves through lesions of certain organs rather than others are still unexplained.

The course of *constitutional diseases* is generally a prolonged one; the tendency to repeated attacks or paroxysms of morbid action exists throughout life; and the local expressions of disease may ultimately assume a persistent or chronic type, complicated with and complicating diseases of another kind, and mutually overlaying or aggravating each other. The primary implication of the constitution is, in the greater number of cases, sometimes demonstrable; but sometimes it can be stated only as a matter of fair induction that the constitution is affected through the blood and the relation of the nutritive processes to it. The constitutional diseases are not traceable to the extrinsic action of a *virus*, and have none of the properties attached to infection. They are thus apparently generated, developed, and sustained under the influence of an intrinsic blood poison, the result of perversion of the nutritive or assimilative functions of the individual, and are frequently determined by congenital constitution or hereditary tendency. "It seems strongly probable that each member of the group has its specific morbid principle in the blood,

uninterchangeable with the rest, just as any one *virus* is uninterchangeable with others; and that, further, there may exist for each constitutional disease its specific curative agent—an antidote for each poison. In the greater number of these diseases a more or less obvious disposition may be traced to symmetrical arrangement of the anatomical characters of the local lesions, whether these be external or internal" (WALSHE). External physiognomical differences may generally be recognised as distinguishing one man from another, and due to the peculiarities of his own constitution—"personal peculiarities." These are due to what have been called "*temperament*," combined with that character of the constitution which tends to the repeated expression of some form of ill-health, always in the same way, and to which the name of *diathesis* has been given. Such external differences between man and man—such *personal peculiarities*—are known to be transmitted from parent to child, and are then said to be due to *hereditary transmission*. The *tendency* to the expression of certain forms of disease being thus born with such children, is said to be due to *hereditary predisposition*, and this tendency may be strong and evident, or it may be but feebly and faintly marked. In the former instance it will become expressed in the midst of circumstances even the most favourable to health. Another remarkable feature in the pathology of constitutional diseases is this,—that the transmission of the hereditary tendency may fail to be expressed in the *children* of a family liable to diseases known to be so transmitted, and yet the tendency may appear in the grandchildren. The tendency is thus expressed in *alternate generations*—the law of "*atavism*," as it has been called. Thus failing to appear in one generation, the tendency may lie dormant, and at last burst forth "in some collateral branch of the family tree;" proving that *tendencies* not obviously expressed by the parent may nevertheless be transmitted by him. A person therefore cannot be considered free from the inheritance of constitutional maladies simply because his parents may not have suffered from any of them; and now it is admitted that, under at least *three generations*, the investigation of hereditary tendency is uncertain. When one only of the parents is the victim of constitutional disease, the tendency to similar constitutional disease is most obviously expressed in those children who most resemble that parent in physical conformation and appearance; and it has been observed that, when both parents suffer, the tendency will sometimes be expressed more often in the daughters of the family than in the sons, or more often in the sons than in the daughters.

It is especially to be noticed that a marked distinction ought to be made between those which are described as "CONSTITUTIONAL DISEASES" and those which are sometimes called "BLOOD DISEASES." The specific general diseases of Section A may emphatically be termed "blood diseases"—diseases in which a poison from without affects the blood and establishes a specific disease like small-pox; but the *constitutional diseases* are "*blood diseases and something more*." They not only exhibit an aptitude, possessed by those who suffer from them, to assume peculiar forms of morbid action, but their existence stamps upon every other morbid state with which they may be combined a most untoward effect. Exudations no longer proceed to healthy resolution. A chill, which otherwise in a healthy man would do no harm, is followed by rheumatic pains and swollen joints in

one whose constitution tends to rheumatism, which is a typical example of a *constitutional disease*; or the growth of tubercle may commence in the lungs of one whose constitution is tuberculous. A bruise is followed by inflammation and an unhealthy suppuration, which has been termed *scrofulous*, in those who are stamped with the diathesis of such a constitutional disease; while the influence of a constitution disposed to attacks of gout or rheumatism is very unfavourable for recovery from wounds, injuries, and other severe diseases. On the other hand, it is to be remembered that local inflammations depending on a constitutional cause are sometimes remarkably fugitive and transient. So much is this the case that an idiopathic inflammation, such as a pneumonia or bronchitis, lasts, as we know, for several days; but it occasionally happens that local affections having all the characters of inflammation will be suddenly established in persons labouring under constitutional *diathesis* or *cachexias*; and these inflammatory affections will run their course and terminate in as many hours as the others may take days—may appear at first even more formidable; but the very fact that they occur in a person suffering from rheumatism or gout enables one to give a more favourable prognosis of the immediate result than would perhaps be warrantable in the case of a person otherwise healthy. This may appear paradoxical; but it is known as a matter of experience that when visceral inflammations appear in the train of rheumatism, for example, they run a materially more favourable course, as a general rule, than if generated under some other influence. This fact it is necessary to bear in mind during the examination of all cases of constitutional diseases, for it may help to explain many apparent anomalies amongst the diseases of this class.

Very complex and dangerous forms of disease result when the General Diseases of Section A (acute specific diseases) are complicated with the diathesis of the constitutional diseases of the following Section B. When they co-exist, they exercise an unfavourable reciprocal influence upon each other.

The reader will observe and remember that the term “CONSTITUTIONAL,” applied to disease, as used in this Text-book, is not synonymous with GENERAL or EXTENSIVE, as used by some writers.

CHAPTER IX.

DETAILED DESCRIPTION OF THE GENERAL DISEASES COMPREHENDED IN

SECTION B.

ACUTE RHEUMATISM—Syn., RHEUMATIC FEVER.

LATIN Eq., *Rheumatismus Acutus*—Idem valet, *Febris Rheumatica* ;
FRENCH Eq., *Rheumatisme Articulaire Aigu* ; GERMAN Eq., *Acuter Gelenk-rheumatismus*—Syn., *Rheumatisches Fieber* ; ITALIAN Eq.,
Reumapira—Syn., *Febbre Reumatica*.

Definition.—*A specific febrile disorder, due to a morbid state of the system by constitutional development, and expressed by inflammation of a peculiar non-suppurative kind in the fibrous tissues about or surrounding the joints, especially in the white fibrous tissues—such, for instance, as the sheaths of the muscles and muscular fibres, tendons, aponeurosis, bursæ, capsular ligaments, periosteum, and pericardium. Many joints may be affected at the same time or in succession. The various local phenomena of the disease have a tendency to shift from part to part, the most remote from each other ; and the febrile state is accompanied by profuse acid excretions from the skin, by the separation, in some cases, of large quantities of uric and sulphuric acid through the kidneys, and by a highly fibrinous condition of the blood.*

Pathology.—(a.) *History.*—Various opinions have been entertained from time to time regarding the nature of *rheumatism*. The term has been used for ages to denote an affection with so much of a distinct character as to justify its being considered a special disease. The descriptions of Sauvages and Vogel are so brief and meagre that it is difficult to recognise in them the ordinary form of rheumatism as we daily see it. Sagar describes an acute and chronic form, and notices that the blood drawn exhibits a buffy crust ; and Sydenham is the first who gives a clear description of the disease from actual observation. It was believed to be of miasmatic origin ; but the most recent inquirers into its nature (BASHAM, GARROD, FULLER) show that it acknowledges no general external source, and it is not even yet demonstrated that any definite offending matter or poison pervades the system. It is presumed, however, that some morbid material is generated by and within the bodies of those in whom rheumatism is fully developed, or is not eliminated by them in the ordinary functions of life.

(b.) *Causation.*—The following circumstances point to the constitutional origin of rheumatism :—(1.) Its victims are apt to experience symptoms clearly denoting, at an early period of life, certain functional derangement, which leads to impairment of general health (FULLER, TODD, CHOMEL). (2.) Rheumatic patients are sensitive to atmospheric vicissitudes, prone to perspire, and their perspiration has a sour, disagreeable odour. Their urine, also, though usually clear when passed, not unfrequently deposits, on cooling, a red, brick-dust sediment of lithates and lactates. By such constitutional signs the “rheumatic diathesis” is indicated. (3.) The

heart of such persons is irritable, prone to take on inflammatory action, and its nutrition is apt to become perverted. (4.) A change of the mode of living has been observed to induce the rheumatic diathesis. (5.) The long continuance and frequent recurrence of symptoms, or paroxysms of severe disease, also point to a constitutional origin of the *materies morbi* of rheumatism. (6.) The disease is hereditary, and tends to run in families in the proportion of 29 per cent. of the cases (Dr. FULLER). (7.) Repeated previous attacks increase the predisposition to this disease. It was first suggested by Dr. Prout that all the phenomena of rheumatism are referable to the presence of lactic acid developed too freely or abundantly in the system—a suggestion adopted by Drs. Furnival and Todd, and subsequently by Drs. Headland, Fuller, and Mr. Spencer Wells. Before the starch of the food can be applied to the maintenance of animal heat, it has to be converted into lactic acid, which then combines with oxygen to form carbonic acid and water; and whatever tends to interfere with this normal series of changes from taking place may lead to the accumulation of lactic acid, or other allied acids, in the system. Dr. Fuller believes the poison of rheumatism to be identical with some natural excretion of the skin, and he grounds his belief chiefly on the following circumstances:—(1.) That when the skin's action is interfered with, especially in old people, pains or stiffness of a rheumatic character are generally the consequences; (2.) That the perfect development of the means which nature adopts to relieve these symptoms suggests a relationship between rheumatism and cutaneous excretion. No sooner is a person attacked by the disease than excessive perspiration is set up, as if with the view of getting rid of some peccant matter, and the secretion is most profuse at the very part where local inflammation is taking place. Dr. B. W. Richardson made some important experiments to try whether the theory admits of any direct demonstration which maintains that superabundance of lactic acid in the system induces pathological phenomena of the rheumatic type. He injected into the peritoneum of a healthy cat seven drachms of a solution of *lactic acid*, with two ounces of *distilled water*. Two hours after the operation the action of the heart became irregular. The animal was left for the night about six hours after the operation, and in the morning was found dead. The *post-mortem* inspection showed *no peritoneal mischief*, but the most marked *endocarditis* of the left cavities of the heart. The mitral valve, thickened and inflamed, was coated on its free borders with firm fibrinous deposit. The whole endocardial surface of the ventricle was intensely vascular. This experiment was afterwards repeated on two healthy dogs. The first dog died on the second day after the experiment, and the inspection revealed the most striking pathological signs of *endocarditis*. The tricuspid valve was inflamed, and swollen to twice its ordinary size. The aortic valve, swollen and inflamed, was coated on its free border with fibrinous beads. The endocardial surface was generally red from vascularity. The pericardium was dry and injected. As before, the *peritoneum escaped injury*. The joints were not affected, but there was distinct *scleritis* in the left eye. These experiments demonstrate that *endocarditis* may be physiologically produced by the injection into a serous cavity of lactic acid (*Medical Times and Gazette*, July 18, 1857). Similar experiments, with similar results, are reported by the late Dr. Brinton to

have been made about eleven years previously by Mr. Simon, of St. Thomas's Hospital (*Transactions of Med. Soc. of London*, Jan. 23, 1858, and reported in various journals). Whatever, therefore, the abnormal condition of the juices or fluids of the body may be which induces the rheumatic state, it is one which appears to be generated within the system as the result of faulty metamorphosis; some derangement of the nutritive and excretive processes—and that many agencies may conduce to the formation of such a poison, and to its retention within the system. Whatever the poison may be, if it be a poison, it does not seem to be absorbed from without. It is inbred, and not derived from extrinsic sources. The researches of Dr. Parkes lead to the belief that it may be some substance rich in sulphur. Nevertheless, although rheumatism is undoubtedly stamped with a peculiar and specific character, yet the material morbid matter is as yet undiscovered; and the exact conditions of the system under which the disease becomes developed are still undetermined. Both Dr. Craigie and Sir Thomas Watson hold that rheumatism implies inflammation of a peculiar and specific kind (see page 93, *ante*); and the latter goes a step farther, and writes that "Rheumatism is a blood disease,—that the circulating fluid carries with it a poisonous material which, by virtue of some mutual or elective affinity, falls upon the fibrous tissues in particular, visiting and quitting them with a variableness that resembles caprice, but is ruled, no doubt, by definite laws to us as yet unknown."

If we look to the course and antecedent phenomena of rheumatism, the disease may be observed to differ from all ordinary inflammations in the tendency it has to subside in one part and to appear in another—phenomena explicable by the laws of morbid poisons of constitutional origin, but which are opposed to all we know of the laws of ordinary inflammation occurring in a joint of a healthy person. An attack of acute rheumatism is a series of febrile paroxysms during which, while one joint gets well, another becomes affected; and this is especially likely to be the case in anæmic constitutions, or in those who are enfeebled from any cause. It is thus a frequent sequel to certain fevers, such as scarlet fever, and that peculiar malarious fever of indefinite duration prevalent in the Mediterranean, especially at Malta. Hence, also, cold and wet, by lowering the vitality of parts, greatly assist or promote that condition of the system which develops rheumatism, but are not the sole agents in its production. Any more express investigation into the remote or predisposing causes of rheumatism is extremely unsatisfactory. They are generally supposed to be identical with those causes which produce catarrh. Those, however, who refer catarrh to the vicissitudes of temperature only, also attribute rheumatism to this cause alone; but the returns of rheumatism occurring in the different commands of our army effectually blast such an hypothesis. It is not in the coldest climates that rheumatism is most prevalent, but at those seasons and in those climates remarkable for damp and variable weather. "Thus, we find in the mild and equable climate of the Mediterranean or the Mauritius the proportion of rheumatic affections even greater than in the inclement regions of Nova Scotia and Canada; and though some of the provinces of the Cape of Good Hope have occasionally been without rain for several

years, yet rheumatism is more frequent in that command than in the West Indies, where the condition of the atmosphere is as remarkably the reverse" (Sir A. TULLOCH). Exposure to wet, however, would appear to have much influence in causing that impairment of health which induces rheumatism, for we find the returns of the navy show a considerably larger proportionate number of attacks than in the army; the number *per thousand annual mean strength* attacked in the Mediterranean fleet being 63·9; in the West India and North American station, 69; and in the South American station 72·3. A predisposing cause of rheumatism lies not so much in the abstract degree of cold as in the range of atmospheric vicissitudes; and Dr. Haygarth has estimated that the number of persons attacked with rheumatism in summer is to those attacked in winter in the ratio of five to seven.

As to age—a very small number of children suffer from rheumatism. Out of seventy-three cases given by Chomel, two only were attacked under fifteen years—thirty-five for the first time between fifteen and thirty—twenty-two from thirty to forty—seven cases from forty-five to sixty—and seven cases after sixty. At whatever age, however, rheumatism occurs, one attack, while it indicates the existing diathesis, does not establish a predisposition to another; and although many are always martyrs to the affection, yet a well-developed rheumatic paroxysm and a complete recovery tend to establish (under attention to diet and the prevention of exposure to cold and wet) a greater or less protection for a time. But anything which exercises a prolonged depressing influence upon the system, especially if there is at the same time a hereditary tendency to the disease, may induce the development of a severe rheumatic affection. Errors in diet, suppression of the menses, scarlet fever, and such like disturbances to the functions of life have been assigned as causes. Men are believed to be more liable than women to rheumatism; but women, after menstruation has ceased, become more liable to rheumatism than males about the same age.

(c.) *Morbid Anatomy*.—The structures affected in *acute rheumatism* are, for the most part, those which are composed mainly of the white fibrous tissue, such as the *ligaments, fasciæ, aponeurosis, periosteum, perichondrium, tendons, bursæ, and serous membrane of the heart and brain*; but the *joints* and surrounding structures are the parts most frequently affected. *The heart, the kidneys, and the arteries* may all subsequently suffer. A red and injected state of the vessels of some of these structures is often the only evidence after death of the existence of any approach to inflammatory action, especially in the synovial membranes, the pericardium, and the membranes of the brain. This diffuse redness and injection of the vessels may terminate by resolution, or serum may be effused. *Serous inflammation* is extremely common, and is evinced by the swollen state of the bursæ and parts external to a joint, often by an evident fluctuation within the cavity of a joint; and should the patient die, the cavities of the arachnoid and of the pericardium may be found loaded with serum, the latter often to the extent of many ounces. One of the most frequent results of acute rheumatism is the tendency to thicken parts, and to cause opposing surfaces to adhere. The connective tissue surrounding the diseased articulation is often not only found thickened, but infiltrated with a loose coagulable

lymph. The tendinous sheaths and capsular ligaments sometimes exhibit the same alterations. After a time the new material becomes consolidated, and in this manner parts are bound down, and the motion of joints greatly and sometimes permanently impaired. The alterations of the synovial membrane are not the least curious of the changes which occur in rheumatic joints from the adhesive process. The tissue is not only often thickened, but villous processes, like the papillæ of the tongue of herbivorous animals (only soft and red), and dipping into the depressions around the neck of the bone, are occasionally formed, which are intractable even to long treatment, and often lead to destruction of the joint. The strongest evidence of the tendency to thickening, consolidation, and adhesion of parts may be seen in the immense effusions of lymph which take place in *rheumatic pericarditis*, sometimes covering the whole surface of the heart and pericardium with a layer of lymph half an inch in thickness, and whose irregular surface has been compared to a honeycomb, a calf's stomach, or to the external aspect of a pine-apple. It is rare that the exudative process in acute rheumatism advances to suppuration. It has been observed, however, sometimes in the muscles, but more commonly within the capsules of the joint (STOHL, CHOMEL, MOREAU, PIORRY, CRUVEILHIER, BOUILLAUD, MACLEOD). Ulceration of texture is by no means unusual in cases of rheumatism, sometimes perforating capsular membranes or destroying ligaments, but more frequently eroding cartilages and the ends of bones. A remarkable change also sometimes takes place in the bones of rheumatic joints when the cartilages have been lost. They become enlarged, and almost eburnified from increased ossific deposit, causing not only a change of form in the articular extremity, but presenting a mechanical obstacle to the motion of the joint. When the hip-joint is affected, the acetabulum becomes deeper and wider than natural, and the head of the femur, flattened and expanded, assumes something of the shape of a turnip.

Evidence of the abnormal state of the blood exists in—(1.) The symmetrical development of the local symptoms—a phenomenon which obtains in all disorders connected with a vitiated condition of the circulating fluid (BUDD); (2.) The constancy of premonitory fever, or of fever associated with the progressive development of the disease, with a large number of local symptoms, and lesions of internal organs, occurring simultaneously; (3.) A series of observations by Chomel (*Leçons de Clinique Médicale*) tend to show that the internal inflammations in rheumatism, like those of *small-pox*, *typhus*, *scarlatina*, and the like, are referable to an alteration of the animal fluids, more especially of the blood; (4.) The phenomena of metastasis, when it does occur, points to a morbid matter in the blood itself as an explanation of the occurrence (HOLLAND, FULLER). The most constant morbid condition of the blood in rheumatism is the abnormal relative proportions which become developed between the *fibrine* and the *saline* elements; the mean of the *fibrine* to the *saline* elements being in the relative proportion of 7·163 to 8·478 in 1,000. In normal blood the *fibrine* rarely exceeds 3 in 1,000.

The lesions from which the heart is apt to suffer in cases of rheumatism are—(1.) Inflammation of the pericardium or endocardium; (2.) Inflammation of the substance of the heart itself; (3.) Fibrinous vegetations on

the valves and on the lining membrane of the heart, independent of endocardial inflammation. These vegetations, like other exudations, are doubtless common in endocarditis, but they are not necessarily dependent on endocardial inflammation, and may take place without its occurrence (KIRKES, ORMEROD, FULLER). These fibrinous deposits seem to be more immediately connected with the abnormal condition of the circulating fluid, or alterations on the surface of the lining membrane of the vessels; for they are almost entirely confined to cases accompanied by acute and widespreading inflammation, and by conditions productive of unusual quantities of fibrine in the blood, which tend to impair its solubility. The statistics of Dr. Barclay, in the *Med.-Chir. Trans.*, Vol. XXXI., confirm these observations. But although endocardial inflammation may fail, in the first instance, to produce the fibrinous deposits on the valves, yet such endocardial attacks leave behind them a tendency to endocardial degeneration. The most common lesion which thus results is *fibroid degeneration of the valves*—a rheumatic lesion which may occur without any articular symptoms. It slowly advances from year to year, with a gradual but constant and more undoubted expression of symptoms; and ultimately the implication of important viscera (*e.g.*, fibroid degeneration of the kidneys, with œdema, and anasarca) ensues.

Symptoms and Varieties.—Rheumatism may be acute or chronic; but the proportion of cases of the latter is infinitely greater than of the former.

The varieties of rheumatism are as follow :—(1.) *Acute Rheumatism*; (2.) *Sub-acute Rheumatism*—a disorder analagous to the acute, but “of moderate intensity, with little or no febrile disturbance;” (3.) *Gonorrhœal Rheumatism*—an analogous affection associated with gonorrhœa, and no doubt influenced more or less by the characters imparted by the virus transmitted through the specific gonorrhœal matter; (4.) *Synovial Rheumatism*—a rheumatic affection in which an accumulation of non-purulent fluid occurs in the synovial sacs, and especially in those of the knee-joints; (5.) *Muscular Rheumatism*—pain in the muscular structures, increased by motion. The local varieties are—(a.) *Lumbago*; (b.) “*Stiff neck*”; (c.) *Chronic Rheumatism*—chronic pain, stiffness, and swelling of various joints. Continuous and relapsing cases ought to be distinguished.

Acute rheumatism expresses itself by a severe inflammation of the feet, of the hands, or of the larger joints, as the wrist, ankle, knee, hip, elbow, and shoulder-joint, or of one or more of these parts, and this is usually accompanied by severe inflammatory fever. Some joints are more prone than others to be attacked, such joints also being the most exposed, as the knees, the feet, the ankles, the wrist, and the hands are the parts most commonly implicated; next in order, perhaps, follow the elbows, and then the shoulders and the hips. The larger joints suffer more frequently than the smaller, and the small joints of the hands and fingers more commonly than those of the feet. Joints which have been injured, such as those which have been sprained, are more apt to suffer than others; so also those parts which are exercised violently, when that exercise is long continued. There is also a tendency to the symmetrical implication of joints (see p. 93, *ante*). Affection of a single joint may constitute the whole disease; but in some cases, either with or without the subsidence of the articular inflammation, the heart or pericardium, or the membranes of the

brain, may become the seat of rheumatic inflammation. In an attack of acute rheumatism the fever often precedes by twenty-four or forty-eight hours the inflammation of the joints; but this is not constant, for in some instances the local and general symptoms are contemporaneous, while in others the inflammation of the joints is established before the accession of the fever. The fever which attends acute rheumatism is well marked and striking, and symptoms of functional derangement present themselves long before its full development. Before actual fever is established, the patient feels "out of sorts," and unusually sensitive to atmospheric vicissitudes; he looks pale, with a sallow, unhealthy complexion, and a dull eye with yellowish conjunctivæ. The chilliness or shivering with which, in common with other acute fevers, rheumatism is ushered in, speedily passes away, and is followed by great heat of skin, copious but partial perspiration, almost invariably acid, reddening litmus paper, and of a disagreeable sour odour. It is a mistake to suppose that much perspiration is useless. It is nature's cure for the disease. It may be "wasting and enfeebling," as excessive perspiration always is, but it is highly sanative. If it does not occur, the pains are always more excessive; and if perspiration should unexpectedly cease, the constitutional symptoms become more severe. The *materies morbi* is obviously got rid of, to some extent, by the sweating, and the natural cure of the disease is mainly effected by these profuse sour-smelling perspirations, which are only useless when they are not of this characteristic sour description. They are then emphatically "useless, wasting, and enfeebling," and ought to be arrested.

The urine in acute rheumatism presents the strongest type of the so-called febrile urine (PARKES, l. c., p. 286). It is scanty, of high specific gravity, deeply pigmented, and deposits, on cooling, deep-coloured sediments of urates. It very much resembles peasoup. The water is lessened; the total solids augmented (chiefly by increase in urea and pigment); and the urea in most cases is very considerably augmented. Dr. Parkes has observed an excess of *one-fourth* or *one-fifth* over the physiological amount; and the amount is greater in equal periods during the day than during the night; and when the very spare diet of rheumatic patients is considered, the great disintegration of nitrogenous tissue is obvious,—represented as it is by a daily excess of from ten to twenty grammes of urea. The uric acid at the same time is somewhat increased; and there is usually a great quantity of water passed during the height of the disease, and at the commencement of improvement, or some days afterwards. The chlorides are often diminished, and sometimes disappear. Dr. Parkes has found a very great increase in the elimination of sulphuric acid, being sometimes double that passed during convalescence. The analyses of others have shown no increase. The pigment and extractives are always greatly increased. Albumen appears in some cases, generally small in amount and very transient. The kidneys suffer much less frequently and much less profoundly than in many other diseases where the system generally is so much out of order.

The pulse rises to 90, 100, and 110, and is large, full, and strong; and if the temperature rises above 104° Fahr., the chances of danger increase, and the case is a severe one. The tongue has usually a creamy-like covering, and is loaded with a white or yellowish-white mucus; the bowels

sluggish; the evacuations dark and offensive. There are many remarkable differences between the fever of rheumatism and the phenomena of continued fevers; for it runs no given course, is not marked by changes of the tongue, nor by any great depressing action; while delirium and headache are of rare occurrence. There are remarkable vacillations of temperature, occurring irregularly during the day; and a very high temperature is occasionally met with before death—in some cases as high as 109.6° and 110° (Dr. S. RINGER). But although the fever appears to show remarkable and extreme differences in different cases, so much so that no range of temperature can be represented as common to every case; yet, when a great number of cases of acute rheumatism are compared, certain typical ranges of temperature are more commonly met with than others (WUNDERLICH). In a large number of cases the temperature rises gradually during the commencement, and reaches its maximum height at the end of the first week, or at the beginning of the second. It then remains for a few days with, if any, very slight fluctuations; and afterwards, with care and good nursing, continues to descend with moderate morning remissions. Nothing like weekly cycles is to be noticed; and the thermometric indications seem not able to aid the diagnosis of inflammation of the internal organs, such as the heart. The fever never commences in the sudden manner of pneumonia, but with an insidiousness like that of enteric fever. Exceptional cases have been noted as high as 104° Fahr., or more, as early as the second to the fourth day; but by the middle or end of the first week of illness, the temperature may be moderate and continue so. The maximum reached in the evening hours may be 104° Fahr., and occurs generally between the fifth and ninth day, and it may remain high for from two to three days, ranging, as a daily maximum, from 101.48° to 103.1° Fahr. The character of the fever up till now may be continuous, exacerbating, or remittent. The defervescence may be by *lysis*; and it is exceptional to meet with a rapid downfall, such as would warrant the name of *crisis*; ten to twenty or more days may be taken in this process, the daily decrease being so trifling in amount. Repeated attacks and hereditary tendency protract convalescence. Hyperpyrexia is frequently observed in rheumatism. It commences by a sudden rise in temperature, with severe rigors and marked depressions. There is also peculiar restlessness and great thirst; respiration is hurried, and the countenance has an anxious expression; and pain disappears from the joints. Grave nervous symptoms succeed, such as delirium, proceeding to coma and speedy death; and sometimes jaundice, diarrhoea, or hæmorrhages. As the temperature continues to rise the skin ceases to perspire, and becomes harsh and dry. A temperature of 107.5° Fahr. has been considered as incompatible with recovery; but Dr. De Costa of Philadelphia has recorded a case of recovery after a temperature of 110° Fahr. (*Amer. Journ. of Medical Sciences*, Jan., 1875). The temperature may reach 110° , 111° , or nearly 112° Fahr. A very large proportion of the cases of hyperpyrexia have been in the young, and the attack has been a first attack. The signs of approaching hyperpyrexia, most conclusively shown by Dr. Henry Thompson (Physician to the Middlesex Hospital), are, in the main, "*nerve symptoms*;" and the prodromata of such are

especially "*delirium, restlessness, rebelliousness to treatment, and, it may be, disproportionate to pain; subsultus, tremors, disorderly respiration, stupor, somnolence, deafness, prostration, apathy, strangeness and waywardness of manner, unnatural anxiety*" and *depression of mind; even unnatural gaiety and garrulity*. The use of the thermometer now becomes all important. It, then, ought to be applied *many times* in the day. If there be delirium alone, or delirium with extreme restlessness, the temperature ought to be taken at least every two hours—unless the patient be really asleep. Delirium and restlessness however, as a rule, pass away; but, nevertheless, they are now and then true prophets of evil, and their warnings are never to be disregarded. In cases of this nature the temperature ought not to go beyond 106° Fahr. before administering the bath. In the presence of *stupor* the temperature ought to be taken every hour, or every half hour. The nerve symptoms invariably precede; and the hyperpyrexia, however it may intensify, or seem to intensify, those symptoms, invariably follows. The mischief originates in some profound and damaging impression upon the nervous system—an impression which gives rise at once to the phenomena of cerebral or cerebro-spinal derangement, and to those of exalted body heat—coincident effects of one common cause (*Brit. Med. Journ.*, Aug. 3, 1872).

Pericarditis or *endocarditis* may occur without elevating the temperature even one-tenth of a degree. *Aortic valve* insufficiency, however, generally causes greater disturbance than *mitral*. But, on the other hand, a marked increase of temperature for more than one day ought to rouse suspicion, and suggest inquiry into the condition of the heart and lungs. The symptoms which indicate cardiac mischief are pains or soreness all over the chest, increased on pressing between the intercostal spaces, and also on taking a deep breath. The patient is restless, his countenance anxious, and occasionally he coughs. On applying the stethoscope to the chest a murmur may be heard, loud and permanent, and evidently arising from some irregular contraction about the orifices of the heart, or from some affection of the valves. Many physicians believe that the exact pathological state of the pericardium can always be determined. Thus, if the inflammation be diffuse, we shall have a crackling sound, like that of new leather, the parts being dry; or if serum be effused, we shall find the heart moving in a larger space than usual. Again, if lymph be poured out, we shall have a rubbing sound; and, lastly, if pus be poured out, it will be determined not only by the greater space in which the heart moves, but by the sudden collapse and rapid sinking of the patient. (See "*Pericarditis*," &c.) The supervention of pneumonia is certain to raise the temperature. High temperatures usually correspond with a high pulse; but an evening temperature of 103° Fahr. may go with a pulse of 84 or 90, so that there is often a disproportion between the temperature and frequency of the pulse, (WOODMAN'S *Translation of Wunderlich*, p. 394, *et seq.*) One of the most remarkable and suggestive facts in regard to rheumatism is, that the fever and constitutional distress are not always commensurate with the extent and intensity of the local symptoms. The rheumatic inflammation of the joints is very frequently *preceded* by febrile disturbance; and sometimes the fever runs so high before any local symptoms have been established, that even cautious and intelligent practi-

RANGE OF TEMPERATURE IN A CASE OF ACUTE RHEUMATISM AFFECTING MANY JOINTS (Wunderlich).

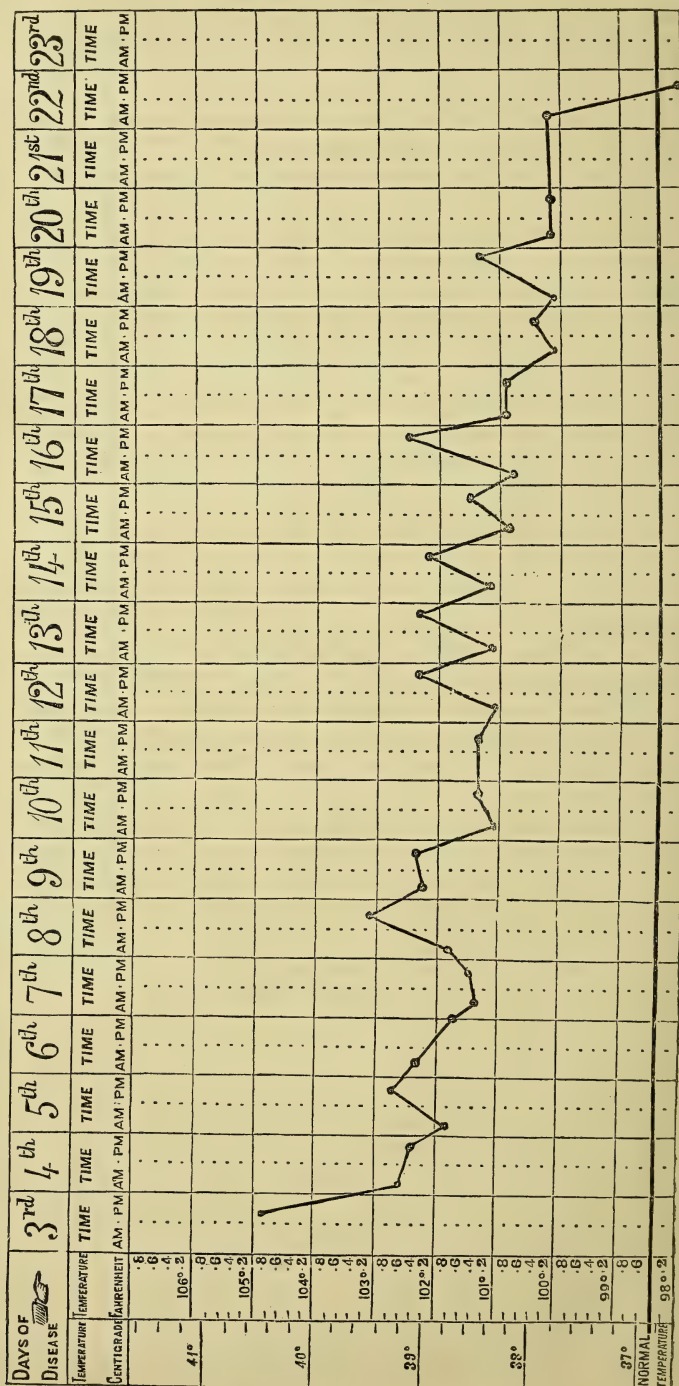


Fig. 94.

tioners may mistake the nature of the impending attack. Moreover, when febrile symptoms do thus precede the establishment of local inflammation, they are not only not increased by its occurrence, but they are very generally relieved, the pulse becoming calmer, the countenance less anxious, and the patient altogether easier (SYDENHAM, FULLER). These phenomena are associated with acutely painful, hot, red, and swollen joints. The *pain* is generally active and severe, although in a few cases it is latent—that is, the patient is at ease, unless the joint or limb be moved. At first the pains may wander capriciously from limb to limb, and produce more or less temporary stiffness. It has many degrees of intensity; in a few instances it is trifling, but more commonly it is atrocious and agonising; and, though generally constant, it is sometimes intermittent. It is always greatly augmented by pressure, so that the slightest touch—even the weight of the bed-clothes—is insupportable; it usually somewhat remits during the day, and is aggravated at night. The *heat of the inflamed joint* is constantly increased, the thermometer indicating a temperature of 100° to 105° Fahr., or even more. *Redness*, though not universally present, is nevertheless the rule, and the affected joint is surrounded by a rose-coloured blush, evanescent on the slightest pressure, yet returning on its removal. The *tumefaction* of the part is generally so considerable, that the shape of the hand, the ankle, or other joint, is completely changed. In affections of the knee the patella is often more or less displaced by effusion into the cavity of the joint; and this, together with the swelling of the external parts, obliterating all the markings of its healthy state, renders the knee misshapen and rounded. The surrounding skin becomes dry, tense, and shining—so much so, indeed, that experience alone enables us to predict that suppuration will not take place. The lesions of motion vary from mere stiffness to an entire binding down of the joint. In this manner the hip and shoulder may be so firmly fixed that the arm cannot be extended nor the leg raised. The knee and elbow-joints are generally semi-fixed, and cannot be straightened; while the fingers, if straightened, cannot be bent, or, if bent, cannot be straightened. When the joint is fixed, the muscles of the limb often become atrophied, sometimes partially so. Chronic rheumatism sometimes disappears in a few hours or in a few days; but, on the other hand, it may last many weeks, or months, or even years. Such are the general and local expressions of a diseased state of the system in acute rheumatism; and at the height of the disorder it is difficult to conceive a more complete picture of helplessness and suffering than that to which the patient is reduced. A strong and powerful man, generally unused to illness, lies on his back motionless, unable to raise his hand to wipe the drops which flow fast from his brow in the paroxysms of pain, or the mucus which irritates his nostrils. Indeed, he is so helpless that he is not only obliged to be fed, but to be assisted at every operation of nature. The sweat in which he lies drenched seems to bring him no relief; his position admits of no change; if he sleeps, his sleep is short, and he wakes up with an exacerbation of suffering which renders him fretful, impatient, and discontented with all around him.

Diagnosis.—The only diseases with which acute rheumatism, when attended with swelling and redness, can be confounded, are *erysipelas*, *gout*.

and *trichinous* (parasitic) disease (see pages 159-168, *ante*). Acute rheumatism is also often of difficult diagnosis when it attacks the intercostal spaces or diaphragm. It is then apt to be confounded with pleuritic pains, or with other affections of the chest. It may also be confounded with many neuralgic affections, as well as with pleuritic diseases.

Prognosis.—The number of deaths from acute rheumatism hardly exceeds one out of every thousand deaths from all causes; whence it is manifest that rheumatism is seldom fatal, although one of the most common, painful, and severe diseases of this country; and perhaps the number of unsuccessful cases hardly exceeds one or two per cent. But although this disease is rarely immediately fatal, yet a considerable number of persons ultimately die from diseases of the heart, apparently resulting from the continued action of the rheumatic poison. The tendency to heart complication is greater in proportion to the number of joints affected (BAMBEGER). The proportion of persons whose heart is thus affected probably varies according to the treatment, and other circumstances. Bouillaud estimates the number at more than one-half, or as 64 in 114 cases, and Dr. Macleod at one-fifth. The affection of the membranes of the brain is much more rare, but the proportionate number is not determined. Prognosis requires to be guarded as to the *results* of acute rheumatism, besides cardiac complication and its consequences. The malady sometimes proves fatal by an unexpected outbreak of overpowering nerve-symptoms, which are complicated with hyperpyrexia in many rapidly fatal results of acute rheumatism (see page 840, *ante*). Hyperpyrexia and nerve-symptoms are in reality one set of phenomena; and it is impossible *practically*—*i.e.*, *clinically*—to overrate the importance of hyperpyrexia. Delirium, coma, or symptoms of extreme nervous depression are unfavourable signs, especially when at the same time the temperature rises high. Meningeal symptoms in acute articular rheumatism may eventually lead to chronic cerebral disturbances, which may last for months or more,—appearing as a form of insanity without fever, characterised by depression, or as a pronounced melancholy, with more or less stupor. It may be followed or may alternate with states of excitement, and sometimes with choreic convulsions. The prognosis even in such cases is favourable; and recovery seems to result more rapidly and certainly if the joints become again attacked by acute rheumatism (GRIESINGER). Chronic rheumatism may also be a more or less permanent result of the acute attack. Grounds of prognosis have been also based upon indications derived from the condition of the urinary excreta:—*e.g.*, (1.) When the excretion of solid materials is large, other things being equal, the patient makes a rapid recovery; on the other hand, in lingering cases the excretion of solids is usually small. (2.) The urine being invariably scanty (12 to 14 ounces in the twenty-four hours), but generally from this cause only of high density, a guide to prognosis may be found, by diluting the urine to the normal amount in ounces, and then determining its specific gravity; and according as that is high or low, will the probable course of the disease be favourable or unfavourable (Dr. STEVENSON, in *Guy's Hospital Reports*, 1866). The state of other excreta by the urine has been already noted, and in proportion as these excreta are free, so will the case progress favourably.

The duration of acute rheumatism varies. In some cases both the fever and local pains are gone in three or four days; but in the majority of instances they continue till about the tenth to the fourteenth day, when the fever disappears and the pains begin to subside. Towards the close of the third week or the beginning of the fourth, the patient is recovered, and generally without injury to the joints affected. In almost all cases, however, the pain continues till after the fever is gone, and sometimes for a very long period afterwards. Under ordinary methods of treatment acute rheumatism continues from four to five weeks or six, and patients are generally able to leave an hospital about the end of the sixth week (FULLER). But the average duration of an uncomplicated attack may be reduced by judicious treatment from a month or five weeks to ten days or a fortnight. The patient, though recovered, is liable to relapse, and often suffers from it.

If acute rheumatism be severe and neglected, the patient may die in three or four days of the cardiac affection; but under proper treatment the complication seldom continues beyond a week. If this attack be altogether neglected, and the patient survive, the pericardial surfaces either become adherent, or the valves of the heart become permanently diseased. The ulterior effects of these lesions are dropsies, asthma, or affections of the lungs, which baffle all the resources of our art, and ultimately they are to be classed among the most fatal maladies incident to humanity.

The heart affections embrace both the immediate and the subsequent dangers in an attack of rheumatism. The occurrence of the heart affection was at one time considered as due to a retrocession of the disease from external parts, and its consequent transfer to the membranes of the heart. But endocardial or exocardial inflammation may occur as the first, and be for some time the only local symptom of the disease (WATSON, HOPE, HACHE, GRAVES, TAYLOR, FULLER, and others). *Acute rheumatism* and *pericarditis* may also co-exist without any articular affection (HACHE, GRAVES, TAYLOR); and the cardiac complication sometimes even *precedes* by *several days* the access of articular redness and swelling; and even in cases where cardiac affection does not take place until after inflammation of the joints has been set up, it is rarely preceded or accompanied by subsidence of the previously existing articular inflammation. In other words, in the great majority of cases it has been shown that no connection can be traced between the two sets of actions, beyond their origin in one common source of mischief—in one poison which excites inflammation now at one spot and now at another. At one time the disease attacks several joints simultaneously, or in succession, and then the investing or lining membrane of the heart; at another time, reversing the order of attack, it excites inflammation first of the heart, and then of the articular structures. Hence, although rheumatic inflammation of the heart may possibly be connected in some rare instances with the sudden subsidence of articular inflammation and the transfer of irritation from the external parts, it must be regarded in most instances as a mere coincidence, and as an extension of the local manifestations of the disease (FULLER). As a general rule, like the severe cases of articular rheumatism, the occurrence of cardiac complication may be expected in cases remarkable for the severity of their general symptoms. But from the nature of this disease

all cases are liable; and the cardiac symptoms may for a time be the only ones. Hence has arisen the apparent difference of opinion as to which are the cases peculiarly prone to heart disease. Dr. Latham and Dr. Watson state that "pericarditis is not more to be looked for when the disease is severe than when it is mild;" but, according to the experience of Dr. Fuller, it is inconsistent to believe that it often occurs in cases which are not characterised by active symptoms of disease. "Whenever I have met it," he writes, "even though the articular inflammation may have been slight or evanescent, the febrile disturbance has always been severe, and accompanied by profuse and sour-smelling perspiration." His experience expressed numerically stands as follows:—"That whereas pericarditis occurs *once* in about every 6·3 patients suffering from *acute* rheumatism, it does not accompany above one in every 66·5 cases of the sub-acute form;" and his experience in this respect is consistent with that of M. Bouillaud, Drs. Macleod, William Budd, and Copland. In many instances the heart remains unaffected throughout the attack; and though it does sometimes suffer even in the milder cases, it is most commonly damaged in those instances which are marked by unusual severity of their general symptoms, by the number and intensity of the articular inflammations, and by the rapidity and frequency of their migration. It is found in youth, in women rather than in men, in those persons who have been weakened by illness, or by large and repeated bleedings, and in those peculiar states of the system marked by a deficiency of red globules in the blood, when the heart's irritability is much increased, and palpitation is readily induced. These are the cases in which cardiac inflammation is most liable to arise during an attack of rheumatism.

Rheumatism is often protracted and rendered complex by other lesions besides those of the heart. The different muscles of the body, their sheaths and tendons (in addition to the joints, or independent of them), are often the seat of rheumatism, and there are few structures of this kind which entirely escape. The scalp, for instance, is often affected. The muscles of the eye are occasionally so: Stohl quotes one case in which the patient squinted while the disease lasted. Rheumatism of the face is by no means unfrequent, and the muscles of the larynx are occasionally affected, causing aphonia. Everybody is familiar with the rheumatic affection termed "crick in the neck," "*stiff-neck*;" at the same time it may affect the articulations of the clavicle and the intercostal muscles. Rheumatism of the abdominal muscles is by no means rare, the principal pain being at their insertions into the *crista* of the ilium. *Lumbago* is well known as a rheumatic affection of the sheaths of the fleshy mass of the lumbar muscles, on one or on both sides of the loins, extending often to the ligaments of the sacrum. The pain is increased by every movement of the back, or pressure on the muscles implicated. The insertion of the *tendo Achillis* into the *os calcis* is another frequent seat of rheumatism; but no parts are more often or more painfully affected than the tendinous structure of the soles of the feet. When the neurilemma of the sciatic nerve is affected, such rheumatism is one form of *sciatica*. *Pleurodynia* designates the rheumatism of the intercostal muscles, or the fibrous sheaths which incloses them. These forms of muscular rheumatism are seldom accompanied by any swelling or other external symptom.

When catarrhal affections are prevalent, inflammation of the lungs or their investing membranes may be expected. Acute inflammation of the sclerotic coat of the eye is not uncommon, and appears to be most of all liable to occur in cases where the tendency to gout prevails as well as the rheumatic condition. Rheumatic inflammation of the investing membranes of the brain is one of the most frightful but happily rare complications. Cases of active maniacal delirium, sympathetic and symptomatic, of cardiac or pulmonary disease, or of the general vitiated condition of the circulating fluid, have also been recorded.

In conjectures regarding the reason why constant lesions of particular textures occur in rheumatism, "it is worthy of note that the textures most commonly implicated are all examples of the albuminous and gelatinous tissues, from the decomposition of which, in the wear and tear of the body, are formed those secondary organic compounds, the lithic and lactic acids, with which gout and rheumatism are intimately connected." The increase of fibrine, as Mr. John Simon has conclusively indicated, might also be added. It may be for this cause, therefore, that the rheumatic poison has a special affinity for the fibrous and fibro-serous textures throughout the body, and fixes more especially upon those which are in any way subject to irritation. According to the intensity of the febrile disturbance, *as measured by the thermometer*, so, *cæteris paribus*, would appear to be the liability to inflammation, whether of the joints, the heart, or any other part of the body; and the increase of temperature above 98.4° Fahr. will give a tolerably accurate measure of the amount of the poison present in the system, and of the patient's susceptibility to its influence. The number and intensity of the articular inflammations, and the proneness they exhibit to shift their quarters, serve also as guides to the probability of heart or other internal affections; and the extreme liability to cardiac inflammation, engendered by the repression or rapid subsidence of the articular inflammation, is explicable by the greater quantity of the poison which is thus suddenly thrown into the blood's current (FULLER).

Cases of a *malignant rheumatic character* reveal themselves by rigors, with intense fever, severe nervous symptoms, jaundice, hæmorrhages, diarrhœa, and enlargement of the spleen; and death occurs with enormous elevation of temperature— 109.4° to 111.2° Fahr. Wunderlich considers that such cases ought to rank with cases of *spontaneous pyæmia*, or *acute yellow atrophy of the liver*. Death often occurs when the temperature of rheumatism reaches 105° Fahr. (RINGER, E. L. FOX, WOODMAN). It is the hyperpyrexia which plays the principal part in the destruction of life. A persistent temperature above 102° for ten or twelve days will generally be followed by protracted convalescence. In most fatal cases there is a rapid rise of temperature; and the patient passes from a condition of apparent safety to one of extreme peril in the course of a few hours. Hence the great necessity of good nursing and of carefully watching the readings of the clinical thermometer. The recoveries are few when the temperature has reached 106° Fahr., except under the influence of the external application of cold by the bath (see p. 841, *ante*).

"But, there are also many actors at work in rheumatism, all conspiring together to compass the death of the patient. If the aggregate of these

factors be overwhelmingly strong, there is an end of the matter, and the patient dies; but let the powers of life and death be more evenly adjusted, then the removal of any single factor (say the temperature) turns the scale in favour of life; or, to use a more homely metaphor, takes off the last ounce that is breaking the camel's back. You cannot, then, conclude with certainty that in fatal cases the hyperpyrexia has done all, or nearly all, the work of destruction, even if you regard the processes of refrigeration as resulting simply in the reduction of heat; still less are we at liberty to draw this conclusion if we change the point of view altogether, and presume (as not unreasonably we may) that our refrigerating agents strike home at the root of the evil, and directly antagonise the first deleterious impression on the nervous system by producing a salutary counter-impression, which controls alike the hyperpyrexia and all the associated elements of death. Nevertheless, it is believed that a continuous body heat, ranging from 108.6° Fahr. or to 112° Fahr., is absolutely incompatible with life;—with the exceptions already noticed (pages 284-5, *ante*). In the hyperpyrexia of rheumatism the temperature rapidly runs up to these heights, and if unsubdued inevitably ends in death. Practically, therefore, *heat* is the main destructive element. There are also certain combinations of symptoms which are held to be of peculiar significance. A dry unspiring skin; a profuse and intensely red miliary rash; an abatement of the pain and swelling in the joints, or their total disappearance; diarrhoea, and a copious flow of pale alkaline or neutral urine, have all been observed to accompany or to precede the paroxysms. They may even precede the nervous prodromata. An unexplained rise either in pulse or temperature would certainly be a suspicious symptom" (Dr. HENRY THOMPSON, *Brit. Med. Journ.*, Aug. 3, 1872).

There is usually some difficulty in fixing exactly the date at which the disease may be said to have yielded, or to have disappeared. Cessation from pain has sometimes been taken as the sole indication. But a morbid state of the system in rheumatism may exist after the joints are free from active pain, and even without the presence of any cardiac affection, which requires but the smallest exciting cause to bring back the articular or heart affection in all its pristine intensity. Patients in such a state, though free from pain at the time, cannot be considered rid of the disease. Dr. Garrod looks upon the following conditions, therefore, as indicating real freedom from the disease:—(1.) Absence of pain and any acute tenderness of the joints; (2.) Freedom from any active cardiac affection; (3.) Absence of marked thirst, with returning appetite; (4.) No unusual rapidity or hardness of the pulse; and, I would add, a normal temperature.

Treatment.—It seldom if ever happens that the cure of rheumatism can be entrusted to any single remedy. The constitutional nature of the disease, as already indicated, points to various methods and combinations of treatment as most likely to lead to a successful issue. A successful issue in a case of rheumatism means that the attack should be passed through without any permanent organic mischief being left behind. The treatment of rheumatism must provide, (1.) for the acute attack; (2.) for the management of the system as regards those conditions of the life of the patient which may have been found to contribute to the constitutional development of the disease (see Causation, p. 833, *ante*). In the

acute attack the patient must be made—(1.) comfortable, being protected from pain and exposure to cold air, especially as regards the joints; (2.) to encourage free excretion from the skin especially, and from the kidneys. The general management of the patient is therefore all important. No linen must touch the skin. A flannel shirt must be worn next its surface, and the patient must be put between soft blankets. "*Bedding in blankets reduces by a good three-fourths the risk of inflammation of the heart run by patients in rheumatic fever, diminishes the intensity of the inflammation when it does occur, and diminishes still further the danger of death by that or any other lesion; at the same time it does not protract the convalescence*" (CHAMBERS'S *Lectures, chiefly Clinical*, p. 147). The patient ought to be carefully wrapped up in blankets of the newest and fluffiest kind that can be got, which are to be so arranged as to shut off all accidental drafts of air (Dr. ROBERTS). On no account are the bed-clothes to be thrown off, however warm the surroundings may become; for an evenly high temperature to the skin, combined with absolute rest, are of the greatest value in rheumatic fever. These two conditions are worth all other means of relief put together (Dr. KING CHAMBERS). The bed should be protected by screens from draughts of air. All the joints, whether affected or not (*i.e.*, the limbs), ought to be enveloped in cotton wool, a layer of which ought also to be put over the chest on both sides, with an arrangement for permitting the region of the heart to be easily got at for its occasional examination (Dr. ROBERTS). On the other hand, the following are the indications which have guided Dr. R. Southey of St. Bartholomew's Hospital, London, in the treatment of acute rheumatism:—

"To relieve local pains; to reduce fever; to diminish the gravity of the various complications; and to promote early and complete convalescence. I do not consider that the *materies morbi* are eliminated either by sweating or by purging. A spring bed with a tightly stuffed horse-hair mattress over it, is that which rests and supports the trunk and limbs, and relieves the joints most. I am no advocate for, and have never allowed my patients to be placed between the blankets instead of the sheets; neither will I permit flannel bed-shirts to be worn. The object I have in view is to keep one uniform temperature about them, which the bed provides for; but not to keep the patient swathed in flannel reeking and saturated with decomposing perspiration. As a matter of fact I observed that a large proportion of hospital rheumatic patients were persons who habitually wore thick flannel shirts and under shirts, and did not change them very often. Cotton shirts and cotton sheetings are better suited than linen to rheumatic people. My practice has been to keep my rheumatic cases cool rather than hot. They are certainly easier in consequence."

As to medical treatment, *venesection*, *calomel*, combined with *purgatives*, *colchicum*, and *opium*, are the remedies which have been hitherto most generally made use of in the treatment of acute rheumatism. To the success of each of these remedies the late Dr. Latham has borne his testimony, "so far as that, under the use of each," he writes, "I have seen patients *get well*." No disease has been treated by such various and opposite methods, so that "acute rheumatism has experienced strange things at the hands of medical men." "Had he written in these days Dr. Latham would have

had many other strange things" to add to his list:—*Alkalies, acids, salines, hot water, cold water, lemon juice, citric acid, chloral, belladonna, iodide of potassium, ergot, digitalis, aconite, guaiacum, emetics, sulphur, antimony, perchloride of iron, quinine, iodine, plaster of Paris bandages, galvanism, subcutaneous injection of carbolic acid, blisters, podophyllum, cynara, propylamine, chloro-hydrate of trimethylamine*, and last, but not least, *salicine, salicylic acid, and salicylate of soda*. All these and many more remedies have been used in rheumatic fever; and each has been extolled as more potent than all the rest (Dr. E. MARKHAM SKERRITT, *Brit. Med. Journ.*, July 28, 1877, p. 104). But as the disease, when uncomplicated, tends to terminate sooner or later in recovery, and may sometimes subside with marvellous rapidity under every variety of remedy, it is obvious that no sound inference can be drawn as to the success of any particular method of treatment, unless such treatment has been largely adopted in cases of exactly similar severity—as estimated by the correlation of pulse, respiration, and temperature (measured by thermometer)—and been attended with tolerably uniform results. Each and every plan of treatment which has therefore been hitherto proposed is regarded by the profession as unsatisfactory. (See account of a discussion upon a paper read by Mr. Dickinson to the Royal Medico-Chirurgical Society of London, and reported in the Medical papers of 21st June, 1862.) If in one person's hands any particular remedial course has proved efficient, it has signally failed in those of another. If at one time a remedy has proved efficacious, it has been found inert or injurious at another, under different circumstances of age, sex, constitution, and the like. These facts ought not to appear strange to those who consider the true nature of the disorder, and the variety of circumstances under which the physician may be called upon to minister to his patient's relief. *Bleeding*, which in the young, plethoric, and robust, may be necessary to allay excessive vascular action and cause free secretion, may in the weakly induce irritability of the heart, and a consequent attack of cardiac inflammation. *Opium*, which in one person may prove of the greatest service in promoting free perspiration and in allaying the general irritability of the system, may in another check the biliary, "lock up" other secretions, and thus prevent the elimination of effete or deleterious matter from the system. The continued use of *calomel*, and constant purgation by it, may be beneficial to one patient, by removing large quantities of unhealthy secretions, but may unnecessarily exhaust the strength of another, and tend very greatly to impede recovery. So, in regard to every remedy which has been proposed: what is useful at one time may prove useless, or positively injurious, at another. Special attention has also been drawn to the fact by the *mint-water* treatment of Sir William Gull and Dr. Sutton, "that rapid recoveries were often made when the only treatment was good nursing;" that symptoms subside wholly irrespective of treatment between eight and ten days. The average duration of acute symptoms under treatment was 9·1 days; or from the beginning of the disease 17 days. With regard to heart complication it is affected early in the disease—generally within the first week; and if it escaped for a week it would probably escape altogether. Sir William Gull and Dr. Sutton thus came to the conclusion that drugs had little

influence in shortening the disease; and many drugs have had the credit of warding off heart complications simply because the time for the occurrence of these lesions had passed before the medicine was given.

To learn how to adapt our present remedies to the exigencies of each particular case is what is now most of all required. Acute rheumatism is manifestly a highly inflammatory disease, but of a peculiar constitutional origin, as has been fully shown. The blood drawn presents a more copious layer of "buff" than it does in most other diseases. The proportion of fibrine being greatly increased, we can hardly feel surprised that bleeding has been largely had recourse to. But although bleeding has been extensively adopted, the profession has hitherto been much divided as to the advantage derived from the practice. A careful analysis of conflicting evidence leads to the conclusion that *general blood-letting is not to be recommended, and on pathological grounds it is not justifiable*. No advantage is gained as to time; but temporary, if any abatement of sufferings follows its use; lastly, this mode of treatment appears to have caused in the practice of Bouillaud an unusually large number of cases of *myocarditis*—a larger number, indeed, than has been witnessed by any other person in the profession. A predisposition to cardiac inflammation is doubtless engendered by copious and repeated blood-letting: one-half the cases of acute rheumatism became so complicated after treatment by bleeding (DICKINSON). It undoubtedly increases the irritability of the heart, and consequently favours the production of cardiac complication. General blood-letting is only beneficial in first attacks occurring in young, robust, and otherwise healthy persons, particularly in those cases which are marked by unusual severity of their symptoms, or are unaccompanied by free perspiration. It ought at all times to be cautiously employed and carried to a small extent only—viz., from *eight to twelve ounces*, according to the age and strength of the patient, the object being to *favour the action of other remedies, and to promote free secretion* by its use, rather than to arrest or cut short the disease (LATHAM, FULLER). If, therefore, perspiration is free, and remedies are absorbed, blood-letting is not to be thought of. In mild cases it is unnecessary; in the delicate, and those of weakly constitution, it is inadmissible; and in the well-expressed rheumatic diathesis, or when the disease is distinctly ascertained to be hereditary, it utterly fails in modifying the morbid action, is ill-borne by the system, and therefore should only be practised under the extremely urgent and specific conditions just mentioned (WILLAN, FORDYCE, ALISON, TODD, WATSON, FULLER, DICKINSON). If circumstances, therefore, ever warrant its employment, it ought necessarily to take the lead of all other measures, and may be used in the young, plethoric, and robust, in whom secretion is insufficient, whose pulse is full and bounding, and whose skin is dry, hot, and burning. A single bleeding is sufficient to relieve the excessive congestion, on which the want of secretion in a great measure depends, and which forms an obstacle to the action of those remedies on which we rely for effecting a cure. *Active purgation* is also to be avoided. It is injurious for three reasons,—*First*, Because it is not necessary to the cure of the patient, and, like bleeding, tends greatly to reduce his strength and protract recovery. *Second*, Because, from the nature of the complaint, the patient is quite incapable of moving, and his sufferings are aggravated,

his irritability is increased, and his heart's action accelerated, by the repeated shifting of his position, which is rendered necessary by the calls of nature. And, *Third*, Because it necessarily gives rise to more or less exposure, which must be prejudicial to a person bathed in perspiration (FULLER). After having obtained one full dejection by the following searching combination, which will secure an abundant flow from the glandular follicles of the intestinal mucous membrane, the subsequent aim should be to obtain merely a single free evacuation every morning.

R Calomel. gr. v.; Pulv. Jalap. Co., ʒij-ʒi; Pulv. Zingiberis, gr. iii.-gr. v.; *misce*—to be taken in a little milk.

A daily evacuation of the bowels is then to be maintained by mild salines, such as the *potassio-tartrate of soda, sulphate and carbonate of magnesia*, taken as a draught in the morning; and preceded every second night by a *Plummer's pill* at bedtime, combined or not, according to circumstances, with a full dose of *opium*; or, a grain of the *watery extract of Barbadoes aloes* may be found sufficient on alternate days. If the bowels act only once a day, a laxative dose of *calomel* and *opium* may now and again be prescribed, with the view of modifying the character of the intestinal secretions. Dark-coloured and offensive stools indicate the necessity for the dose, followed by a draught of *infusion of senna*, together with half an ounce of the *potassio-tartrate of soda*, and *twenty minims* of the *vinum colchici*; and these should be repeated every evening and morning till healthier evacuations are obtained—i.e., till the motions are light-coloured, more bilious, and less offensive. *Opium* may be given with the greatest advantage in the early and most painful stage of the disease occurring in adults, to the extent of *six or eight grains* of the powder in *twenty-four* hours, or two grains of the *extract of opium* may be given every night; to children, *half-grain* doses of the powder, or less, according to age, may be given every three or four hours. The bowels require to be kept open during its use; and *its influence* requires to be *closely watched*. If the urine increases in quantity, if the motions become more healthy in appearance, and the coated tongue cleaner and less red, the influence of *opium* is beneficial. It encourages sweating. *Hydrate of chloral* is another valuable soporific and sedative. *Colchicum* administered alone is a dangerous agent; but its virtue as a remedy may be obtained in small doses in combination with other medicines. It may be given with small doses of *ipecacuanha, alkalies, and opium*. It promotes evacuation by the kidneys. It is only in some cases, however, that it appears to be of service. It is far less efficacious in the weak and nervous than in the more robust and less easily depressed, and of less value in purely *fibrous rheumatism* than in cases where the *synovial inflammation* predominates. It has proved less advantageous in proportion as the fever has exceeded the articular swelling, and as the urine has been less highly charged with lithates. The operation of this remedy must under all circumstances be most carefully watched; and a daily evacuation from the bowels must be secured during its use. If the lithates disappear from the urine, if the pulse becomes weak, if faintness, nausea, or purging supervene,

the *colchicum* must be at once discontinued; but till some one of these symptoms occurs, a grain or a grain and a half of the acetous extract or the inspissated juice, or from fifteen to twenty minims of the wine of *colchicum*, may be safely and with advantage administered two or three times a day (Dr. FULLER).

Veratria is to be used in small doses. The ordinary formula recommended is:—

Veratria and extract of opium, one grain each, to be divided into ten pills, of which two pills are to be given the first day, three the second, four the third, five the fourth, and so on, increasing one pill each day, until the condition of the pulse or the irritation of the mucous membrane compels a diminution.

The beneficial effects of *guaiacum* are obtained in those cases which are unaccompanied by perspiration, and in which the excretory organs are gently excited by the action of the remedy; but when the patient is perspiring freely, and when it neither purges nor causes diuresis, very little benefit is obtained from its use. Combined with *bitartrate of potass, sulphur*, and *rhubarb*, it constitutes the chief ingredient in a celebrated empirical formula, well known to army medical officers under the name of "*Chelsea Pensioner*," which has obtained a high reputation amongst old soldiers as a remedy for the "pains," or chronic rheumatism. In some large hospitals, both civil and military, this remedy is in common use in the treatment of old chronic cases. Its composition is as follows:—

R. *Pulv. Guaiaci*, ʒi.; *Pulv. Rhei*, ʒii.; *Bitart. Potassæ*; *Sulphur Sublim.*, ā ā ʒi.; *Pulveris Nucis Moschatæ*, ʒii.; *Mel. vel Glycerina*, lbi.; *misce bene*. Of this compound two large spoonfuls may be taken night and morning.

Another formula is the following:—

R. *Pulv. Sinapis*; *Sulph. Sublim.*, ā ā ʒiii.; *Pulv. Guaiac.*, ʒiss.; *Pulv. Rhei*, *Potas. Nitrat.*, ā ā gr. xlv.; *misce bene*. Honey, treacle, or glycerine, sufficient to make the dose into an electuary, is the most convenient form for use, of which a teaspoonful every alternate evening is useful; or a teaspoonful of the powder may be mixed with milk, and so taken at bedtime.

But, the general treatment of the constitutional disease originally recommended by Brocklesby (1764), and followed successfully by Macbride (1772), Basham, Fuller, Furnival, Garrod, and now by many other physicians, is that by *alkalies* and the *neutral salts*, with *colchicum*, a little *antimony* being sometimes added, with the occasional aid of *calomel*, used only as a remedy to promote evacuation from the glands of the intestinal mucous membrane. *Alkalies*, and the neutral salts, may be given in combination with *colchicum*, full doses of *opium*, and a little *antimony*. They aid disintegration, and increase the elimination of sulphuric acid, by augmenting the alkaline condition of the blood (PARKES). The *alkalies* ought to be given largely. Patients lose their pains under their influence,

and proceed rapidly to convalescence. The pulse is generally tranquillised within *forty-eight* hours from the commencement of the alkaline treatment; and if in twenty-four hours the pain is lulled and the local inflammation greatly subdued, the constitution is evidently coming under the influence of the remedy. The *duration* of rheumatic fever is undoubtedly shortened by alkalies:—6 to 7 days, as compared with 9·1 days of *mint-water cure*, and the total duration 13 to 14 instead of 17 (GARROD). The form in which the remedy is to be given is that of a simple *saline* or *nitre draught*, to which from two to three drachms of the *potassio-tartrate of soda* may be added, with ten to fifteen minims of the *vinum colchici*, from fifteen to twenty minims of the *vinum antimonii*, and from ten to fifteen minims of the *tincture of opium*, or of *Battle's sedative solution*, to prevent the salt running off by the bowels. This draught is to be repeated for the first twelve or twenty-four hours, at intervals of three or four hours, according to the strength of the patient and the severity of the attack; and if pain is excessive, a pill containing half a grain to a grain and a half of *opium*, or an equivalent dose of *Dover's powder*, may be given once or twice daily. In the use of these remedies, constipation and narcotism are to be avoided on the one hand, and diarrhoea to be guarded against on the other (FULLER). If the saline treatment is to be used alone, then a solution of *nitrate*, *acetate*, and *bicarbonate of potash* should be given in such doses that *ten* or *twelve* drachms of the two latter salts together are taken in the twenty-four hours. Half a drachm of the *acetate*, with a drachm or a drachm and a half of the *bicarbonate*, and *ten grains of nitre*, dissolved in an ounce and a half of water and sugar, or lemonade, or barley water, and given every two hours, night and day, until the joint affection and pains have begun to yield in severity, are sufficient for this purpose. Or the following:—*Nitrate of potash*, one drachm; *acetate of potash*, three drachms; *water* or *barley water*, eight ounces;—of this mixture one ounce for a dose ought to be repeated every two, three, or four hours, according to the urgency of the symptoms; or from twenty to sixty grains of the *bicarbonate of soda*, or of *potash*, may be given every three or four hours in half a bottle of soda or seltzer water; or in an effervescent *citrate of ammonia*, or *potash draught* (TANNER). This is the treatment which most of all seems efficient in warding off the cardiac complications; and Dr. J. J. Furnival was the first (after Prout) to direct the attention of the profession to the use of alkaline remedies (*carbonas potassæ*) in the treatment of *Rheumatism*, and especially as a preventive of the cardiac complication (*Lancet*, 1841, p. 305). The good effects of such treatment as a preventive of the cardiac affections have been since fully demonstrated by Fuller, Garrod, Basham, Goodfellow, and others. Dr. Dickinson has given statistics in which the percentage of heart disease occurring under the alkaline treatment was 2:1 as against 31·7 under various other remedies. To be effective, the remedy must be carried out with energy and perseverance, till the articular and febrile disturbance are lessened, and till an alkaline condition of the urine is established as soon as possible. *Liquor potassæ* in ʒss -doses may also be given to the extent of ʒiii . to ʒvi . in twenty-four hours (PARKES). Brocklesby is the earliest authority for the use of large doses of *nitre* in the treatment of acute rheumatism. He enjoined, for a diluting drink, water-gruel boiled smooth, in each quart of which he dissolved

two drachms of *nitre*, with or without sugar. He often thus prevailed on soldiers to take *ten drachms* or more of *nitre* in *twenty-four hours*. Dr. Garrod speaks well of the combination of *quinine* with the alkaline treatment after ten years' trial. It is simply following out the treatment by *bark* (unpractical, from the large doses required) recommended by Fothergill, Morton, and Haygarth. Five to ten grains of *sulphate of quinine* are to be given two or three times a day. The *alkaline* and *saline treatment* of rheumatism as the basis of operation seems the most rational; for it has been demonstrated that in cases of acute rheumatism there is an absolute deficiency of the saline ingredients of the blood; and while there is also a liability to fibrinous exudation, the tendency of saline remedies is to suspend the separation of fibrine (STEVENS). The treatment which has saline remedies for its basis thus contributes to restore the balance of the saline ingredients in the blood, and controls the tendency to fibrinous deposition. The progress of inflammatory action being thus retarded, time is gained for other remedies to effect the diminution of the excess of fibrine present in the blood, as well as the destruction of the rheumatic element, whatever that may be. On the other hand, however, the patient is often left weak and anæmic to a marked degree by the use of alkalies; and also, that when heart complications do come on under alkalies they are more troublesome, the effusion of pericarditis is often very large in amount, and there is a greater tendency to carditis. Thus alkalies, while they shorten the disease, do not always prevent complications, and they debilitate the patient. Cases treated by *alkaline* instead of *neutral salt* are also slow in convalescence, the patients being weak and pallid for some time afterwards. The experience of Dr. Reginald Southey is also against the alkaline treatment, especially in the relapsing cases. The alkaline treatment or mint-water is, however, equally well adapted for the acute continued form; but alkalies, unless pushed to full and frequent doses, are wholly useless in relieving pain; and in such large doses as half a drachm or a drachm every two or three hours, they quickly reduce the strength, and render the patient extremely anæmic. They thus retard convalescence not inconsiderably. (*Bartholomew's Hospital Report*, l. c.)

In 1869, Dr. Russell Reynolds reported, in the *Brit. Med. Journ.*, cases of rheumatic fever treated by the *tincture of the perchloride of iron*, in doses varying from 15 minims to a drachm every four hours, with or without 20 to 30 minims of *glycerine*, and *spirits of chloroform*. In *Brit. Med. Journ.* of Oct. 2, 1875, he again gave a summary of results in a series of sixty-five cases, in which the relief of pain was often marked and early. In more than half the cases the pain was gone by the tenth day; in 22 per cent. all pain had gone by the fifth day. The pulse sometimes fell from 120 to 40 in two days; and, in several cases, Dr. Reynolds observed a remarkable diminution in the frequency of the pulse, at and after the time at which temperature had become normal, namely—40, 30, and even 28; but regular in rhythm and in force, the patient feeling neither pain nor faintness. The temperature became normal by the fifth day in 36 per cent.; and in severe cases before the tenth day in 50 per cent.; in moderate and slight cases in 71 per cent. Heart disease was present in 48·1 per cent.; but no distinction is made between those cases in which it existed before, and those in which it came on under treatment. In twenty-

three cases treated with *iron* in University College Hospital in 1875, the heart was affected in 30·4 per cent.—a large proportion when compared with those cases of Dr. Dickinson's treated by *alkalies*, where the percentage was only 2·1. As to duration of the cases also, the cases treated by *iron* do not compare favourably with those treated by *alkalies* by Dr. Garrod, where the average duration of the whole number of cases was six to seven days. Dr. Samuel S. Dyer of Ringwood has also given the treatment by the *tincture of perchloride of iron* an extended trial, and writes as to the satisfactory nature of the result (*Brit. Med. Journ.*, May 6, 1876, p. 563). *Quinine* has been largely used in France—in large doses—from 15 to 90 grains in the twenty-four hours. The pulse and temperature rapidly fell; the joints were quickly relieved; and the heart was seldom implicated. Combined with the alkaline treatment Dr. Garrod has found *quinine* more efficacious than either alone—there being less tendency to relapse; and the fever leaves the patient in a much more satisfactory condition. *Salicine*, *salicylic acid*, and *salicylate of soda* have all been recently and largely used in the treatment of acute rheumatism; given in doses of gr. x. to gr. xx. at intervals of from one to three hours, and in larger doses of gr. xxx. every hour. Under all of these remedies the average duration of the fever has been remarkably shortened—to 4·9 days under *salicine*, and 4·1 under the *acid*—an advantage of from one to three days over the results of the *alkaline* treatment; and in comparison with the *tincture of iron* treatment, in which the temperature became normal in half the cases in ten days, under *salicylic acid* it became normal in half the cases in three days. The duration of rheumatic fever has, therefore, been shorter under these remedies than it has been under any other plan of treatment. But there is no evidence that either of these are specific remedies, for they do not cut short the disease; nor prevent relapses when under their influence; nor do they ward off complications. They seem rather to act by reducing temperature as they do in other febrile conditions, and they lessen the chance of complications in the same proportion as they shorten the disease (Dr. MARKHAM SKERRITT). The observations of Fürbringer, Reiss, Fischer, Stricker, and others have established the fact that these remedies possess a remarkable power of lowering the abnormally high temperatures in febrile diseases. In Dr. Stricker's experience of the drugs in acute rheumatism, *salicylic acid* may be given in hourly doses of from grains vii. to xv. without injury to the human system; and for a longer time to young and vigorous subjects than to the old and feeble, as toxic symptoms appear sooner in the latter than in the former. These toxic symptoms are of various degrees—such as, noises in the ears, difficulty of hearing, and perspiration, which, when they appear, contraindicate the further use of the remedy.

“In an interesting paper in the *Lancet* of 4th and 11th March, by Dr. T. MacLagan, recently of Dundee, on the treatment of acute rheumatism by *salicine*, the author relates seven cases of acute and subacute, and one of chronic rheumatism, treated with this remedy, and gives the following conclusions as the result of his experience:—(1.) We have in *salicine* a valuable remedy in the treatment of acute rheumatism. (2.) The more acute the case, the more marked the benefit produced. (3.) In acute cases, its beneficial action is generally apparent within twenty-four, always within

forty-eight, hours of its administration in sufficient dose. (4.) Given thus at the commencement of the attack, it seems sometimes to arrest the course of the malady as effectively as *quinine* cures an ague, or *ipecacuanha* a dysentery. (5.) The relief of pain is always one of the earliest effects produced. (6.) In acute cases, relief of pain and a fall of temperature generally occur simultaneously. (7.) In subacute cases, the pain is sometimes decidedly relieved before the temperature begins to fall; this is especially the case when, as is frequently observed in those of nervous temperament, the pain is proportionally greater than the abnormal rise of temperature. (8.) In chronic rheumatism, *salicine* sometimes does good where other remedies fail; but it also sometimes fails where others do good. These conclusions, it will be seen, agree closely with those arrived at by Stricker and Reiss with regard to *salicylic acid*. The dose of *salicine* used by Dr. Maclagan was from ten to thirty grains every two, three, or four hours; fifteen grains being an average dose."

As to *salicylate of soda*, Dr. R. Southey gives fifteen grain doses every two hours till sixty grains are taken, and then continues with fifteen grain doses every four hours, to keep up the effect of the drug; and if pericarditis is present he adds five drops of *liq. opii. sedativ.* with each dose. His experience of the *perchloride of iron*, as recommended by Dr. R. Reynolds, is not favourable. In the acute continued cases it has been associated too frequently with hyperpyrexia to regard it as altogether free from danger; and in the relapsing forms, its administration neither shortened the cases nor prevented relapses. These relapsing cases of rheumatism may be kept on *salicylate of soda* very well for the first seven days; but the opportunity of the spontaneous remission which then generally takes place, and is indicated by the thermometer and the patient's own sensations, should be seized to shake the periodicity of the complaint. This *quinine* has the power of doing, given in the proper doses, as in ague, and at the proper date. Dr. Southey gives two ten-grain doses at two hours' interval, between five and seven p.m., on the evening of the seventh day, and repeats the dose on the evening of the ninth day again. The natural relapse is then postponed, and the patient may be permitted better nourishment, and given a little *pepsine* and *acid* with his dinner to ensure its better digestion. Should no relapse happen, again on the fourteenth and sixteenth days full doses of *quinine* should be given; but it is not till the thirty-second day is past that all risk of a relapse is over. During convalescence, although the patients are anæmic, Dr. Southey does not recommend *iron*, as the *mineral acids* and *tincture of nux vomica* promote appetite and digestion far better. *Quinine* and *iodide of potassium* has long been a favourite draught of his in treating the anæmic relapsing forms. *Two grains of quinine* are combined in it with *five of iodide of potassium*, to be taken every four or six hours after the fourteenth day (*St. Bartholomew's Hospital Report*, Vol. XIV., 1878, p. 22). Hyperpyrexia may occur suddenly and rapidly in acute rheumatism, as in other diseases. The temperature may rapidly rise to a height incompatible with life, with evidence of profound nervous disturbance; and usually by the disappearance of pain from the joints. Such hyperpyrexia has supervened under all sorts of treatment (see page 840, *ante*). The extremest vigilance on every one entrusted with the care of the patient must be inculcated; and

on the appearance of anything strange in the aspect or demeanour, to use the thermometer assiduously; and, if ever it rose to 105° Fahr., to send at once for the physician (Dr. H. THOMPSON). Treatment directed simply to the reduction of temperature may save the life of the patient. A temperature in rheumatic fever above 106.5° Fahr. has hitherto been invariably fatal; spontaneous recovery has never been known to occur when the temperature has passed 106.5° Fahr.; but death has occurred before this temperature has been reached—at 105.8° and 106° . Hence the commencement of active treatment by the cold bath ought not to be delayed when such a temperature has been reached. If left alone, the patient *must* die; and the only treatment which has succeeded with such cases has been the effectual external application of cold, by the bath, at a temperature of 90° or 95° Fahr. in the first instance; gradually reduced by the addition of cold water to 70° Fahr., or even lower in some cases (see p. 303 *et seq.*, *ante*). As far as the bath is concerned we must take the thermometer for our guide.

“It is important to remember that the bath should be prepared and everything got ready before this; for the final rise of temperature is often very rapid— 5.5° in an hour and a quarter—from 104° to 109.5° ; and the patient may die before the treatment can be applied. There is another practical point to be borne in mind—that there is almost always a considerable fall of temperature after the patient is removed from the bath, so that it is not safe to bring the temperature down to the normal in the bath. I remember, in the early days of this treatment, how a friend of mine reduced the temperature of a rheumatic patient to normal in the bath, and, to his consternation, found it would not stop there, but kept going down till it reached 93.2° ; there it was fortunately brought to a stand. The amount of this subsequent fall varies, but the average may probably be safely taken as 4° . In accordance with this, I removed the patient from the bath when the temperature was 102° , and the subsequent fall, occurring in the next ten minutes, was exactly 4° , bringing the temperature down to 98° ” (Dr. MARKHAM SKERRITT, l. c.)

So singularly calming is the bath, even when there is no great exaltation of the body heat, that it may be administered as a simple palliative in all exacerbations of delirium and restlessness, although the heat may have fallen far below its maximum intensity, even to 102° Fahr.

External topical applications are efficient aids in treatment. Warm anodyne fomentations are always employed with advantage; and of all applications a mixed *alkaline* and *opiate solution*, applied as a fomentation, is the most powerful in allaying pain. The solution so highly recommended by the late Dr. Fuller for this purpose, is composed of an ounce of the *carbonate of potash* dissolved in a pint of the decoction of poppies or of rose water, to which six drachms of *Battley's solution* (*Liquor sedativus*) is added, or common *tincture of opium*, which is less expensive and equally efficient as an external application, but less strong. *Chloroform* and *belladonna liniment* (as recommended by the late Dr. Sibson) is an excellent pain-relieving local application in either the acute or relapsing form of the disease. If the hands, elbows, knees, or feet are the seat of inflammation, gloves or caps are made of the *spongio-piline* to fit these parts; if a greater extent of surface be involved, a portion of the *spongio-piline* is cut large enough

to envelop the entire surface. The spongy surface of this epithem is first moistened freely with water, and any superabundant fluid squeezed from it, so that the linen or bed of the patient may not be unnecessarily wetted. *Nitrate of potash*, or the salt to be employed, *in powder*, is then freely and plentifully sprinkled over the moistened surface, or rubbed in, to secure its solution and the thorough impregnation of the epithem; it is then applied to the inflamed part, and lightly secured by a roller. Nothing further is required than once in about six hours to remoisten the spongy surface; fresh addition of nitre is never required, if sufficient quantity has been used in the first instance. The salt in powder is hence better than a saturated solution, which nurses seldom succeed in making (BASHAM, *Med.-Chir. Trans.*, Vol. XXXII., p. 10). Alkaline baths are recommended by Dr. Austin Flint. Two pounds of the *bicarbonate of potass* and one pound of the *nitrate of potass* are to be dissolved in water, at a temperature of 100° or 99° Fahr.; and the patient ought to go into the bath at a temperature of 98° or 98·5° Fahr., and remain for not longer than ten minutes. A tepid bath at 98° or 96° Fahr., into which the patient can be dipped suspended on a sheet, and lifted similarly back to bed, comforts them and secures their cleanliness (Dr. R. SOUTHEY). Hot air, or vapour baths, different forms of cold bath, the hot blanket pack bath (as advocated by Dr. T. S. Dow, see in *Brit. Med. Journ.*, 1875, p. 40), or sponging the skin with cold or tepid water, are each in turn methods of treatment which have given relief; but baths ought never to be employed if the skin is acting freely; only if it is hot, dry, and burning, its action may be successfully stimulated by means of some of these baths.

In contradistinction to the principle of cure now laid down, and which has been called the method of *neutralisation*, Dr. Herbert Davies, Physician to the London Hospital, has recently advocated a method by *elimination*, originally recommended by Dr. Dechilly of Vancouleurs, in France, in a Memoir to the Academy of Medicine, in 1850 (*Bul. de l'Acad. de Med.*, t. xv.), as a safe and rapid mode of combating rheumatic fever. For this purpose his treatment is *absolutely* and *entirely local*, and consists of *free blistering* only. Believing that the *virus* localises itself for a time in the inflamed joints, and that the intensity of the local inflammation is a measure of the amount of *virus* collected, he orders blisters varying in width, but of considerable size, according to the locality, to be applied round each limb, and in close proximity to the parts inflamed. Thus he affords, through the serous discharge from the blistered surface, a ready means of exit for the *virus*. The blisters are to be applied *near to*, but *not upon every joint* inflamed, at the very height of the inflammatory stage, even when the local pains are the most severe, and the constitutional disturbance the greatest. Dr. Dechilly enveloped the whole joint in a large blister, followed by others, according as joints became affected. Dr. Jeaffreson, of St. Bartholomew's, and Dr. Greenalgh, of the Middlesex Hospital, report favourably as to the results of Dr. Davies's method of treatment of acute articular rheumatism. So also does Dr. Thomas B. Peacock of St. Thomas's Hospital (*Brit. Med. Journ.*, Jan. 18, 1873). It has been tried in France also, and reported on favourably by Dr. Lasèque, of the Necker Hospital in Paris (*Arch. Gén. de Med.*, Nov., 1865). The success of the plan is said to depend entirely upon the

blisters being applied and allowed to remain until they have thoroughly acted. Linseed-meal poultices subsequently applied will be found highly serviceable in promoting a sufficient flow of serum. The blisters should be put entirely round the affected limb; and when the knees are the joints which suffer, the blisters should be cut at least three inches wide. If this method of treatment be adopted, no medicine ought to be given beyond an occasional purge, and no advantage results from combining the *alkaline* or neutralisation system of treatment with that by *blisters*, or the method by local elimination. If the two methods are combined, the period of convalescence is protracted. In some few cases, however, Dr. Peacock is of opinion that constitutional treatment ought to be freely used in combination with the blistering. In other cases it is of advantage to combine the blister treatment with such tonics as *quinine* and *iron*. In the practice of Dr. R. Southey of St. Bartholomew's, London, painting the affected parts in the acute continued form with a mixture of equal parts of *liniment of iodine* and *tincture of iodine* has appeared to procure all the benefit advocated by Dr. Davies for his blister treatment. That the poison is really thrown out by the blister method of treatment is deduced as well from the rapid and permanent relief resulting from the local treatment as from the *neutral and even alkaline condition of the urine*, which is a usual and early result of the treatment, as well as a rapid diminution in the force and frequency of the pulse (*London Hospital Clinical Reports*).

The diet of the patient, in acute rheumatism, should be strictly limited to slops, such as arrow-root, beef-tea (*Liebig's Extract of Flesh*), milk and lime-water, or milk made warm, to which a little *carbonate of soda*, *nitrate of potash*, or *bicarbonate of potash* is added; light puddings, to which sherry or brandy may be added, if depression exist; and even in many chronic cases it is desirable to limit the diet to puddings and white fish. To obviate the great drain upon the system, it is necessary, however, that the rheumatic patient be better fed than in cases of idiopathic inflammation. Beef-tea and jellies may be given, and strong coffee might also be administered frequently, on the same principle that it has been given in typhus fever by Dr. Parkes, following up the indications obtained from the physiological action of that beverage as expounded by Lehmann.

The nature and treatment of the local complication, such as *pericarditis*, will be considered subsequently; but in addition to the alkaline remedies, which have an undoubted influence in shortening the duration of the illness, the bedding in blankets, referred to at p. 849, is a most important measure for preventing the cardiac affection, especially in cold weather and in cold climates.

GONORRHOEAL RHEUMATISM.

LATIN EQ., *Rheumatismus Gonorrhoeicus*; FRENCH EQ., *Rheumatisme Blennorrhagique*; GERMAN EQ., *Gonorrhoeischer Rheumatismus*—Syn., *Tripper Rheumatismus*; ITALIAN EQ., *Reumatismo da Gonorrea*.

Definition.—*An affection analogous to acute rheumatism, associated with gonorrhœa.*

Pathology.—(a.) *History.*—It is only since the commencement of the present century that rheumatism, as a distinct complication of gonorrhœa, has been recognised. It was first recognised by Swediaur, who described it under the name of "*Arthocele, Gonocoele, or Blennorrhagic Swelling of the Knee.*" It has since been described by Sir Astley Cooper, Sir Benjamin Brodie, Ricord, Rollet, and many others—more recently in this country, especially by Barwell, Chambers, and Berkeley Hill.

(b.) *Causation.*—The rheumatic diathesis, conjoined with urethritis, is held to explain the connection; but, as a rule, patients who are liable to gonorrhœal rheumatism are generally those exempt from acute rheumatism, or anything like the rheumatic diathesis. It is maintained also that gonorrhœal rheumatism is not rheumatism, but the phenomena are considered the result of some reflex influence of the gonorrhœa exerted through the inflamed urethra (GRISOLLE, FOURNIER); that the gonorrhœal matter acts like a disease-poison, and infects the system like the poison of *scarlet fever*; and that the discharge of pus and urethral suppuration correspond to the cuticular eruption of scarlet fever (TEXIER.) In this respect such a theory would bring the nature of the disease in correspondence with the view taken by Barwell, who regards it as a "slower form of purulent infection, produced by inflammation of the prostatic veins;" and Dr. T. King Chambers considers that the "disease ought to be classed with pyæmia." If such is the correct interpretation of gonorrhœal rheumatism, the pyæmia must be of that chronic kind described by Sir James Paget, which is much slower in its progress, and less severe and dangerous in its course, than the pyæmia described at p. 796, *ante*. The form Sir James Paget describes under this name has a close alliance with rheumatism (*St. Bartholomew's Hospital Reports*, Vol. I., 1865). That the rheumatic phenomena are due to the gonorrhœa may be inferred from the fact—namely, that it attacks patients who never suffer from rheumatism, except when they contract gonorrhœa; and that it sometimes recurs in the same patient with every gonorrhœa he contracts. The influence of sex is also remarkable in the pathology of gonorrhœal rheumatism. Women rarely suffer. All the undoubted cases of the disease have occurred in men; but as men contract gonorrhœa from women, a question arises of great pathological importance, but very difficult to collect evidence upon—namely, suppose a woman inheriting or suffering by habit or diathesis from rheumatism, and at the same time from gonorrhœa, *may she not convey to man the rheumatic condition with the gonorrhœa?* Knowing as we do that persons subject to rheumatism are particularly prone to contract gonorrhœa, may not the cases of gonorrhœal rheumatism be those contracted from rheumatic women suffering from gonorrhœa? These questions have not yet received any attention.

Symptoms.—The disease presents certain characteristic features sufficient to indicate its origin. This special form of rheumatism commences either when the gonorrhœal discharge continues in a profuse degree, or when it has been more or less speedily suppressed, or after it has quite disappeared. The parts affected are generally the knee joints, or the knee, ankle, and toe of one side (see page 95, *ante*). Sometimes these affections are accompanied, preceded, or followed by severe rheumatic ophthalmia, in the form of a "*scleritis.*" But the sclerotic and the iris are

less frequently attacked than the joints. The knee-joint is affected, however, far more frequently than any other joint; next the ankle; then the hips, fingers, and toes in about an equal ratio; then the shoulder and wrists. The bursæ connected with the muscular tendons, especially the *tendo Achillis*, and sometimes the sheaths of the muscles, are affected, and fasciæ of the plantar region. Generally two or three joints at the most are affected in each case; and it is not usual for the affection to change about from joint to joint, as in ordinary acute articular rheumatism. In about one-third of the cases only one joint suffers (ROLLET). It also differs from acute rheumatism in another respect—namely, that when it extends to more joints than one, the first affected joint does not recover its normal condition, but continues after the affection has seated itself in other joints. It is extremely rare for the heart to suffer. The sheaths of the great sciatic nerve sometimes suffer, when the pain along the course of the sciatic nerve is exceedingly obstinate (*gonorrhœal sciatica*). The tissues of the eye affected in gonorrhœal rheumatism are—the lining membrane of the anterior chamber, causing cloudiness and misty vision; the iris, and the sclerotic; the affection sometimes passing from one eye to another. The rheumatic affections usually appear before the urethral discharges have ceased. It has been known to occur so early as the first or second week of the gonorrhœal discharge; but in the majority of cases it is at later periods, either when the discharge has been suddenly arrested or suppressed, or when it has passed its climax, and is gradually passing into a gleet. It is then generally observed that the running becomes more copious for a day or two preceding the rheumatic phenomena; and this occurs with each exacerbation of the rheumatism and each joint affected. The affection expresses itself by stiffness and severe aching in one, generally the knee-joint, and usually more severe on one side than on the other. Sometimes a chill and wandering pains are experienced before any particular joint becomes implicated; but the general symptoms are at no period comparable in violence to those of acute rheumatism. When a joint becomes affected the pain soon becomes acute and burning, and increases in severity. Effusion rapidly takes place, distending the synovial sacs of joints or bursæ of tendons; but the integument over the affected part generally retains its normal colour—the blush of inflammation being rare. This collection of fluid greatly interferes with the movement of the limb; and pain is also felt on the slightest movement or on pressure. The tongue is generally loaded and the bowels confined. Perspiration is often copious, and generally offensive.

Prognosis.—The more decidedly inflammatory the symptoms, the more likely will the case be to recover soon; and the more passive the effusion into the synovial sacs, the more obstinate is it likely to prove. In any case recovery is rare under six weeks, and is often delayed for months or even years, especially if the gonorrhœa continues, which it is apt to do, as a scanty gleet fluid from the urethra. The subacute and chronic stages are the most obstinate. Relapses are apt to take place, generally in a chronic form; the urethral discharge generally returning with renewed violence, immediately before each relapse.

Treatment.—The condition of the digestive organs generally requires

attention and correction. *Anodyne* fomentation generally affords relief to the pains of the joints. *Dover's powder* is to be given in the acute stage at intervals; and after the more acute symptoms have been subdued, the greatest benefit is obtained from blisters, repeated several times, when effusion within the capsule of the joint is a prominent symptom. An electuary of *guaiacum*, *sulphur*, and *cream of tartar* (in equal proportion) in *glycerine* I have found of use. Vapour baths and Turkish baths are both of great service. The gonorrhœa must be cured as soon as possible by the usual remedies.

The preparations of *iron*, *iodine*, *quinine*, combined with *ammonia*, *potass*, or *soda*, in bitter vegetable infusions, according to the nature of the urinary symptoms, are the best tonic remedies, together with fresh air and good diet. *Iodide of potassium* ought to be used in the chronic form of the disease. Pressure over the joint by strips of adhesive plaster, aided with bandaging from the toes upwards, is often of great service in subduing the swelling which remains, after all other symptoms have subsided. Passive motion must afterwards be used.

MUSCULAR RHEUMATISM.

LATIN EQ., *Rheumatismus Musculorum*; FRENCH EQ., *Rheumatisme Musculaire*; GERMAN EQ., *Muskel-rheumatismus*; ITALIAN EQ., *Reumatismo Muscolare*.

Definition.—*Pain in the muscular structures, increased by motion.*

Pathology.—The aponeurosis and fibrous tissues of the loins, as well as the muscles of the back and the neck, are the parts usually affected. Hence two local varieties—namely, (a.) *Lumbago*; (b.) *Stiff-neck*—especially challenge attention. (1.) *Lumbago* affects the sheaths of the fleshy mass of the lumbar muscles, on one or on both sides of the loins, extending often to the ligaments of the sacrum. It is characterised by the severity of the pain, and frequently its very sudden occurrence. A patient who, a few minutes before, could move with perfect ease, suddenly finds himself unable to rise from his seat, or to move from the position he is in when thus seized with pain, if movement is attempted in any direction. At last he finds some peculiar attitude favourable to the pained parts, which enables him to move to a very limited extent. The pain is increased by every movement of the lower part of the spinal column, or on pressure upon the muscles implicated. Hence the patient walks bending forward, or leaning on a stick, and he dare not attempt to raise himself erect. *Lumbago* is apt to be confounded with phenomena due to congestion of the venous sinuses of the lumbar vertebræ and of the kidneys. These forms of congestion generally cause marked disorder of the urinary excretions, pains, numbness, cramps, or pricking or lancinating pains in one or both limbs. (2.) *Stiff-neck*, *crick in the neck*, *wry-neck*, *torticollis* or *cervical rheumatism*, generally follows upon exposure to currents of cold air on the neck. The pain is either in the back of the neck, or affects only one side. It complicates many cases of sore throat. Sometimes the articulations of the clavicle are also affected. (3.) Other forms of muscular rheumatism

affect especially the *intercostal muscles and pectorales*, as *pleurodynia rheumatica*. Coughing, sneezing, and bending of the body or arm, influencing those muscles, increase the pain, and cause the affection to resemble *pleurisy*. Respiratory movements are restrained on the affected side. Pain of a catching character may be localised in a point, over which it is increased on pressure. The *frontal, occipital, and temporal muscles*, as well as the *aponeurosis or periosteum* of the skull—*cephalodynia* or rheumatism of the scalp—are sometimes affected, and may be confounded with *neuralgia*, or with *syphilitic periostitis*. Diagnosis requires that the pain be traced to muscular tissue or its connections. Rheumatism of the abdominal walls may simulate peritonitis. Muscular rheumatism is common amongst labouring men exposed to cold and wet, especially in the muscles of the shoulders, the upper part of the back, and in the muscles of the soles of the feet.

The changes induced in the tissues by muscular rheumatism are sometimes in the form of callosities, produced by germination of connective tissue, which replace muscles; but this is only in chronic forms of the disease.

Treatment.—In muscular rheumatism, the local appliances which ensure rest and warmth afford most relief. In *lumbago* nothing is so instantaneously beneficial as strapping the back from the level of the “seat” upwards, in imbricated layers of straps of adhesive sticking-plaster—*emplastrum roborans*—spread upon chamois leather, or the common warm plaster of the pharmacopœia may be used (TUFNELL). The patient ought to sit with his bare back before a large fire during the process. *Calomel* purgation is generally indicated, combined with *rhubarb* and *soda*, as the excretions are generally greatly at fault. The subcutaneous injection of morphia will generally give temporary relief. *Opium* may also be necessary internally; and a course of *bicarbonate of potash* with *iodide of potassium* may be also indicated.

CHRONIC RHEUMATISM.

LATIN EQ., *Rheumatismus Longus*; FRENCH EQ., *Rheumatisme Chronique*; GERMAN EQ., *Chronischer Rheumatismus*; ITALIAN EQ., *Reumatismo Cronico*.

Definition.—*Chronic pain, stiffness, and swelling of various joints.*

Pathology and Symptoms.—This is one of the most common forms of rheumatic disease, especially among old persons, usually coming on gradually as age advances. The knee, ankle, hip, elbow, or shoulder joint are those which usually suffer. The soreness, stiffness, and pain generally extends from the joint along the fibrous structures to a greater or less extent, the limb thus becoming the seat of severe pain. Commencing generally in the aponeurotic expansions over the large joints, the affection is apt to pass to the *periosteum*, and to induce there and in the interior of the joints such a chronic morbid action as in some cases occasions the removal of the synovial membrane and cartilages, while a porcelain-like substance comes to supply the place of the cartilage, having a polished surface, and without any of its elasticity, and destitute of secreting power,

from being destitute of synovial structures. The affection of the joints, however, even when most severe, rarely causes immediate destruction of the cartilages. But if forcible or passive motion is prematurely resorted to, the joint may become the seat of inflammation and suppuration in the usual form. When chronic rheumatism is primary, it most frequently attacks those joints or places which had previously been the seats of dislocation, contusion, sprain, or other severe injury. The immediate effects are expressed first upon the ligamentous structures of the joint, passing thence to the periosteum covering the articular ends of the bones, and to the tendinous attachments of the muscles in connection with the bone and its periosteum. These textures all become finally thickened; they lose their flexibility, becoming impaired in tone and in vital cohesion (TODD, COPLAND). When effusion of lymph occurs in the synovial connective tissue, the synovial membranes also become thickened and opaque, and effusion of fluid may take place into a joint. The pain is then generally aggravated by pressure, and still more by motion. If the effusion is moderate, the pain may be relieved by the swelling and separation of surfaces it causes; but if the effusion is considerable, or very great, pain is increased by the distension, but is still less than it was before effusion took place. These phenomena are most expressed in the knee-joints, and in their greatest severity are the result of frequently repeated attacks and long continuance of the rheumatic diathesis.

Prolonged and repeated attacks of chronic rheumatism chiefly affect the hip-joint, and generally all the large joints. It is most common after thirty years of age; and is especially frequent among the labouring poor, and among soldiers and sailors, who are exposed to the changes of season and weather—to cold and wet. The symptoms are always aggravated at night, and by vicissitudes of weather, especially the prevalence of east winds, humid and cold states of the atmosphere, and is mostly associated with derangement of the digestive organs. The joints of the hands are often also liable to be affected; when those of the fingers are generally most deformed, the joints being liable, in extreme cases, to dislocation, when the fingers are drawn more or less out of their normal position.

Pain in chronic rheumatism is often absent, unless the part be moved, and then the agony is severe. In many cases it is quiescent during the day, but it is extremely acute during the night. This pain has a great tendency to shift from joint to joint, often subsiding and again recurring. Redness is rarely present in chronic rheumatism; while pain is the character by which it is most commonly indicated, so that the term "pains" is not unfrequently understood to mean "chronic rheumatism."

In military medical practice much caution and circumspection are necessary in assigning to "pains" alone their proper significance. It is often impossible at first sight to state positively, from the appearance of a soldier, whether a man who complains of "pains" is or is not suffering from chronic rheumatism, expressing itself by "pains" amongst the extensive white fibrous tissues which constitute the sheaths of the muscles, the covering of the bones, and the ligaments of the joints. The man may really suffer excruciating pains in the muscles of a limb, or in the fibrous tissues round a joint, and may be quite incapable of using his limbs in any way, or at all events of performing the active duties of a soldier; and

yet there may be no appreciable heat or local swelling—no material disturbance of the system at the time—no altered state of the tongue or disturbed condition of the pulse, to indicate what he really suffers. Such cases must have the benefit of the doubt, with due caution to prevent “pains” becoming epidemic amongst the men. The military medical officer must therefore be prepared to meet with two classes of cases, namely—(1.) Old soldiers who have previously suffered from rheumatism, or who are predisposed to the disease, and, it may be, “worn out” by previous disease and mode of life, and who suffer from chronic rheumatism, expressed by “pains” diffused over all the joints and general fibrous tissues of the body. The continued exercise of military duties induces so much fatigue that the digestive functions become impaired, and “pains” are induced. Such men must not be regarded as schemers. (2.) Another class of men present themselves with “pains,” who have resolved, by all and every means in their power, to escape as much duty as possible, and endeavour to make the hospital a house of refuge. “Pains” are complained of in the knees, hips, shoulders, or loins; yet the men may be in good health. Such men, in the first instance, must also have the benefit of the doubt till, by care and patient investigation of all the phenomena of life in those men, the nature of the case is fully made out (GORDON, in *Indian Annals*, 1860). Records of temperature taken regularly and continuously will be found especially useful in coming to a conclusion regarding such cases.

Prognosis.—Few deaths occur from chronic rheumatism, so that the numbers that die bear but a very small proportion to those that more or less constantly suffer. The constitutional state associated with rheumatism is one in which attacks of bad health culminate at last in more or less severe paroxysms, after which the person for a time appears to have improved in health, and been better for his illness.

The Treatment of chronic rheumatism does not differ in its general details from that of acute rheumatism. *Decoction of cinchona*, preparations of *guaiacum*, with *alkalies* and *colchicum*, are the best remedies after the action of such searching evacuants as *calomel* and *jalap* have removed morbid accumulations, and improved intestinal secretions. The various forms of the so-called “*Chelsea Pensioner*,” comprising *guaiacum* and *sulphur*, the composition of which is given at p. 853, *ante*, are of great service. *Iodide of potassium* is also of great benefit in the *arthritic* forms of chronic rheumatism, combined or not with preparations of *aconite* and *colchicum*. Warmth of flannel and the warmth of bed, with free perspiration, generally tends to mitigate and shorten the severity of the paroxysms of chronic rheumatism.

ACUTE GOUT.

LATIN EQ., *Podagra Acuta*; FRENCH EQ., *Goutte Aiguë*; GERMAN EQ., *Acute Gicht*; ITALIAN EQ., *Gotta o Podagra Acuta*.

Definition.—*A specific febrile disorder, characterised by non-suppurative inflammation, with considerable redness of certain joints,—chiefly of the hands*

and feet, and (especially in the first attack) of the great toe; attended by excess of uric acid in the blood, and probably also of phosphoric acid. The constitutional affection tends to culminate, at longer or shorter intervals, in a paroxysm, or "fit of the gout," when various joints, textures, or parts of the body are apt to become affected.

Pathology.—(a.) *History.*—About 1787 to 1793, Mr. Murray Forbes, remarking the close connection between gout and gravel, and the tendency to form concretions, ascribed gout to the presence of *lithisic*, or what has since been named *uric* or *lithic acid* in the blood. These concretions are liable to periodical deposition in organs whose vessels are of the smallest order—for example, tendons and ligaments (*Treatise on Gravel and Gout*, pp. 78-80). About the same time, Berthollet had advanced an opinion not dissimilar with regard to the presence of *phosphoric acid*. From numerous observations he concluded that *phosphoric acid* is more sparing in the urine of the gouty and the rheumatic than in that of healthy persons; and, that during a paroxysm it became more abundant and equalled the proportion found in the urine of the most healthy persons. He therefore ascribed gout to the retention and accumulation of this acid, and to its diffusion through the system (*Journal de Médecine*, Juin, 1786, p. 476). The reverse of this doctrine was afterwards maintained by Richerand, who observed that, on the approach of gouty fits, the phosphoric ingredients of the urine diminish, and seem to be conveyed to the joints to produce the arthritic concretions (CRAIGIE). In 1848, Dr. Garrod, of London, published a paper in *The Med.-Chir. Transactions*, in which he proved experimentally the statements of Forbes; and showed that the blood, in cases of gout, contains *lithic acid* in the form of *lithate of soda*; and that in cases of chronic gout with chalky deposits round the joints, *lithic acid* is *always* present in the blood and deficient in the urine, both absolutely and relatively to the other organic matters. He believes that the *acid* is always in excess in the system during gout, and constitutes a very important and almost a pathognomonic sign of gouty disease. The uric acid is found to exist not only in the blood-serum, but also in the fluid effused by blisters, and in the abdominal and pericardial fluids. The abstraction of a very small amount of blood or serum is required to discover the uric acid if it is present in quantity. From one to ten fluid drachms of the serum being taken, it is to be put into a broad and flat glass dish (not watch-glasses), about three inches in diameter, and about a third of an inch deep. Add acetic acid, of the strength of the London pharmacopœia, in the proportion of about six minims to each fluid drachm of serum; a few bubbles of gas are generally evolved at first. When the fluids are well mixed, a very fine thread is introduced, consisting of from one to three ultimate fibres, from a piece of unwashed linen fabric, about an inch in length, which should be depressed by means of a glass rod. After resting for from eighteen to forty-eight hours, depending on the warmth (temperature at or below 70°) and dryness of the atmosphere, the uric acid will crystallise upon the thread. To observe this, a microscope must be used with a linear magnifying power not below 60.

(b.) *Causation.*—Although the specific lesions and morbid elements in the blood are but indications of the gouty condition, they nevertheless

confirm the belief again gaining ground, which teaches the ancient humoral doctrine, that the phenomena of gout are induced by some peccant matter (probably uric acid) which, through malassimilation of food, or of food and drinks of particular kinds, combined sometimes with excessive labour of body as well as of mind, becomes inbred in the constitution, which it is the business of the paroxysm of gout to eliminate. The fact seems to be established by several independent observers, that the blood in gout is charged with *uric acid* in the form of *urate of soda*, to an extent considerably beyond its normal amount; and there are several theories, however, put forth to account for its abnormal excess:—(1.) That it depends upon imperfect oxidation of the blood, together with impaired nervous energy, as the cause of the imperfect oxidation (DUNCAN, in *Dublin Quarterly Journal of Medicine*, May, 1865). (2.) *Urea* and *uric acid* already existing in the blood in the form in which they are to be eliminated; the kidney is regarded as the organ of their excretion. The elimination of *uric acid* is thus impaired in *gout*, just as the elimination of *urea* is impaired in *Bright's disease*. Each of these products accumulates in the blood in each of these diseases respectively; and, acting as a poison, the accumulation of *uric acid* ultimately gives rise to a paroxysm of gout, which “would thus appear partly to depend on loss of power (temporary or permanent) of the acid-excreting function of the kidneys, the premonitory symptoms, and those also which constitute the paroxysm, arising from an excess of this acid in the blood, and from the effort to expel the *materies morbi* from the system. Any undue formation of this compound would favour the occurrence of the disease; and hence the connection between *gout*, *uric acid*, *gravel*, and *calculi*; and also the influence of high living, wine, porter, and want of exercise in inducing it” (GARROD). Thus, Dr. Duncan's theory tends to account for the abnormal and excessive formation of *uric acid*; while Dr. Garrod's tends to explain its non-elimination. But the experiments of Hoppe-Seyler and Zalesky show that *urea* and *uric acid* are both produced in the kidneys; and when found in the blood, their existence there is due to reabsorption. They tied the ureter in geese and chickens, and, in a few days after the operation they found almost all the viscera, including the stomach, heart, and lungs, covered with crystals of the urates, and the joints encrusted with them in a manner very strongly suggestive of gout. From such experiments the following theory regarding the cause of gout is the most probable—namely, that “The gouty diathesis depends on an anomaly of nutrition, in which far more *uric acid* is produced than in healthy persons. As long as the excess of *uric acid* is regularly excreted by the kidneys, the affected person gets along tolerably well, or with no trouble at all. But the case is different when the uriniferous tubules are plugged up by deposits of the urates, and the excretion of the urine is impeded; for then there is a collection of urates in the blood, whence they are deposited in the joints and other organs, when we have an attack of gout. If the deposit of urates in the uriniferous tubules be soon washed away, and the red sandy sediments form in the urine, which are sometimes found even on the diaper of infants, the attack of gout passes over; if this do not take place and the kidneys atrophy, the acute attack becomes chronic gout” (NIEMEYER). Dr. Bence Jones holds views somewhat

similar to Dr. Duncan, so far as a process of oxygenation has to do with the attack.

The natural history of gout shows—(1.) That there is a disposition or tendency in the morphological relations between the solids and fluids of the whole system to develop some specific constitutional poison, which betrays itself by certain constant effects at periodical intervals, although these may be irregular. (2.) That these local effects are prone to develop themselves in the joints of the extremities, or to express themselves by symptoms of a particular kind in the internal organs, and in various textures of the body. Dr. Graves has given the most interesting illustration of these propositions in his clinical lectures; and especially of the development of peculiar local affections in connection with gout. For example, the occurrence of—(1.) “Fugitive pains,” or twitches, which suddenly attack persons of gouty habit. The pain is, no doubt, due to congestion. Sometimes the congestion is more lasting, as in the lobes and cartilages of the ear, and where, as Dr. Garrod shows, concretions very often occur. (2.) A singular affection of the teeth, which consists in an insuperable desire to grind them, is noticed by Graves. (3.) The occurrence of *tic-douloureux* of the several branches of the fifth pair. (4.) Daily paroxysms of intense heat of the nose, which continues for three or four hours, the part becoming first of a bright and then of a purplish red colour, spreading over the upper portion of the cheek. (5.) The occurrence of *gouty sciatica*, in which the specific inflammation affects the trunk of the sciatic nerve, and which, extending by the neurilemma of the nerve, may in process of time extend to the spinal marrow and its investments, and give rise to derangements of the latter, terminating in ramollissement and structural degeneration. This complication of gout was first decidedly pointed out by Dr. Graves. These affections are always greatest when the stomach is most deranged. Dr. J. R. Reynold has also recently described several affections of the nervous system dependent on a gouty habit (*Brit. Med. Journal*, December 15, 1877); and so also has Sir James Paget.

It is now generally admitted that gout is hereditary; and in many instances it is so, whether the intemperate habits of ancestors are followed out, or whether the mode of living be abstemious. In some families it attacks only alternate generations, following what has been called “the law of *atavism*.” The power of hereditary transmission to induce gout has been illustrated in a most interesting way by Mr. Spencer Wells in his treatise on this disease. He shows that the children of a patient born prior to the development of gout in their parents were free from this affection in after-life; but that those children born subsequently to the development of gout in the same parents became afterwards gouty. The potent influence of hereditary predisposition betrays itself amongst some of the labouring classes, and in the upper classes in a still greater proportion. Dr. Garrod instances the case of a gouty gentleman who is able to *trace* the disease in his family for four hundred years. The eldest son has invariably inherited, *with the estates*, gout of the worst form, developed at an early age. On the other hand, again, it is certain, and consistent with the constitutional origin of this disease, that an unusually large proportion of non-hereditary cases are met with among the indolent and luxurious

inhabitants of large metropolitan towns. A flattering hallucination has, indeed, been transmitted traditionally, hereditarily, and historically, which connects the gouty diathesis with high mental development. But the conditions for the constitutional development of this disease must either be more common, or physicians must be getting more acute; for, eminent and observant men are now inclined to discard the doctrine which teaches the exclusively noble origin of gout, and its necessary association with high mental development. The disease is now certainly common and plebeian, as well as aristocratic. It may have been, in the days of Sydenham, that the gouty patients of a physician were to be found amongst "*magni reges, dynastæ exercituum, classiumque duces, philosophi, aliique his similes.*" Now-a-days it is no less certain that the physician, in London, at least, must pay his visits and prescribe for gout amongst "the London labour," as well as among "the London poor;" and his list will number "coal-heavers, bakers, brewers, draymen, house painters, butchers, innkeepers, publicans; and, in wealthy families especially, butlers, coachmen, and porters" (COPLAND, BUDD, GARROD, TODD). A gouty conformation of the body has been accurately described by Sydenham, Frank, Scudamore, and others; and one of the constant characteristics of such a conformation is former or existing corpulence. It is now also known that the disposition to develop gout may be brought about by abnormal habits of existence; and if the hereditary predisposition is present, the conditions for developing the latent diathesis are more easily made efficient. All are agreed as to the influence of full living (*i.e.*, of eating more than the needs of life require), with the free and habitual use of wine; and, especially as to the influence of what may be called gross living—*i.e.*, great and indiscriminate consumption, especially of animal food, with indulgence in beer and malt liquors generally (BUDD and TODD). It appears, also, from the observations of Sydenham, Craigie, Wm. Budd, Todd, and Wood, that it is not so much the particular variety of alcoholic drink used, as the mode and extent of the use, which tends to develop the gouty state. The influence of malt liquors in producing gout is especially obvious in those cases which occur in the lower classes. "There is a body of men employed on the Thames, whose occupation is to raise ballast from the bottom of the river. As this can be done only when the tide is ebbing, their hours of labour are regulated by that circumstance, and vary through every period of the *night and day*, with long intervals of nothing to do, except eat, drink, and smoke. They work under great exposure to inclemencies of the weather; their occupation requires great bodily exertion, occasioning profuse sweating and much exhaustion. Each man drinks from *two to three gallons of porter daily*, and generally a considerable quantity of *spirits* besides. *Gout is remarkably frequent among them*" (Dr. W. BUDD). Such an instance contrasts strongly with the prophylactic influence of corporal labour displayed amongst other classes of the people who do not *labour under the influence of gallons of malt liquor*; and it is interesting to notice that in the non-beer-drinking countries the lower classes escape. Gout is rarely seen in Scotland, at least among many classes in whom it is common in England. They limit their drink especially to whisky. Generally, it may be stated that,—(1.) *Hereditary tendency* is the most important factor in causing gout in any individual

case. It can be traced to this source in about one-half the cases. (2.) The supply of more nourishment (and especially of *nitrogenous* elements in the form of flesh meat) than is required by the wants of the system, to replace what has been used up in the body, is another most important factor in the development of gout. Gout is never developed without being associated with indigestion, or as a sequence of impaired digestion from some error in diet or drink. The foul breath, the loaded tongue, the nausea and sense of weight in the stomach, and the flatulence that most gouty patients are subject to, all tend to show that much more work is thrown upon the organs of digestion, by the quantity and quality of the food and drink, than they are capable of using. Of all wines, port wines and Burgundy are most apt to induce such kind of digestion as accompanies or precedes an attack of gout. A belief in the prophylactic virtue of labour and moderate living probably instigated the abrupt reply of Abernethy to the question, "What is the cure for gout?" when he said, "Live on sixpence a day, and earn it." The disease very rarely occurs before puberty, but is seen in both sexes under twenty. Many cases occur between twenty and thirty; but the period of greatest liability is perhaps from thirty to fifty. After this the chances of exemption increase with age, probably from the more temperate habits of advanced life. But at whatever age gout may appear, every attack establishes a greater disposition to another. Women often suffer greatly from gout, but not in an equal degree with men.

The immediately exciting causes of this disease are very much similar to those of rheumatism, but may not always be obvious as regards any particular attack; which may be set up by excessive fatigue of body or mind, or both; by violent or depressing emotions, grief, rage, fear; by over eating and drinking, and especially by indulgence in indigestible food.

The varieties of gout, in addition to the acute form, are:—(2.) *Chronic Gout, a persistent constitutional affection, characterised by stiffness and swelling of various joints, with deposits of lithate of soda*; (3.) *Gouty Synovitis*.

Sir James Paget has also described a capillary form of phlebitis as due to gout; and has directed attention to the minor signs of gout in the hands and feet; and to gouty affections of the skin and of the digestive and vascular systems (*Brit. Med. Journ.*, May 22, and June 3, 1875). Dr. Owen Rees is indeed inclined to entertain the belief that the phenomena of the acute gouty paroxysm are best explained on the theory that the disease is essentially a *capillary phlebitis*, the venous inflammation being caused by the circulation of a blood-poison. He also considers that there must be some condition necessary to the production of gout, other than the presence of urinary constituents in the blood, while it must still be admitted that such contamination plays a considerable part in the causation of the disease. Dr. Gairdner argues in opposition to Dr. Garrod, that if the excess of uric acid is the cause of gout, the inflammatory symptoms ought only to set in when the effusion of material takes place, whereas the severe pain and vascular excitement appear *before* the swelling takes place, and subside immediately on its appearing. "Is it not probable that, in gout, the blood, charged as it is with urinary material, becomes so changed that a poison is elaborated which inflames the venous capillaries? Sir James Paget has shown that the large venous

trunks are especially prone to disease in gouty persons. If so with the larger trunks, why not with the capillaries? The disease thus beginning in the elaboration of a poison from the urinary materials found in the blood, the venous capillaries inflame as a consequence; severe pain is experienced, which is relieved immediately when effusion and swelling occur. Dr. Gairdner's views have special reference to the veins, but he does not believe in any poisonous influence, nor in the phlebotic state. He considers, however, that the real seat and cause of the painful phenomena of gout is in the distended venous capillaries." (Dr. G. O. REES, *Brit. Med. Journ.*, Feb. 24, 1877.) This distended state, however, is not always present. Its absence is observed in spare subjects, and in those who strongly inherit the disease.

(c.) *Morbid Anatomy*.—Genuine gout is characterised by the encrustation of the articular surfaces of the affected joints by chalk-like masses, composed of *urates*, generally of *soda*. The first joint of the great toe may be the only one showing such a lesion; but in severe and old cases numerous other joints may be so affected. A thick layer of urates covers the surfaces of the joints; the cartilages are rough; and the ligaments, periosteum, and bursæ



Fig. 95.*

near the diseased joints are covered with thick deposits of these salts. Thus the joints are often greatly deformed, and the chalky masses may sometimes be seen actually protruding through the skin covering the joints. These urates may also exist in the eyelids and cartilages of the ear, forming concretions like white pearls, surrounded by varicose veins—a certain sign of gout. These appearances are very well represented in Plate I., facing page 64 of Dr. Garrod's classic work on gout. In the pyramids of the kidneys, there are usually white striæ, composed of urates running in the direction of the *tubuli recti*. (See p. 249, Fig. 71, *ante*.) When gout has thus become fully developed, and has assumed its specific inflammatory character (see p. 94, *ante*), it produces all the forms of articular inflammation which have been described in rheumatism, and these inflammations attack nearly the same parts, as the bones, cartilages, synovial membranes, bursæ, ligaments, muscles, tendons, and aponeuroses. These inflammations have little to distinguish them from rheumatism, except the singular pathological phenomenon of a tendency to the deposition of the urate of soda—a discovery we owe to the late Dr. Wollaston. Occasionally the urate of soda appears to be nearly the sole secretion from the affected part, nothing being seen on the poultice or fomentation cloth applied to the part but this salt in a more or less fluid state. It is secreted from the joints of the toes or fingers, and probably from all their different tissues. Portal gives a case in which the articulations of both hands presented deposits of urate of soda, both within the capsules of the joints and externally among the ligaments, while the tendons of the extensor muscles of the fingers were almost destroyed. In the Hunterian Museum of Glasgow there is a finger from a gouty hand, with a joint opened and bent upon itself, showing not only a deposition of the salt, but an erosion of the cartilages; also another, in

* Urate of sodæ in stellæ, from WEDL.

which the joint is full of this peculiar secretion, and a third in which the joint is everywhere invested with it. In the Museum of St. Thomas's Hospital there is a specimen in which the femoral cartilage of the knee-joint is coated with it, as if smeared over with plaster of Paris; and another in which it is deposited on the ligaments of the extensors of the hand. Guibert gives a case in which the metatarsal articulation of the great toe was surrounded by urate of soda of a rose tint; and, on the inside of the foot, in the cellular tissue, was an abscess containing urate of soda making its way to the surface. On opening the joint the same substance was found, and on cutting through the tendons, pieces of urate of soda were distinctly seen between the fibres. Simon gives an account of a gouty skeleton, of which the bones were completely ankylosed. In the preparations of diseased joints in the Museum of the Army Medical School at Netley (and described by Mr. Gulliver in *The Edin. Med. and Sur. Journ.*, Vol. XLVIII.), the material is said to be deposited outside the joint in the cellular tissue, exterior to the periosteum and articulation capsules. Similar dissections are described by Watson, Moore, and Parry. The bones affected in cases of gout have been found swollen, and sometimes so soft as to have been easily cut by the scalpel. The concretions afterwards harden, and form what, from their colour and appearance, have been termed *chalk-stones*, *tophi* or *tophaceous* deposits, often superficial and of considerable size; so that, when the skin has ulcerated, a patient has been said in one instance to have scored his game of cribbage with his knuckle, and in another to have written on the table with the *chalk* penetrating through the ulcerated tips of his fingers. Not unfrequently, after a time, deposits of *phosphate* and *carbonate of lime* ensue in and around these semi-fluid concretions; but it is not believed that such deposits ever replace the *urate of soda* in gouty subjects.

Symptoms.—These vary according as the disease attacks the joints—*regular* or *articular gout*; or some internal organs, such as the heart, the stomach, or intestinal canal—*irregular* or *misplaced gout*. The proportionate frequency with which these different parts are attacked is not yet ascertained. It may be acute or chronic. Sydenham was himself a great sufferer from this affection, and laboured under it for more than thirty-four years. He thus describes an acute attack or fit:—

“It comes on a sudden towards the close of January or beginning of February, giving scarce any sign of its approach, except that the patient has been afflicted for some weeks before with a bad digestion, crudities of the stomach, and much flatulency and heaviness, which gradually increase till at length the fit begins. The patient goes to bed and sleeps quietly till about two in the morning, when he is awakened by a pain, which usually seizes the great toe, but sometimes the heel, the calf of the leg, or the ankle. The pain resembles that of a dislocated bone, and is attended with a sensation as if water just warm were poured upon the member; and these symptoms are immediately succeeded by a chilliness, shivering, and slight fever. The chilliness and shivering abate in proportion as the pain increases, which is mild in the beginning, but gradually becomes more violent every hour, and comes to its height towards evening, adapting itself to the numerous bones of the tarsus and metatarsus, the ligaments whereof it affects so as sometimes to resemble

a tension or laceration of those ligaments, sometimes the gnawing of a dog, and sometimes a weight and coarctation or contraction of the membranes of the parts affected, which become so exquisitely painful as not to endure the weight of the clothes, nor the shaking of the room from a person walking quickly therein: and hence the night is not only passed in pain, but likewise with a restless removal of the part affected from one place to another, and a continual change of its posture. Nor does the perpetual restlessness of the whole body, which always accompanies the fit, especially in the beginning, fall short of the agitation of the gouty limb. Hence numberless fruitless endeavours are used to ease the pain by continually changing the situation of the body and the part affected, which notwithstanding abates not till two or three in the morning—that is, till after twenty-four hours from the first approach of the fit. Being now in a sweat, he falls asleep, and upon waking finds the pain much abated, and the part affected to be swelled; whereas before only a remarkable swelling of the veins thereof appeared, as is usual in all gouty fits. The next day, or perhaps two or three days afterwards, the part affected will be somewhat pained, and the pain increases towards the evening, and remits towards break of day; and what we call a fit of the gout is made up of a number of these small fits. At length the patient recovers, which, in strong constitutions, and such as seldom have the gout, often happens in fourteen days; and in the aged, and in those who have frequent returns of the disease, in two months; but in such as are more debilitated, either with age or the long duration of the distemper, it does not go off till summer advances. In aggravated cases it attacks both feet and hands, wrists, elbows, knees, and other parts; sometimes bending the fingers crooked and motionless, and at length forms stony concretions in the ligaments of the joints, which, destroying both the scarf skin and the skin of the joints, stones not unlike chalk or crabs' eyes come in sight, and may be picked out with a needle. Sometimes the morbid matter is thrown upon the elbows, and occasions a whitish swelling almost as big as an egg."

The functions of digestion, and especially the hepatic and urinary secretions, are much deranged in all cases of gouty paroxysms. Besides loss of appetite, flatulence, heartburn, stomachache or colicky pains prevail, the tongue is loaded, and the bowels are bound. Air, and impacted fæces, distend the intestines, especially in the epigastric and umbilical regions. The hypochondriac regions, especially the right, are the seat of painful tension and uneasiness. The first alvine dejections are generally solid and dark coloured, not unfrequently very fetid; and in some instances large quantities of dark coloured excrement are brought away. *The urine*, when scanty and of a deep red colour, is voided with pain or scalding along the urethra. The sediment already mentioned, and soluble in water, is rose-coloured or lateritious, and is deposited during the whole course of the attack. It becomes more abundant towards the close or subsidence of the febrile symptoms.

During the first fourteen days the urine is high-coloured, and after separation lets fall a kind of red gravelly sediment; and not above a third part of the fluids taken is voided by urine during the febrile paroxysm, the bowels also being generally constipated during this time. The fit is accompanied throughout with loss of appetite, and chilliness of the whole body towards the evening. Marked remissions are usual

towards morning. *The urea* is probably not below the normal standard, its elimination not appearing to be interfered with during the paroxysm (GARROD, PARKES). The elimination of uric acid by the urine is impeded during the paroxysm; but it exists in large amount in the blood. It has also been suggested, as a hint for further inquiry, that the substances which, in a healthy state, would furnish urea, are converted into uric acid in gout (GAIRDNER). The phosphoric acid is also greatly lessened, and its retention is probably as common as that of the uric acid. Albumen is not uncommon in small quantities, but its occurrence is generally temporary; and if it is persistent, there is reason to suspect the chronic disease of the kidney which is produced by gout (TODD); and casts without albumen may be found in the urine sediments. *After the paroxysm* the water is usually copious; and the uric acid increases as the fit is passing off. *Before the paroxysm* of gout the urine is found to be very deficient in solids, especially in urea, uric acid, extractives, and phosphate of lime; there is, in fact, a diminution of all the chief ingredients. *During the paroxysm* there is insufficient elimination of uric and phosphoric acids, while the urea and sulphuric acid are in sufficient amount; and after the paroxysm the elimination again increases, (PARKES, l. c.) When the fit is going off, a violent itching seizes the foot, especially between the toes, and the skin peels off.

When the paroxysm of gout has taken place, and has terminated in the manner described, the patient appears to enjoy better health than formerly. The appetite is good, the mind more cheerful and active, the body more agile, and the patient is delivered from many feelings of languor and oppression with which he had previously been afflicted. In this state he may remain for two or three seasons without being conscious of any complaint or symptom which would indicate a return of the disorder. In the course of time, however, about the same season of the year, generally in spring, he begins to be sensible of the presence of some of his former feelings, and at length a fit takes place much in the same manner as it did on the first occasion. This second paroxysm is, in some instances, shorter and less violent; in others it is accompanied with various circumstances which show some deviation from the first attack. But whatever be the mode or duration of its attack, another succeeds after a less lengthened interval; in some cases, the same year, in the autumn—in others, next year, in the spring or summer. In general the intervals are shorter the younger the patient. The most common, perhaps, is the biennial or triennial attack, for the space of eight or ten years; then the attacks are annual, then twice in the year; and, in some severe cases, the attacks are so frequent that the patient can scarcely be said to be free from gout the whole year round. Much in all this depends on the habits and constitution of the individual. The disease tends to acquire a chronic character, and rarely quits the patient till it destroys him, either by complete lameness, helplessness, and debility, or by attacking the heart or the brain (CRAIGIE).

Diagnosis.—The diagnosis between gout and rheumatism is often exceedingly difficult, so much so that nosologists have described a hybrid disease, and named it “rheumatic gout.” Mr. Hunter warmly opposed this compound appellation, as many have since done; and, for reasons

afterwards stated, the College of Physicians have excluded the name from the nomenclature. Hunter's opinion was founded on the belief that no two distinct diseases, or even distinct diatheses, can co-exist in the same constitution—a belief which must now be admitted to be incorrect. Such a hybrid disease has been described by Craigie, Wood, Spencer Wells, and Fuller, depending on the influence of the combined cachexia of gout and rheumatism. Mr. Hutchinson also has remarked that,—

“Gout is chronic rheumatism made special. Some condition of the system is added to the simple rheumatic condition, as manifested by the presence of lithate of soda in the blood and in the seat of inflammation. It is generally accompanied by a bad digestion, and at least a functional disturbance of the kidneys, and very often by organic renal changes, so that lithic acid tends to accumulate in the blood. Again, gout is probably chronic rheumatism *plus* a dietetic derangement. Arguments in favour of this view are found in tracing the family history of cases. In most instances of gout, the family history will show chronic rheumatism in some members, frequently on the female side, the males being liable to fully developed gout with chalky deposits. In seeking to ascertain whether a case of chronic arthritis was true gout at its commencement, inquire as to the duration of the primary attack, whether it lasted a short time and was acute in its course, or a longer time in a chronic condition—whether one joint was long attacked, the inflammation lasting many weeks, as in chronic rheumatism—or whether the early attacks were acute, on the various occasions attacking different joints—particularly inquiring whether the first onset was an acute inflammation of the great toe. True gout may be inherited, and thus the tendency to its development inherent to the individual, or it may be acquired by him as the result of his habits—the latter class are usually in early life full eaters, with good teeth and vigorous digestion (*Brit. Med. Journ.*, June 2, 1877.)

Cases of chronic pain, with stiffness and swelling of various joints, may be thus far cases of chronic rheumatism; but when they are attended with deposits of lithate of soda, such cases are directed by the College of Physicians to be returned as cases of “*chronic gout*,” and those in which there is marked distortion are to be returned as cases of “*chronic osteo-arthritis*”—a disease which may be defined as follows:—“*An affection characterised by pain, stiffness, and deformity of one or more of the joints, associated with deposition of new bone around them.*” The disease is also named “*chronic rheumatic arthritis*,” and is described in its place in this volume.

Prognosis.—Every Assurance Office objects to a gouty person as liable to a disease which tends to shorten life from the deterioration of the constitution it occasions. The objection is unquestionably well founded; for although a few persons may reach advanced age notwithstanding repeated attacks of gout, yet many die prematurely, especially from renal disease (gouty kidney), some from asthma, disease of the heart, apoplexy, or from the accidents to which helplessness and debility render the patient liable, while it materially diminishes the chances of recovery from acute diseases and severe injuries.

Treatment.—Seeing that the disease is clearly of constitutional origin, its treatment resolves itself into—(1.) The selection and administration

of those remedies which shall tend to subdue, control, or eradicate the latent disposition, constitutional tendency, or gouty diathesis; (2.) The adoption of such means as may be safely used to modify the severity of or shorten the paroxysms. The paroxysm must be interfered with cautiously. It is the means which nature takes to rid the constitution of the *materies morbi*, and which it undoubtedly relieves for a time, if allowed to run its course. But the removal of the paroxysm, or its subsidence, does not necessarily remove the constitutional diathesis. That must be modified by promoting the transformation of the tissues of the body as much as possible in a healthy way. As to any local treatment during the fit, Sydenham writes, "If outward applications be required to ease the pain of the gout, I know of none, though I have tried abundance both on myself and others, and I have laid aside the use of topical remedies for many years." It is generally admitted that cold is dangerous, while warmth is productive of little relief. In some instances the urate of soda is deposited in such quantity that the skin ulcerates, and the salt is discharged in considerable abundance in a fluid state. It might appear the right practice to apply a poultice and encourage the discharge, in order that, by its entire removal, the joint might be saved. This, however, is by some considered dangerous practice, for the discharge is so debilitating that patients are said to have sunk under this mode of treatment. It is much safer to wait till the chalk-stone becomes concrete, and then operate, by the smallest possible incision, for its removal. With respect to the use of cold water, the practice is as old as Harvey, and subsequently it has been adopted and abandoned by many practitioners. Dr. Parry had at one time two patients who attempted to cut short the fit by plunging their feet in cold water. The relief was instant, but in a few hours both were dead of apoplexy. The fatal result of this remedy in Sir Francis Burdett's case is another instance in point. "A gentleman," writes Dr. Wood, "contrary to the advice of his physician, and anxious for speedy relief, ordered a bucket of water to be taken into his chamber at bedtime, with the view of employing it in this way. In the morning he was found dead in bed."

Antiphlogistics neither alleviate the pain of gouty inflammation nor shorten the attack; while their untimely use, especially general and local bleeding and active saline purges, favours the passage of *acute* into *chronic* gout. Rest is absolutely necessary. *Warm* anodyne lotions or fomentations may be used, and the part afterwards lightly covered or encased in flannel or fine wool, while the limb is at the same time kept elevated. The "bootikins" of Horace Walpole, so strongly recommended and given away by him to all his gouty friends, seem to have been merely a fine bandage of *flannel*, applied moist and firmly over the limb, and then a roller of oiled silk over it, giving moist warmth like a poultice. Writing to Horace Mann, he says,—“You must put them on at night, and tie them as tight as you can bear—the flannel next to your flesh, the oil silk over. In the morning, before you rise, you must dry your feet with a hot napkin, and put on a pair of warm stockings freshly aired. Over the bootikins at night draw a pair of thread stockings” (*Life and Letters*, Vol. VII., p. 224). Dr. Wood uses a *warm* mixture of *tincture of camphor* with milk, applied by means of linen compresses, and frequently renewed.

Tincture of aconite may be similarly applied (ʒiiss. to ʒiv. of milk). The hypodermic injection of a fourth to a third of a grain of *muriate* or *bimeconate of morphia*, in any convenient part of the body, often relieves the local pain. *Blisters* have been recommended, and are of most advantage in asthenic chronic cases, when the inflammation has a tendency to linger in the articulations, and to cause liquid effusion. In the early fits of sthenic gout they are unnecessary; and are contraindicated in advanced gout, when the kidneys have become impaired, and also in cases of extreme gouty deposits (GARROD). As a general rule, however, the less that is done to the *local affection* the better. When the pain is very intense, relief may be given by the application of a *solution of atropine*, made by dissolving from *two to three grains of the alkaloid in a weak mixture of spirits and water*, and applying it to the inflamed joint by means of compresses of lint, preventing evaporation by a covering of oil silk (GARROD).

In the general treatment of gout bleeding is now generally restricted to two methods, namely—(1.) By leeches to the part where the inflammation rises so high, or is so chronic, as to threaten the patient with the permanent loss of the use of some joint; and to cases of metastasis of the disease to the stomach or other internal organ, when leeches are absolutely necessary; (2.) By general blood-letting. The following rules are laid down by Dr. Garrod as to the use of bleeding in gouty cases:—(1.) Never bleed with the idea of directly subduing gouty inflammation; (2.) Never bleed in advanced gout, or when the constitution is much weakened; (3.) If bleeding be required in order to relieve general or local plethora, abstract only as much as will effect that object, remembering that further depletion tends greatly to aggravate the disease and prolong its duration. Blood-letting produces a decided sedative action on the heart and other parts of the circulating system, and it has a most powerful influence upon the progress of gouty inflammation—an influence not merely limited to the removal of gout when it attacks the joints, but also of great efficacy in its marked and irregular forms, in the ophthalmia of gouty habits, in gouty bronchitis, and in headaches connected with the gouty diathesis (Sir H. HOLLAND, GARROD).

With regard to *Purgatives*, Sydenham, who was as great an enemy to purging as he was to bleeding, says,—“I am abundantly convinced, from much experience, that purging, either with mild or strong cathartics, whether used during the fit or in its declension, or in a perfect intermission or healthy state, . . . endangers the life of a patient by hurrying on the disease to the viscera, which were quite safe before.” The objection taken by Sydenham to purgatives was quite valid against those in use in his day, which were of the most drastic kind; but it may be laid down as a rule, that neutral salts are not only safe but efficient in relieving gout, though perhaps not to be depended on alone for its cure. The theory on which they are prescribed is, that the alkaline base of the neutral salt is absorbed, and combines with the insoluble urates deposited in the joints, forming a soluble sub-urate, which can readily be absorbed; and, again, more alkali being sent to the kidney, that organ is enabled to remove more uric acid, in a soluble state, from the system than under ordinary circumstances. The salts most in use are the *sulphates of magnesia* or of *soda*, and especially the former; and half a drachm to a

drachm should be given every eight, six, or four hours, according to the state of the bowels and the acuteness of the symptoms. It is also necessary to afford some relief to the patient from his excessive suffering. With that view an anodyne should be added, such as the *tincture of hyoscyamus*, *syrup of poppies*, or *some preparation of opium*. This method of treatment relieves the patient and shortens the paroxysm; but when the relief is complete, it should be abandoned, for sometimes a paroxysm of gout will return even under its use. *Colchicum*, or meadow-saffron, was long ago introduced as a specific in gout, of which the once popular "*eau mèdeicinale*" was supposed to be a preparation. *Colchicum* in some cases seems to be almost specific in its effects, and may be given as an *extract* or *tincture*, or as a *wine*, combined with some form of saline draught. Its mode of action is unknown. It very generally promotes secretion from the skin and kidneys; but it is useful even when it does not exercise any such physiological action. The quantity of uric acid and urea in the urine is said by some to be increased during its use (CHELIUS). Dr. Graves makes an opposite statement; and Dr. Garrod proves conclusively that colchicum does *not increase* the amount of uric acid. Dr. Laycock suggests that it may have a sedative effect on the vital actions going on in the tissues themselves, and so may arrest the formation of the gout poison. The wine of the root of *colchicum* is generally the form preferred, *ten to fifteen or to thirty minims being given every four, six, or eight hours*, and this remedy ought to be continued for some time in reduced doses, after all symptoms of gout have disappeared (BUDD). The bowels ought to have been freely moved before *colchicum* is given; and a full dose having been given at first, much smaller doses may be continued, as from ten to twenty minims two or three times a day, in Seltzer or Vichy water, or in other alkaline solutions, such as *bicarbonate of potash* much diluted, or combined with *iodide of potassium*, carefully watching its effects on the pulse, and never allowing sickness or depression to ensue. It may also be given with *carbonate of lithia*. Scudamore's mixture of *colchicum*, *magnesia*, and *sulphate of magnesia*, is said to be an excellent purgative in gout when a purgative is required. Although Dr. Garrod has shown that purging is not necessary to the action of colchicum; yet, there are many who believe that the action of colchicum is promoted by its combination with laxative remedies. Abernethy was wont to prescribe,—

R. *Sodæ phosphatis*; *Sodæ sulphatis*—āā ℥iv.; *Ammoniae sesqui Carb.*, ℥ss.; *Acid benzoici*, gr. xx.; *Vin. sem. Colchici*, ℥iss.; *Infus. Calumbæ*, ad ℥viii., of which a sixth part is to be taken every four or six hours.

Dr. Wood is in the habit of using it in the following formula in a draught:—

R *Magnesiae*, ℥ss.; *Magnes. Sulphat.*, ℥ss.; *Vin. Colchici. rad.*, ℥ xx.; *Aquæ Fluv.*, vel *Aq. Acid Carbon.*, f ℥iss.

Mercury in the form of "blue pill," or in the form of the "compound calomel pill" (*Pil. Plummeri*), followed by the draught just written, is useful where it is desired to act upon the intestinal secretions. But

mercury in any form must not be given if the urine contains albumen. If mere constipation is to be got rid of, *rhubarb pill*, *colocynth* and *scammony pill*, or *podophyllin*, may be advantageously used. The *iodide of potassium* has been much recommended by Mr. Spencer Wells. Alkaline remedies are of much value in the treatment of the paroxysms of the joint affection, and may be prescribed in the form of the *bicarbonate*, or of the *citrate* or *acetate* of potash. Dr. Garrod prefers the *bicarbonate*, and Dr. Parkes has shown that it increases the elimination of uric acid and organic solids by the urine (PARKES *On the Urine*, p. 298). Much benefit is also derived from the continuous administration of salines in small doses, repeated two or three times a day, in a *very* dilute form, and always on an empty or nearly empty stomach, and some little while before food (GARROD). If acute gout should have “retroceded,” as it is called, and the stomach or intestinal canal be inflamed, *leeches* should be applied to the abdomen or epigastrium, followed by a poultice, while the *neutral salts*, with the *tincture of hyoscyamus*, should be given at least every four or six hours. It is very rare that more active medicines are necessary.

For further indications regarding the management of gout, see treatment of the next subject—namely, *chronic gout*.

CHRONIC GOUT.

LATIN EQ., *Podagra Longa*; FRENCH EQ., *Goutte Chronique*; GERMAN EQ., *Chronische Gicht*; ITALIAN EQ., *Podagra Cronica*.

Definition.—*A persistent constitutional affection, characterised by stiffness and swelling of various joints, with deposits of urate of soda.*

Pathology and Symptoms.—The nature of chronic gout does not differ from acute gout, except as regards its *chronicity*. This chronicity is shown by the frequency of the attacks of gout, and by other persistent and permanent signs of the gouty constitution, especially referred to by Sir James Paget, as already noticed at p. 871, *ante*. The dyspepsia is especially persistent. It is more common in the gouty than in almost any other class of patients. It has prominently marked characteristic features. Patients suffer especially with acidity and flatulence, and many articles of diet habitually disagree with them, making them, as they vaguely express it, “*bilious*,” or giving them headaches. These offending articles of diet also frequently bring out some of the minor signs of gout with great rapidity. Many persons if they take beer, or champagne, or port, or any drink which is incompletely fermented, are almost certain to find on waking next morning that their knuckles are stiff and painful, or that they have burning palms or soles, or some neuralgia about the scalp. This development of certain symptoms after the use of certain articles of diet is indeed so uniform, that it affords a valuable test of the existence of the gouty constitution, just as the speedy effect of mercury or iodide of potassium indicates the presence of syphilis, or that of quinine the presence of ague. The tongue, too, is often characteristic. There are many persons who could tell almost certainly by the state of their tongue that they were about to be attacked with gout. It was not meant

that there was any one condition of the tongue that was met with in all patients alike; but that many men learnt by experience that when they found their tongue coated in a particular way, it indicated the coming of gout. Each man knew his own tongue in this sense. All Sir James Paget had been able to observe in these cases was, that a thin white fur was coated thinly over the whole tongue; yet some patients could tell by it, and tell truly, that their gout was at hand. There was one disease of the tongue, however, viz.—*psoriasis*, a condition in which the mucous membrane became smeared all over with a thickish opaque, white covering, which some had compared with a layer of mucus left behind by a snail tracking its way over wood. This condition of *psoriasis* might be frequently seen in syphilitic patients; and, it was very difficult by the characters of the disease itself, to discriminate between *syphilitic psoriasis* of the tongue and the *gouty*; but it might be taken that in the great majority of cases the disease depended on one or the other of these constitutional conditions (Sir JAMES PAGET, l. c.). Although distinct paroxysms are accompanied by less pain and fever than in acute gout, they last for weeks or months, and several joints are affected at once or in rapid succession. The chalky deposits described as a characteristic lesion, in and about the joints, are most common in cases of chronic gout. The swelling and redness develop very slowly. The redness is generally less intense and the swelling more diffuse and œdematous than in acute gout, and it does not subside with the desquamation of the cuticle. It continues, feeling at first soft and doughy, but latterly it contains firm nodules of variable size, and at last a hard mass of concretion is left, which is small in comparison with the swelling, the remains of which it represents. After repeated attacks, however, it grows by new deposits, and may eventually attain a considerable size; and the continued irritation of these deposits causes pain, difficulty of motion, and great deformity of the parts implicated. Phlegmonous inflammation is common; abscesses sometimes occur about the joint, and the pus contains masses like soft mortar, from the breaking up of these chalky concretions. Observations on body-temperature are wanting alike in acute gout and in chronic gout. The febrile phenomena may be characteristic. Chronic gout may persist at intervals for fifty years; but in some cases the paroxysms become frequent and irregular, so that instead of occurring annually or half-yearly, only a month or two may elapse between the attacks. These attacks are generally more prolonged than in acute gout, and a fresh paroxysm may ensue before the subsidence of the preceding one, so that the patient is scarcely ever free, except in a few of the summer months (GARROD). Sydenham thus describes an attack of chronic, or, as he terms it, "*inveterate gout*:"—"After yawning, especially in the morning, the ligaments of the bones of the metatarsus are violently stretched, and seem to be squeezed with great force, as if with a strong hand. And sometimes, though no yawning has preceded, when the patient has disposed himself to sleep he feels a blow on a sudden, as if the metatarsus were breaking in pieces, by a large stick, so that he wakes crying out with pain. The tendons of the muscles of the tibiæ are sometimes seized with so sharp and violent a convulsion or cramp, that if the pain it occasions were to last only a short time it could not be borne with patience." The succeeding paroxysms, after many

racking pains, become less painful, when, "instead of the usual external pain, a certain sickness, a pain in the belly, a spontaneous lassitude, and sometimes a tendency to diarrhoea succeeds." Besides the pain and sickness, the patient becomes lame, and almost incapable of motion, and is perhaps obliged to be wheeled or carried from room to room; and he is not only reduced to this helpless condition, but, to complete his misery, his mind sympathises with his body. "For every paroxysm may be justly termed a fit of anger, the rational faculties being so enervated by the weakness of the body as to be disordered on every trifling occasion, whence the patient becomes as troublesome to others as he is to himself." Gouty disease of the veins—gouty phlebitis—is "undoubtedly a very common affection. It is by far the most frequent form in which anything that could be called idiopathic phlebitis occurred. It was perhaps one of the most frequent of the irregular forms of gout; and any case of phlebitis occurring without an apparent cause in an elderly person might be suspected to be of this nature, particularly if the attack were very sudden" (Sir JAMES PAGET, l. c.). Another form of chronic gout is known as *atonic gout*, when the joints enlarge, and the tissues and ligaments become thickened, and the seat of various effusion, so as often to distend and even to dislocate the bones; and yet, if the patient be kept quiet, he suffers no pain. The debilitated organism is believed to be in a condition too feeble to develop a normal *sthenic* or *acute* attack of gout. The general symptoms, however, are most distressing. The permanent constitutional diathesis is generally apparent, by the excessive general hyperæsthesia, muscular weakness, and dyspepsia, increased perspiration and thick cloudy urine. The patient suffers from loss of appetite, indigestion, sickness, nausea, flatulence, acid eructations, pains of the stomach, cramps in the legs and in various parts of the body; also great dejection of spirits, vertigo, palpitation, fainting, asthma, and also, perhaps, from stone or gravel. The most insignificant causes, such as errors of diet, excitement, exposure to cold, changes of weather and the like, will bring about these general constitutional symptoms, accompanied sometimes with pains in one or more joints, resembling commencing attacks of gout. These affections, in some form or other, continue with occasional intervals during the remaining life of the patient, who believes that he has the gout *flying* about him, and that he should be well if he had a regular fit. In the course of this disease there may also be a metastasis to the stomach or other part, and the affection is then termed "*retrocedent gout*," the pain in the joints being trifling, or having entirely subsided. This form is the *arthritis metastatica retrograda* of the older authors, whose belief in this form of gout is not only supported now by such experiments as those of Hoppe-Seyler and Zalesky, already referred to, but by clinical observation also. The term is "*applied to cases of gout in which some internal organ becomes affected on the disappearance of the disease from the joints, and is referable either to acute or chronic gout*." The organs most frequently affected are the *stomach, brain, and heart*. When the metastasis is to the *stomach* or *intestines*, *gout in the stomach* may be either of a spasmodic or inflammatory character. The spasmodic is the most frequent. The patient is seized with violent pains in the stomach (*cardialgia*), with faintness, coldness of the extremities, and a quick, small, and scarcely perceptible pulse, accompanied with much flatulence, acidity,

or vomiting. If, on the contrary, the attack be of an inflammatory character, the pain is perhaps equally great, but is increased on pressure, and there is more reaction, some fever, a fuller pulse, with vomiting, and sometimes with *hæmatemesis*, and perhaps obstinate constipation. *Gout affecting the encephalon* may sometimes present the phenomena of an apoplectic seizure, or be indicated by severe circumscribed headache, giddiness, and vomiting. *Gout affecting the heart* may induce irregular and feeble action of that organ, associated with disturbed circulation, dyspnœa, and fainting. Such cases are usually attended with gaseous distension of the bowels, which tends to increase the difficulty of the heart's action. *Gout affecting the spinal canal* may induce sudden paraplegia. The duration of these attacks is short, as the patient must be quickly relieved or quickly perish. Besides metastasis to the stomach and intestines, this retrocedence may take place to other parts—as to the testicle, bladder, or rectum. The transition of the gouty virus is often marked by a pain shooting along the nerve, as sudden and as rapid as a galvanic shock, and so violent as to have been compared to stabbing with a knife. It is only when gout runs its course with unusual symptoms, when the patient suffering from gouty inflammation of the joints suddenly is attacked in some of these inward parts, especially when the gouty affection of the joint is subsiding or developing, or terminating by copious excretions of urates by the kidneys, that we are warranted in considering such attacks as “retrocedent gout.” Besides being thus a migratory disease from part to part, gout often alternates with other chronic diseases, such as *asthma* and *rheumatism*, and may co-exist with them. A condition may also be brought about by the gouty poison which it is difficult to separate from ordinary pyæmia—the disease commencing as gout (Dr. G. O. REES, l. c.).

Treatment.—In chronic gout the treatment is the same as in acute gout; but, as next to hereditary predisposition, a disproportion between the amount of food and drink taken and the necessity for it, is a great cause of the disease, means ought to be taken to regulate the diet and promote healthy metamorphosis of tissue during intervals of freedom from gouty paroxysms. The habits of the patient ought to be regulated by written rules for him to abide by, if he would be free from gout. The form, the quantity, and the quality of the food ought to be precisely prescribed. Most gouty patients eat too much. Vegetables, with soups and meat, must be allowed only once a day—not oftener. Beer, wine, and alcoholic fluids generally are injurious, and must be forbidden to gouty patients, as they retard the metamorphosis of tissue. The same is true of tea and coffee. Water, pure water only, taken in quantities as large as possible, promotes the metamorphosis of tissue to a greater extent than any remedies we know of. The drinking of a certain *extra* quantity of water is essential for the maintenance of health in gouty patients. It ought to be taken in the early morning—to the extent of half a pint while dressing in the morning—and before any solid food is taken. Its use does not lessen appetite for food—as is the case with beer, wine, spirits, tea, and coffee. It promotes the flow of fluid by the kidneys, and increases the excretion of *urea*. Combined with muscular exercise, the use of pure water hastens, to the greatest possible extent, the transformation of tissue; and combined with the use of certain mineral waters and baths, a connecting link is

established between the dietetic and medicinal treatment of gout. Whenever an excess of *lithic acid* or of *lithates* is seen in the urine, some alkaline water should be taken in the morning, instead of plain water; and enough should be taken to render the urine clear and free from deposit. But strong alkaline waters are not to be prescribed in large quantities, else too great depression and a tendency to impair the digestive power of the stomach will be the result. In moderation, the *milder* alkaline waters taken as a morning draught are of value. The mineral springs which exercise the most favourable influence on the gouty constitution are those of Aix in Savoy, Bath, and Buxton, Ems, Gastien, Homburg, Karlsbad, Kissingen, Marienbad, Neuenahr, Pfäfers, Schwalbach, Pyrmont, Spa, Toplitz, Wiesbaden, Wildbad, and Vichy. Dr. Garrod lays down the following general rules as a guide to the use of them:—(1.) Their use should be prohibited when there is much structural disease in any important organ, especially in the heart or kidneys; and even when organic mischief is slight, the greatest caution in their use is necessary. (2.) They are to be avoided when an acute attack is either present or threatening. (3.) The particular mineral water must be selected according to the nature of the individual case; for the robust, and those of full habit, the alkaline saline springs should be chosen; when torpidity of the bowels predominates, the purgative waters may be used; when the skin is inactive, the sulphur springs should be used; and when much debility prevails, or an atonic state exists, then the more simple thermal springs may be prescribed.

These waters contain either *alkaline carbonates*, *chlorides*, or *sulphates*. Some are impregnated with *sulphurated hydrogen*, and others contain *iron*; many, like Wiesbaden, Karlsbad, and some of the Vichy springs—Wildbad and Neuenahr—are of an elevated temperature.

The beneficial influence of these natural mineral waters is especially perceptible in the reduction of plethora by the alkaline springs, and in the regulation of the functions of the bowels and skin. The springs of *Vichy* are all rich in *carbonate* or *bicarbonate of soda*—about forty grains to the pint. Some have a temperature of 101° Fahr. In moderate doses they cause the urine to become alkaline or neutral; and used as baths, they have a similar effect. They influence especially the functions of the liver and the skin, and are more adapted for the robust and persons of full habit of body; but if the system is enfeebled by frequent attacks of gout, or if there is a tendency to the formation of chalk concretions, the waters of Vichy are not to be recommended. The waters of Wiesbaden contain a large amount of *chloride of sodium*, and are as high in temperature as 160° Fahr. They are more highly stimulating and more generally debilitating than those of Vichy, and are of service in those cases where the circulation is slow and the secretions deficient. The springs of Neuenahr range in temperature from 90·5° to 104° Fahr., and contain nearly 50 per cent. by volume, of free *carbonic acid*, with a considerable quantity of *soda salts*, chiefly as carbonates. They stimulate the circulation, augment the excretions from the kidneys and the skin, increase the appetite, and diminish stomachal acidity. They are unsuitable for plethoric habits and where the liver is enlarged; but where the constitution is impaired or enfeebled, they are of great service. The waters of Wildbad, in the Black Forest, have also a high temperature—92° Fahr.

—with much free *carbonic acid*, holding *salts of soda*, and *magnesia*, and *iron* in solution. They are alkaline, and their influence is similar to the springs of Neuenahr. The waters of Aix are slightly saline, and of a temperature as high as 135° Fahr., containing *chloride*, *carbonate*, and *sulphide of sodium*, with free sulphurated hydrogen. They stimulate the liver, kidneys, and skin, and are indicated where there may be sluggish action of these functions, and where the joints are stiff. The Karlsbad waters are rich in *sulphate of soda*, with *carbonate of soda* and *common salt*. Their temperature is about 167° Fahr. They are purgative, diuretic, and diaphoretic, and are beneficial where there is constipation, with congestion of the liver; and are unsuited for feeble patients. The waters of Baden-Baden contain *common salt* combined with *iron*, and are also rich in *lithia*. Toplitz, Gastien, Buxton, and Bath waters are also all somewhat elevated in temperature, and are peculiarly adapted for the treatment of gout in the infirm, feeble, and old patients. The chalybeate waters of Spa and Pyrmont are most suitable for anæmic patients and in the asthenic forms of chronic gout. The *salts of lithia*, so abundant in the waters of Baden-Baden, were originally proposed by Dr. Garrod as a remedy for gout. They have since been extensively used, and found generally beneficial. They abound also in the Karlsbad, Aix, Marienbad, and some of the Vichy waters. The *salts of lithia* are actively diuretic, powerful solvents of *uric acid*, and generally they tend to render the urine neutral. Taken for a length of time in a dilute form, they tend to prevent paroxysms of gout in chronic cases, and cause solution and absorption of chalky matter. They certainly lessen the deposit of *water* and *uric acid* in the urine; and are of service both in the acute and chronic forms of gout. The dose is from five to ten grains of the *carbonate of lithia*, dissolved in aerated water, three or four times a day; of the *citrate*, eight, ten, or more grains may be given (GARROD, FLINT, CLYMER). *Iodide of potassium* is a remedy of very great value, especially in cases where “the pains are increased at night;” but the *bromide of potassium* is still more useful. It seems more highly anodyne and soothing. The *iodide of potassium* is useful in removing the recent thickening in the tissues round joints; and of gouty inflammation, when fluid is effused into the cavities of joints, and is slow of absorption. *Guaiacum* is especially useful in the asthenic gout of old subjects, and to younger patients also it is beneficial. A combination of *quinine*, *iron*, and *arsenic* is often of special service, in very minute doses, in the form of *chlorides*; and it is advantageous to unite small doses of *colchicum* with the *quinine*.

The patient generally receives some warning by which he may try and ward off the gouty seizure. When such warnings are recognised a combination of *quinine*, *colchicum*, and *colocynth*, taken twice or three times a day, is an excellent medicine. The pills of Becquerel are also recommended by Trousseau. They consist of:—

R. *Quiniae Sulphatis*, gr. xx.; *Ext. Digitalis*, gr. iii.; *Ext. sem. Colchici*, gr. x. Divide into ten pills, of which two or three may be taken daily for three or four days.

Laville's *anti-gout liquid and pills* are French preparations, and are so

popularly known and so much esteemed by many sufferers, who have used them with excellent effect, that they challenge attention, although they are open to the objection of being *secret* as to their composition, like *chlorodyne*, *James' powder*, and *Warburg's drops*. According to Dr. Tanner, "from an analysis, the *liquid* contains the active principle of *colocynth*, *quinine*, and *cinchonine*, with unimportant salts of *lime*. It is used at any period of the attack; a teaspoonful being taken in sweetened water or tea, and repeated in six hours, if the pain continue and the bowels be not moved. Twenty-four hours are to elapse before the next dose, when half the quantity is to be taken two or three times daily, unless the bowels are irritable. The pills consist of *physalin* mixed up with *silicate of soda* and powdered *chamedrys*." One is taken just before a meal, for several weeks. In the chronic forms of the disorder, where there is no organic disease of the brain, heart, or lungs, *wet-packing*, and the *Turkish bath*, cautiously and occasionally used, but not during a paroxysm, under the immediate supervision of the attending physician, would sometimes seem to be of service, in lessening stiffness of the joints and restoring or improving the action of the skin (CLYMER). Trousseau speaks well of the *wet-packing*. Hot salt-water baths are also beneficial. The inhalation of oxygen when followed by clear urine, has also given great relief, and in some cases cures have resulted.

In *atonic* gout some light tonic medicine may be given, as *five to ten grains* of the *citrate of iron*. A large number of chronic cases, however, though the general health is improved by this treatment, are often altogether unrelieved as to the local symptoms, and are often quite unable to assist themselves. In these instances the *terebinthine* remedies appear to be beneficial, as *spruce beer*, and *Canadian balsam*; or, one drachm of the *oil of turpentine* may be taken in an effervescing draught once or twice a day. Sydenham's method of treatment by *manna* may also be used. He recommends, from experience in his own case, large doses of *manna* in all cases of what he terms "bloody urine." If the chronic or *atonic* gout should become retrocedent, and the stomach and intestinal canal be the seat of the spasmodic form of the disease, Sydenham strongly recommends that *laudanum* should be given; but perhaps the following draught is more efficacious, namely—

R. *Aquæ Camphoræ*, ʒx.; *Sp. Ætheris Sulphurici*, ʒi.; *Sulphatis Magnesicæ*, ʒss.

It will remove from the stomach any undigested matter which may remain as an irritating cause. This should be given every hour till the patient is relieved; and while it is being prepared, hot brandy and water should be freely administered, or the *spiritus ammoniæ aromaticus*, in doses of sixty minims, and hot cloths applied to the abdomen, as well as hot bottles to the feet.

As indiscriminate feeding appears to have a great influence in the production of gout, so we expect the regulation of diet should have great influence in its removal. During the fit the diet should consist of slops and light puddings, and afterwards white fish, till the paroxysm has terminated. Chronic gout is so distressing that many persons are inclined

to diet themselves with great strictness during the interval. Sydenham says that a milk diet, or drinking milk as it comes from the cow, or boiled, without adding anything to it, except perhaps a piece of bread, once a day, had been much used for twenty years past in his time, and had done much service to gouty patients. But on quitting it, and returning to the mildest and tenderest diet of other persons, gout has immediately revived; and he adds, that many cannot bear this regimen. An entirely water regimen he considers hurtful. The most digestible meats, such as mutton, well-kept beef and poultry, with the white kinds of fish, as codfish, sole, and whiting, may be eaten; but salmon, veal, and pork are to be avoided, as well as cheese, salads, highly seasoned dishes, and rich sauces, or other "elaborate preparations on the part of the cook." The use of alcoholic stimulants requires great caution. For those who inherit the gouty constitution, total abstinence should be made a rule absolute in youth; and for all others, unless some obvious necessity arises for their use and prescription by the physician, when the nature and quantity of the stimulant must be definitely stated, and measured out as any other medicine would be. If alcohol in any form is required, it may be taken as weak brandy, gin, or whisky and water, or pure dry sherry, like Amontilado or Manzanilla. Port, Burgundy, and sweet wines must be avoided; but wines of the Rhine vintages may be taken, if they do not contain a large percentage of alcohol. The wine should be of the highest quality, and fully matured and mellowed. Really good wines are generally used to advantage, and the vegetable extractions of the pure, mature, and mellowed wines are easy of digestion by the gouty stomach. The best spirit is whisky, which should also be very old, soft, and mellow, and free from the smoky flavour. All of these spirits ought to be taken much diluted with water. The patient should go early to bed, keep the mind free from all disquietude, live with the greatest moderation, clothe himself warmly, and ride on horseback. When exercise cannot be taken, friction over the surface of the body is exceedingly useful. He should be rubbed down with a flesh-brush once or twice a day, just as a horse is groomed. Turkish baths taken regularly once a week, or as often as may be indicated by the medical attendant, are most beneficial. The custom of going to bed for an hour or two immediately after the bath—as in Germany—adds greatly to the good obtained from the bath; the body being covered and completely at rest, the nervous system is tranquillised, and the skin acts freely. But much harm may be done by excessive limitation of the supply of nourishment, as well as by the sudden and complete abstraction of spirituous fluids that had been used for years; as well as by other debilitating courses of treatment set about too hastily and pursued too vigorously. If the patient is cachectic, debilitating treatment will make him worse. "There should be a due admixture of animal and vegetable food; it is an error to suppose that an animal diet tends more to the formation of uric acid than a vegetable one. The tortoise, feeding on a simple lettuce, excretes a large quantity of urate of ammonia, far more in proportion to the weight of the animal than is excreted by the dog exclusively nourished with meat. Vegetables, as potatoes, greens, and the like, may be partaken of with advantage; the soluble salts they contain are of value in keeping up the activity of the secreting organs. The same

remarks hold good with regard to soft fruits when eaten in moderation, as strawberries, grapes, and oranges; also other fruits when stewed or baked, as apples and pears; but these latter, as likewise plums, and stone-fruit in general, should be avoided in a raw state. Extreme moderation should be exercised when saccharine fruits are eaten, as sugar is liable in many subjects to lead to the production of acidity. The same precaution is necessary in reference to the addition of sugar to other articles of diet" (Dr. GARROD). "Gouty folks should be very moderate in all their food—not, however, too abstemious, for such patients are seldom really vigorous, or able to dispense with a nutritious diet. Moderation should be observed, particularly in respect to the use of stimulants; and each patient must select for himself, or be advised to take what suits him best. There is a prevalent opinion that spirits are better than wines; but this is doubtful. The chief point is, that whatever stimulant is taken it should be used in the most strict moderation" (Sir JAMES PAGET, l. c.).

One other point with regard to the treatment of the patient during the fit is, that if it be necessary to move him, either on account of his restlessness or other cause, this should be done with great care and tenderness by the attendants; for although the pain may be latent while the parts are quiet, yet the least shock often causes the most excruciating agony. The irritable state of mind of the patient during the paroxysm has been mentioned; and it is well known that slight moral causes will often produce a fit, while powerful emotions have sometimes cured one. It is quite essential, therefore, that the minds of gouty patients should be kept as tranquil as possible, both for their own sakes as well as for the comfort of those who minister to their needs.

CHRONIC OSTEO-ARTHRITIS—*Syn.*, CHRONIC RHEUMATIC ARTHRITIS.

LATIN EQ., *Osteo-arthritis Longa*—Idem valet, *Arthritis Rheumatica Longa*; FRENCH EQ., *Arthrite Rhumatismale Chronique*; GERMAN EQ., *Chronische Deformirende Gelenkenzündung*; ITALIAN EQ., *Osteo-artrite Cronica*.

Definition.—*An affection characterised by pain, stiffness, and deformity of one or more of the joints, associated with the deposition of new bone round them.*

Pathology and Symptoms.—By some this affection has been regarded as a form of *chronic articular rheumatism*; others regard it as essentially different; and although the College of Physicians have retained a synonym to indicate the connection of the disease with rheumatism, they have placed the affection after gout, and not after rheumatism. It seems also to have been one of the numerous affections comprehended under the name of *rheumatic gout*—a term which has been advantageously omitted in the nomenclature of the College of Physicians. "It is regarded by the most competent observers as one conveying altogether an erroneous impression of disease; and it has not been mentioned as a synonym, because it was found that any two members of the committee were not in the habit of hearing it applied to the same form of affection" (*Med.-Chir. Review*,

1869, p. 365). The disease may attack any joint indiscriminately, when it becomes so swollen and misshapen that the term "*arthritis deformans*" has also been given to the disease. The articular inflammation generally commences with the synovial membrane; the synovial capsule and ligaments, the cartilages and ends of the bones becoming involved in lesions, which are characteristic of the disease (CRUVEILHIER, BRODIE, ROBERT ADAMS). The articular cartilages and surface of the bone eventually disappear, an induration of the central parts of the joint succeeds, followed by an extensive germination of new bone (*osteophytes*), which grows round the peripheral portion of the joint ends of the bones, giving them a very ragged appearance. The ends of the bones are in immediate contact by smooth articular surfaces, without any intervening cartilage. The disease develops very slowly—most commonly between the twentieth and fortieth years of life; but it may begin late in life, and even in advanced age. Affections of the smaller joints are more common in women than in men (HAYGARTH); but men more frequently suffer in the larger joints, especially the hip, and the disease is much more common among poor people than among the well-to-do classes.

At the commencement of the disease there is considerable pain in the joint, sometimes so severe as to prevent sleep at night. The pain is also increased on pressure and on movement of the joint; and if the hand be laid on the joint when it is moved, a crackling or crepitation may be felt, the joint being almost dry, or containing a very small amount of synovia. The disease generally begins in both hands, and passes to the feet. Great deformity takes place, from subluxation of joints, enlargement of the epiphysal ends of the bones, and destruction of the articular cartilages. The fingers ultimately are flexed on the metacarpal bones, and drawn over *seriatim* to the ulnar side of the palm, so that the fingers lie over each other. These characteristic enlargements of the joints acquired originally for the disease the name of *nodosity* of the joints (HAYGARTH), affecting chiefly the hands and feet; and more recently the term *rheumatic gout* has been most commonly applied to it. When the larger joints were implicated in a similar morbid process, the disease was then generally considered to be *chronic rheumatism*; and when seated in the hip-joint, as is usually the case in old people, the disease was described under the name of "*morbus coxæ senilis*." Now, the name placed at the head of this subject is that which is meant to comprehend all these forms of the disease. Chronic osteo-arthritis sometimes succeeds chronic rheumatism, when, as a rule, many joints are affected, and the disease is no doubt influenced by the constitutional diathesis of rheumatism, and there is generally a great resemblance to rheumatism in the phenomena of this disease; but the table on the following page, based upon that by Dr. Garrod, and published at page 544 of his valuable work on gout, will show the characteristic differences between *gout*, *rheumatism*, and *chronic osteo-arthritis*, which last name I have substituted for *rheumatic gout* and *rheumatic arthritis* in Dr. Garrod's table.

Treatment.—*Chronic osteo-arthritis* must not be treated either as gout or as rheumatism. *Colchicum* is considered by Dr. Garrod to be generally injurious, and so also is alkaline treatment. The course of management may be shortly indicated as follows:—*Gregory's powder* at bedtime, and

TABLE EXHIBITING THE DIFFERENTIAL DIAGNOSIS OF GOUT, RHEUMATISM, AND CHRONIC OSTEO-ARTHRITIS (Dr. Garrod).

GOUT.	RHEUMATISM.	CHRONIC OSTEO-ARTHRITIS.
Strongly hereditary.	Less so than gout.	Less so than gout, if at all.
Much more frequent in males.	As frequent in females.	More frequent in females.
Seldom occurs before puberty, generally much later.	More frequent in the young and before middle age.	Occurs both in young and old.
Induced by gross over-feeding, wine, and malt liquors.	Occurs in the weak; not caused by wine, &c.; excited by cold and damp.	Often induced by depressing causes, and sometimes excited by cold.
One or more of the smaller joints particularly affected in early attacks, especially great toe.	Large joints, usually several, more affected than small.	Large and small joints about equally affected.
Great pain, shiny skin, œdema, swollen veins, and desquamation of cuticle.	Pain less intense; seldom œdema. No enlargement of veins, nor œdema.	Less pain; much swelling, and often some œdema.
Does not induce acute inflammation of the structures of the heart, but affects stomach, brain, and kidneys, and causes nervous disturbance of heart.	Often causes acute pericarditis and endocarditis, and pulmonary inflammation.	No tendency to cause heart or other organic disease.
Febrile disturbance moderate; much constitutional disturbance; morning remissions.	Febrile disturbance great, more than from local inflammation, and continuous.	Little febrile disturbance.
Paroxysms periodic in early attacks, and early attack short, lasting but a week or ten days.	Attacks not periodic, and generally much longer.	No periodicity; duration of attacks indefinite.
Blood rich in uric acid.	No uric acid in blood.	No uric acid in blood.
Perspiration not peculiar.	Perspiration profuse and acid.	Perspiration not peculiar.
Constant deposit of urate of soda in inflamed cartilages and ligaments.	No deposit of urate of soda.	No deposit of urate of soda; ulceration of cartilages.
Often leads to kidney disease (gouty kidney).	No tendency to cause kidney disease.	No tendency to induce kidney disease.
Often produces chalk-stones externally, as in auricles.	Never causes chalk-stones.	No chalk-stones produced, but swelling of joints.

more active aperients at regular intervals, are required as a necessity. The action of the skin ought to be encouraged by hot-air baths, the frequent use of the *Turkish bath*, and *Dover's powder* at bedtime. *Warm bathing* is generally attended with relief, and recourse should be had to the mineral springs of high temperature, already mentioned at p. 884. Schwalbach, Spa, Wildbad, Neuenahr, and Tunbridge Wells, are the best. *Guaiacum*, if borne by the stomach—combined or not with *sulphur*, and taken as an electuary in *glycerine*—is of great assistance (GARROD). The free use of *dilute phosphoric acid* is recommended by Mr. William Adams. *Iodide of potassium* is also well spoken of. Whichever of these remedies may be considered suitable for any particular case, it ought to be steadily persevered in for several weeks. Warm clothing and residence in a warm climate, during the winter months, are necessary elements for increasing the comfort of the patient. If much emaciation has resulted, cod-liver oil and fats must be prescribed, combined with the usual tonic remedies—*quinine*, *iron*, *strychnia*, *arsenic*, and mineral acids—according to the nature of the case. The local remedies must be regulated by the surgeon. Chief of these Dr. Julius Althaus has used with advantage that most active stimulant of nutrition—the *continuous galvanic current*. It acts as a general tonic to the system, more especially to the nervous system; and relieves one of the chief complaints, want of sleep, when *hydrate of chloral* fails. The best mode of application for this purpose is to direct the positive pole, armed with a conductor of large surface, to the cervical spine, while the negative pole, similarly armed, is placed on the pit of the stomach. A gentle current, which even patients of exquisite sensibility can well bear, is thus allowed to flow for from three to five minutes in the direction just described. By this application, the base of the brain, the spinal cord, and the coeliac plexus of nerves, are simultaneously brought under the influence of the current. It also relieves pains, for which object the constant current must be applied to the suffering parts, so that the positive pole, armed with a small electrode, is made to touch the sore points; while the negative, connected with a large electrode, is placed in the neighbourhood. The current used may in this instance be somewhat more powerful than where it is employed for general tonic effects; and if a short application, say of one or two minutes, be not successful, this may be extended to four or five minutes. The effect is, in many instances, almost magical, inasmuch as pain, which has been fixed in some particular spot for months and years, is, as it were, charmed away by one or two applications. If such treatment is followed up for a sufficient length of time, even extensive deformities may be improved (*Brit. Med. Journ.*, Sept. 28, 1872).

SYPHILIS.

LATIN EQ., *Syphilis*; FRENCH EQ., *Syphilis*; GERMAN EQ., *Syphilis*;
ITALIAN EQ., *Sifilide*.

Definition.—*The result of a specific living poison; or, of "a something" of an uncertain nature, but capable of self-multiplication, which breeds in the blood and tissues, which is introduced into the system solely by contagion, inoculation,*

or implantation on some part of the body of a healthy person—i. e., a not yet syphilitic person—generally through an abrasion or sore consequent on sexual intercourse with an infected person (acquired syphilis), or by inheritance (congenital syphilis). Three weeks or a month after contagion and absorption of the poison, a peculiar series of phenomena begin to show themselves, which mark the general infection of the system, namely:—First,—a distinct specific fever is followed by or attends the development of, Second,—the principal anatomical signs of general infection, which consist of—(a.) induration (specific) round the spot where the virus has been implanted; (b.) induration of the lymphatic system of glands; (c.) the development of a cutaneous and mucous exanthem; (d.) the formation of nodes or gummatous nodular tumors in the connective tissue generally, and especially in that of the true skin, bones, mucous membranes, and solid visceral organs—e. g., liver, brain, lungs, and heart—(visceral syphilis); (e.) a cachectic condition of the system which follows, and accompanies the phenomena of infection; and, (f.) indurations which may remain in the form of hardened fibrous tissue in various parts of the body for an indefinite period of time. The whole course of the disease is characterised by slow, but progressive development, often marked by periods of recrudescence.

Pathology.—(a.) *History.*—Venereal sores, commonly known as chancre and gonorrhœa, were described in Chinese systems of medicine 4,500 years ago; also in Hindoo, Arabic, Greek, and Latin literature. Towards the close of the fifteenth century a great epidemic, believed to be syphilis, pervaded Europe, especially Italy (1490 and 1500); and our first knowledge of syphilis as a separate and distinct disease dates from that period. Physicians regarded it as a new disease; and they traced its origin to the army of Charles VIII. of France, whose Spaniards carried it from Spain (as did also the army led by Gonzalo Hernandes de Cordova) into Italy in 1495, for the disease was in existence in Spain before the campaign of Charles VIII. Descriptions and historical notices extant also make it probable that the disease was not unknown in certain parts of Europe before the return of Columbus from the West Indies. At the end of the fifteenth century various circumstances combined to favour a great general epidemic outbreak. Crusades and pilgrimages set the people in motion, and established intimate relations between nations and sexes. During the campaign of Charles VIII., the disease spread in Italy, Spain, France, and Germany—a spread which was extremely rapid during the last ten years of the fifteenth century; but it was not recognised as a special disease until the close of that period (BÄUMLER). It was known as the “Great Pox,” to be contagious, and to be communicated most readily during sexual intercourse. In the sixteenth century syphilis was clearly recognised as the result of a specific poison or virus. It was believed to be capable of combining with all other diseases, and so to modify them as to give them new forms. Even at that early period in medical history, syphilis was recognised as producing phthisis, diarrhœa, dropsy, skin diseases profoundly affecting the constitution, and demonstrating the presence of a poison in the system by remote general symptoms of ill-health (PARACELSUS). Towards the close of the seventeenth century the ulterior results of venereal disease were also fully recognised; but they were

believed to be due to bad treatment. Van Swieten taught that *no organ* escapes the influence of the venereal poison. He recognises it as the source of gummy tumors, exostosis, deep-seated pains, apoplexy, epilepsy, blindness, deafness, and paralysis.

There can now be no doubt that there are several distinct diseases which, from their most common origin in sexual intercourse, have been conveniently grouped under the name of "*Venereal diseases*," namely:—(1.) *Gonorrhœa*; (2.) *Contagious ulcers of the genitals, chancroid or soft chancres*, with suppurating bubos; (3.) *Syphilitic ulcer or hard chancre*, with indurated non-suppurating glands. From time to time it has been a subject of discussion, "Whether these *several affections* are due to one and the same virus, whose action is modified by admixture with secretions, or by peculiarities of constitution on the part of the recipient?" or, "Whether a separate specific poison exists for each form of venereal disease?" In 1767, Balfour maintained their non-identity. This latter alternative is now proven to be true; and the following are the classes of venereal affections which are specifically distinct:—(a.) *Gonorrhœa*; (b.) "Simple" soft "non-infecting" chancres, ulcers, or sores; (c.) "Infecting" hard chancres, papules, ulcers, or sores; (d.) Mixed chancres—the combined result of the virus of (b.) and (c.); (e.) Subsequent lesions retaining specific powers of contagion, especially some local forms of syphilitic lesions.

The history of the identification of the separate poisons which give rise to the several venereal affections, of which they are the cause, arranges itself into three periods as to time, comprehended in the medical records of the past century.

I. *The Period of Hunter* (1787), who taught the doctrine (now known, and shown before by Balfour, 1767, to be an error), "That the various forms of syphilis and gonorrhœa depend upon one and the same poison; that the matter or virus produced in both is of the same kind, and has the same properties. He believed that he had established, by experiment and observation, that the discharge from a gonorrhœa will produce either a gonorrhœa, or a chancre, or the constitutional affections of syphilis—and that the matter from a chancre will indifferently give rise to either of these venereal affections. Hunter rested his belief and his doctrine mainly on an experiment on himself. He dipped a lancet in the venereal matter from a gonorrhœa. He made two punctures in the tissue of his own penis with the lancet so charged. One inoculation he made on the *glans*—the other on the *prepuce*. Two distinct results followed, *each of them marked by a distinct and specific period of incubation*. The inoculation on the *PREPUCE* was followed by itching from the *third to the fifth day*. On the fifth day the site of the puncture was red, thickened, and swollen. A speck became visible; and in a week this speck had commenced to suppurate, the urethra at the same time indicating the commencement of a discharge. The inoculation on the *GLANS* was followed by itching *fourteen days after the puncture was made*: three days later a speck appeared where the puncture had been made. The speck became a papule, then a pimple, and ultimately discharged yellow matter. The sore on the prepuce broke out several times after it healed up; but the sore on the glans never

broke out again after it healed. The secondary lesions of syphilis followed this experiment, demonstrating the "infecting" nature of a virus with which he had been inoculated. Ulceration of the throat commenced in due time, and copper-coloured blotches on the skin followed in the usual sequence. The time the experiment took, from the first infection to the complete cure and elimination of the poison, was three years. With the knowledge of syphilis which we now possess, can we say from which of these sores the constitutional disease arose? Hunter believed he had inoculated the discharge of a specific gonorrhœa *only* and *alone*: but two important questions suggest themselves, concerning which Hunter does not enlighten us, namely:—Had the person a concealed infecting chancre from whom Hunter took the virus? Was the patient suffering from constitutional syphilis at the time he had a gonorrhœa? Besides Hunter, Carmichael in this country taught the same pluralistic doctrine, and Cazenave in France; but in neither country did the doctrine obtain any wide acceptance.

II. *The Period of Ricord* (1831), who established, by numerous experiments repeated in various ways,—(1.) That the inoculation of gonorrhœal discharge on the skin is followed by no specific result; (2.) That *at least two*, if not *three*, distinct poisons exist—namely, *one virus* which would produce a gonorrhœa; a second *virus* which would give rise to a specific ulceration, called a chancre; which he observed to follow a very definite course. It commenced, as a rule, within twenty-four hours after the inoculation of the poison. A pustule formed, which, breaking, a *soft* or suppurating chancre was the result. Ricord, however, eventually recognised *two* classes of chancres—the *soft* and the *hard*; and he described them as originating in the same way, namely—by contagion from a similar primary sore. His experiments were of one or other of two kinds: either they were made on persons who had been already affected by syphilis (involving a most vital fallacy in drawing conclusions regarding the nature of syphilis), or on persons concerning whom it was not ascertained whether they had been infected with syphilis before or not. Hunter had already shown that the secretion from *one* kind of syphilitic sore is not capable of being inoculated on the same body that produced it; and now we know that the discharge from the "*infecting*" sore cannot be inoculated on the already infected person. Ricord has further shown that the *plastic lymph*, the increased growth of tissue round a true chancre—the *specific sclerosis* or induration—does not take place a second time on the same subject; while Sigmund and many other observers are now agreed that the "*infecting*" disease does not repeat itself. This fact is analogous to the freedom from farther attacks of *scarlet fever*, *measles*, and *small-pox*, which is enjoyed by persons who have once suffered from these diseases. In the present century, Benjamin Bell is the first writer on syphilis who puts forth clinical facts to prove that "the venereal disease induces blindness, amaurosis, deafness, phthisis, rheumatism, epilepsy, mania;" and he demonstrated that *Gonorrhœa* was distinct from *Syphilis*. As far back as 1856, Mr. Lee showed, and Mr. Rollet since then has also shown, that not only is the infecting sore not capable of being transmitted from one part of the body to another, but it is not inoculable upon a person who has been already contaminated

by syphilis, more particularly so long as lesions continue to develop themselves. It is, therefore, as necessary now to distinguish "infecting" and "non-infecting" sores as it is necessary to distinguish the various forms of continued fevers. In future experiments and observations as to the effects of primary syphilis, it must be remembered that the subject cannot be studied or experimented with, to any extent, upon the patient himself. For this reason many of the early observations of Hunter and Ricord are limited and fallacious: one might as well attempt to study the vaccine disease by re-inoculation of it on the same person a second time, immediately after it has produced its specific effects. This brings us to—

III. *The Present Period in the History of Syphilis*, the commencement of which is of very recent date—since 1852; and is characterised by a belief in the *duality* of the venereal virus exclusive of gonorrhœa—when Bassereau restricted the term by separating from it the *soft chancres* with the suppurating bubos to which they often give rise. In addition to the virus of gonorrhœa (which may now be eliminated as distinct from those about to be noticed), we must recognise at least two forms of venereal disease, distinct in *their origin, propagation, and development*, and also in the sequence of phenomena which distinguish an "infecting" and a "non-infecting" sore. The sore which eventually *contaminates* the system commences differently from the sore which does not infect the system, and which is not the cause of syphilis. It will be noticed fully afterwards under "diagnosis," p. 938.

The disease of the present day manifests much the same forms and course as it did during the first half of the sixteenth century; and even now we occasionally see a repetition, upon a small scale, of the first known epidemic at the end of the fifteenth century, in cases where syphilis has suddenly invaded localities previously exempt. Where conditions are favourable to its spread and the locality retired, the first outbreak of syphilis is apt to show a remarkable intensity; and after the more virulent forms of the disease have become extinct, a hereditary taint is often left behind which manifests itself amongst the later generations in the form of manifold chronic affections: these have received the name generally of the locality where they made their first appearance:—*e.g.*, *Radesyge* (Sweden and Norway); *Jutland Syphiloid* and *Ditmarsch disease* (Holstein); *Sibbens* (Scotland); *Skerljevo* (the Illyrian coast). All these diseases (happily in the course of extinction) have been shown to be the results of acquired or inherited syphilis, which has often become inveterate and been neglected (CRAIGIE, BAÜMLER). The causation of syphilis in modern as well as in ancient times has often been rendered mysterious. In ancient times (1494) the air and atmospheric conditions have been considerably regarded as the media of contagia; and so also in modern times has accidental contact with any inanimate object—on the principle that what one wishes to be true he believes, and would like to persuade others to concur in his belief. The whole subject, from the earliest to the latest times, has always been, if possible, enshrouded in mystery; so that it is difficult to get at the truth from the very nature and history of the disease. After a period, indeed, of scepticism and doubt, we are now confirming, by actual observations (aided by all the advanced knowledge and appliances

of the day), the crude surmises of the physicians of four hundred years ago regarding the pathology of syphilis.

The different specific qualities of the poisons of syphilis and of the soft chancre are now established upon the same evidence as naturalists determine the identity of species in the animal and vegetable kingdom—namely, by the immutability of certain characters which the diseases that result from the respective poisons exhibit through successive generations. “Immutability of species” lies at the foundation of all classification in natural history and specific diseases. It is the groundwork on which the whole superstructure of the pathology of specific diseases rests. Into this consideration the mere *external* appearance of venereal sores or ulcers does not enter as an element. Even if we could not recognise one sore from another, by any outward sign (and sometimes we cannot), the existence of two different diseases would be established sufficiently by the fact, that the action of one poison was always local, and of the other poison as certainly always general—*i.e.*, constitutional.

Syphilis must therefore be looked upon as a special and peculiar disease—as a disease *per se*. There is no other disease like it in our nosology. It is one of such vital importance to the community (civil and military) in its prevalence, in its social relations, and to the State; and a disease of such intense pathological interest, that he who knows the history of syphilis, and can appreciate its phenomena, possesses in himself—it has been justly said—“the key to all pathology.” Among the many diseases we have to study few are so variable in their phenomena, taking them altogether, as syphilis; and our knowledge of this disease “has indeed been set in the light of many minds.” More has been learned of its nature within the last twenty years than was known during the four preceding centuries; and the Pathological Society of London has had a large share in this advancement—especially in the direction of “*visceral syphilis*.” The valuable discussions that have taken place—(1.) On the Pathology of Syphilis, opened by Mr. Jonathan Hutchinson on February 1, 1876; and, (2.) On Visceral Syphilis, opened by the President (the late Dr. Murchison) on January 16, 1877—have not only added to our facts regarding this disease, but have set those facts in a more intelligible relation to each other than heretofore, and so have helped to elucidate and to set the pathology of syphilis on a firmer base. The results have thus been the gradual outcome of many workers: and if we were to pass over in silence all excepting those who have contributed recent additions to our stock of knowledge regarding syphilis, and of them take only those who have worked at the departments most interesting in pathology, the list would still be very long, and the difficulty of doing justice to all is very great. In France, Ricord, Rollet, Diday, Viennois, Lagneau, Lancereaux, and Fournier; in Germany, Virchow, Wegner of Berlin, Dittrich of Prague, Von Bäreusprung, and Zeissl; Sigmund of Vienna; in Scandinavia, Bergh, Boeck, and Bidentkap; in America, Dr. Bumstead and Dr. Taylor are those who, by experiment and observation, have thrown most light on this disease: but (as Mr. Jonathan Hutchinson observed in his eloquent address at the Pathological Society, on the subject of syphilis) “their names do not even tithe those who have

a claim on our gratitude. At home, Dr. Wilks led the way as regards visceral syphilis; and the earliest contributions were most of them made before the Pathological Society. Drs. Murchison, Moxon, Weber, and Payne have also contributed most valuable facts. Our knowledge of the diseases of the nervous system, to which Dr. Reade, of Belfast, was the first of our countrymen to contribute, was early helped on by some cases from the practice of the late Dr. Todd, published in the *King's College Hospital Reports*; and more recently by the able researches of Dr. Hughlings-Jackson, and the valuable works of Dr. Buzzard, Dr. Russell of Birmingham, and Dr. Broadbent. Nor must I omit to mention, as zealous and successful workers at the general subject, the names of Lee, Berkeley Hill, De Méric, and Gascoyen; nor, last, the author of the most recent, and, I think, most comprehensive and able treatise on the disease that has yet appeared—Dr. Bäumlér—whom we would gladly claim as, at any rate in training, half an Englishman”* (*Med. Times and Gazette*, 5th Feb. 1876).

At the present day syphilis is prevalent throughout the world. Its principal seats are the great centres of traffic and luxury, whence it is continually being conveyed to other and remote parts; especially to great seaport towns, so that in many countries the coast is invaded before the inland regions. The countries where syphilis is especially prevalent are the South Sea Islands, where the disease threatens to exterminate the natives. In South America, especially Mexico, it is extraordinarily prevalent. In the East its principal seats are the great seaport towns of China and Japan; in Africa, Egypt, Abyssinia, Algiers, and also the western coast; in Europe, the northern provinces of Turkey, and the northern portion of European and Asiatic Russia (HIRSCH, BÄUMLER).

The etymology of the term “*syphilis*” is unknown. It was probably invented with reference, etymologically, to the most frequent mode of origin of the disease, and was first employed incorporated in a myth by the Veronese physician Hieronymus Fracastorius, who relates (in a poem concerning the disease) that a herdsman of King Alkithous, Syphilus by name, was afflicted with it by Apollo, in punishment for paying divine homage to the king instead of to the god. Other names employed in the fifteenth and sixteenth centuries were:—*Morbus Gallicus* (Italians, Germans, Spaniards), *Morbus Napolitans* (French, who afterwards called the disease “*la grosse Vérole*”). In Spain the popular name was “*bubas*” (pox); in Germany, *welsche*, *borsen*, or *purpeln*, also *mala franzos*. The older writers employ the term “*Leues Venerea*” (FERNCLEUX, 1556; Dr. BÄUMLER). As now used, the term “*syphilis*” comprises,—(1.) The primary specific infecting sore, and the changes it undergoes; (2.) The successive constitutional symptoms or phenomena which denote the contamination of the system, and which characterise the disease to which the name of syphilis is given in terms of the definition. The following classification and

* From 1863 to 1866 Dr. Bäumlér was Resident Medical Officer at the German Hospital, London. In the latter year he became a member of the Royal College of Physicians, London; and he practised in London from 1866 to 1872, at the same time acting as Assistant Physician to the German Hospital and the Victoria Park Hospital for Diseases of the Chest. In 1872 he was called to a Chair of Medicine in the University of Erlangen; and in the autumn of 1874 to Freiburg. His treatise on Syphilis is unsurpassed in its erudition and sound pathology, to which the account given in this Text-book is largely indebted.

definitions of the forms of syphilis have been given by the College of Physicians:—

(A.) *Primary Syphilis*. Definition—*Syphilis while limited to the part inoculated, and the lymphatic glands connected with it. The varieties are, hard chancre, with its indurated bubo; soft chancre, with its suppurating bubo; phagedenic and sloughing sore.* (B.) *Secondary Syphilis*. Definition—*Syphilis when it affects parts not directly inoculated.* Tertiary syphilis is a term sometimes applied to the later symptoms, when separated by an interval of health from the ordinary secondary syphilis. (C.) *Hereditary Syphilis*. Definition—*Constitutional syphilis of the child, derived during foetal life from one of the parents.*

Considerable objection may be taken to this nomenclature. *Syphilis*, comprehending, as now generally understood, primary and successive constitutional phenomena of contamination, ought now to be distinguished from simple *venereal* ulceration not followed by specific gland complication nor contamination of the system. The time has certainly come when primary syphilitic sores ought to be distinguished from soft chancres, or simple contagious ulceration not followed by contamination of the system; and the term *syphilis* should therefore now be reserved to designate the more serious affection, in which the constitution is implicated, and in which the infecting phenomena occur. Syphilis may be due to venereal disease, but not necessarily—so that etiologically “syphilis” and “venereal disease” are not synonymous terms. If the term “chancre” is used, it should be stated whether it is a “soft,” “non-infecting chancre,” or a *hard* and “infecting” one; otherwise diagnosis is incomplete. Men or women may have primary sores on the genitals, with suppurating bubo, and yet remain free from any taint. Such cases cannot be regarded as “*syphilitic*,” or even as general diseases. They are simply cases of “local venereal ulceration,” with gland complication, consequent on irritation from the sore, and not going on to contaminate the system. Such are the so-called soft chancres. (See under diagnosis, page 938.)

The disease ought to be considered as ONE, from the earliest to the latest manifestations; even into the so-called tertiary condition as modified by time and by daily change of structure. Thus it gradually approaches a termination where the patient may be considered free, but after which the disease may return afresh (recrudescence) by exposure to certain influences, such as distress, ill-health, bodily ailments, and the like. (President’s Address, Mr. GEORGE POLLOCK, *Med. Times and Gazette*, April 8, 1876.)

“The terms *primary, secondary, tertiary, quaternary*, are coming to have no meaning as applied to syphilis. It is a disease *per se*—one disease from beginning to end—including all the *sequelæ* following an infecting sore. Either a man has syphilis or he has not; he either has poison in him, producing all these peculiar morbid products, or he has not; and I believe that all these visceral changes that we observe are due to the true syphilitic process, and take place, I have no doubt, at the same time. When we examine the body of a man who has had syphilis, who is the subject of inveterate syphilis, we find, no doubt, these hard fibroid nodules in his body; but, at that time, or previously, he is comparatively well. One knows, as a fact, that these nodules have been felt, it may be,

in the liver, for years and years. I have always regarded them as a sort of *débris* of the true syphilitic deposit. That they may undergo further change, or grow, as Sir James Paget has said, I have no doubt, because we see that similar inert masses do increase in size in the body. Then, again, we know that there must be something previous to that. We have evidence of it in those very cases where fibroid deposits have been found; we know that the man, during the exanthems, has really had enlargement of the liver, with the affection of other organs; therefore we are bound to believe that there was a previous stage, and we want to know exactly what the condition of his organs is at that period. The early products in the viscera are known to be absorbable to a certain extent" (Dr. WILKS, l. c.)

(b.) *Causation, modes of propagation, and general phenomena of development.*

—A comparison of the pathology of the three poisons whose lesions are expressed by the names of, (1.) Gonorrhœa, (2.) Soft chancre, (3.) Syphilis, leads to the following conclusions:—(1.) That the only property common to them all is their communication, for the most part, by contact of the genital organs; hence *venereal lesions*—a term now far too indefinite, because it comprehends several distinct diseases, which are clinically distinct, having no pathological connection with each other, except in the mode of acquiring them. (2.) That the poisons of gonorrhœa and of the soft chancre are alike in this—(a.) That their action is limited, and never extends to the general system; (b.) That one attack does not afford the slightest protection against a second. They differ, however, in this, that gonorrhœa may arise from different sources of infection—purulent discharges of different kinds in the female; while the *soft chancre* does not arise except by inoculation from an ulcer of its own kind. In gonorrhœa ulceration is rare. The catarrhal inflammation which is characteristic of the disease affects the surface only of the mucous membrane; and its complications mainly attack parts continuously connected by mucous surface with the original site of disease, as the *prostate, bladder, testicle*, and sometimes the *kidney*. The soft chancre, on the other hand, is an ulcer involving the whole thickness of the integument or mucous membrane, and its complications are limited to the adjoining absorbent vessels and glands. Of the three venereal poisons the *syphilitic* is alone capable of infecting the system at large, and also of affording protection by its presence against subsequent attacks. The *infecting sore* (the one which contaminates) commences as a *dry papule, pimple, abrasion, fissure, or crack*, around which, after a *period of incubation* of three or four weeks (*average twenty-four days*) a *specific growth of tissue takes place* (the *PRIMARY lesion, a specific sclerosis or induration*), forming a lump or protuberance more or less voluminous. A *pustule is no essential part of the process, nor is suppuration*. They are accidental phenomena, the result generally of irritation, pressure, or laceration, which produces a sore or ulceration—a result always very easily established and maintained in connection with infecting sores, as compared with other sores. There is then established an *erosion*, as a second form of primary lesion often seen; and lastly, the indurated chancre as the third form of primary syphilitic lesion. Unlike the poisons of gonorrhœa and of soft chancre, the poison of syphilis is not only conveyed by pus or by the particles of bioplasm, but it exists in and poisons the blood

for a certain time, and is present in the fluid serum of certain lesions, in the serum of the blood itself, and probably in other secretions. The secretion of a superficial hard syphilitic chancre, as shown by microscopic examination, is often entirely destitute of pus, but not the less certainly full of minute *living* particles (or bioplasm) capable of propagating syphilis. Unless irritated, the secretion is not purulent. The presence of the poison of syphilis in certain lesions is proved by the power which the serum merely, or discharge from these lesions, possesses of communicating the disease; and in semen by the occurrence of hereditary syphilis in the offspring when the father alone is infected.

On the other hand, it is of the greatest importance pathologically to bear in mind that the co-existence of the three venereal poisons is not incompatible. They may all co-exist in the same person, who may at the same time have *gonorrhœa*, *soft chancre*, and *syphilis*. Hence there are also "*mixed*" chancres, or venereal sores of a mixed kind. The *vaccine virus* may also carry *syphilis* (besides the blood, serum, and seminal fluid). Thus the disease develops itself after the introduction of a specific living virus, and the source of the poison is more distinctly traceable than that of the diseases known to be inoculable (and which have been considered in the previous section), such as *small-pox* or *measles*. The actual substance or matter which contains the virus can be obtained, and can be inoculated. Yet the *active principle* of the poison has not been isolated by any chemical process; and in this respect it is in exactly the same position as the poison of any other inoculable disease. If it is a material, living, definite particle, germ, or protoplast, it may not be within the scope of chemistry to find any active chemical principle in it. But there can be no doubt that some kind of virulent contagious principle does lie at the root of the disease. This was made evident even in the first great outbreak at the end of the fifteenth century.

Next comes the important question put by Bäumler:—"In what constituents of an organism infected with syphilis does this poison reside?" and in general—"What are the relations which it bears to the organism in which it is, or towards one not infected?" Three cases are on record where syphilis has been communicated by inoculation to two guinea-pigs and a kitten (*Brit. Med. Journ.*, 1871, Sept. 30, p. 376). In the kitten and one of the guinea-pigs the inoculation was followed in two or three weeks by a local thickening of the site of inoculation, and afterwards by constitutional syphilis. The guinea-pig died within one month from the commencement of induration, with destruction of one eye, extensive ulceration of the mouth and of the soft palate. At the end of the eighth week the kitten was killed, when syphilitic gummata were found in the kidneys and liver (S. MESSENGER, BRADLEY.) In the third case—a guinea-pig—Ch. Legros produced an indurated ulcer by inoculation. During cicatrisation of the wound the animal commenced to grow marasmic, and died five and a half months after inoculation, when there were found extensive swellings of the lymphatic glands, enlargement of the liver, with the cicatrices in its surface and little cellular tumors in its substance, besides a number of gummy nodules in the deeper layers of the cutis, and a fine yellow nodule, the size of a pea, in one epididymis (LANCEREAUX, quoted by BÄUMLER). In 1844 a young French physician—Auzias Turenne—commenced a series of experi-

ments with the view of testing John Hunter's doctrines regarding the non-communicability of syphilis to the lower animals. He succeeded at length in producing, on monkeys inoculated with chancre matter, a disease which had all the characters of a chancre. A disease was communicated to them capable of being transferred to rabbits, cats, and horses. It appears, also, that syphilis was established in these animals; and the chancres produced by inoculation became less and less in each animal, until at length a period arrived at which the virus seemed to lose all its power. No sores of any kind occurred. When a sore was established, however, in these animals, the virus was preserved, and was capable of transmission, and of re-transmission back to man. It was inoculated from a cat upon a German physician and Professor in the University of Wurtzburg. On him it gave rise to a hard chancre, then to constitutional syphilis, demonstrating that the virus lost none of its virulence by the transference from man to animals, and from animals back to man. Nevertheless, Turenne believed that by prolonged inoculation the system became protected; and hence arose the practice of "*syphilisation*."

But besides the discharge (non-purulent or mixed with pus) from an ulcerating sclerosis, the infecting sore, there are other sources of infection; namely,—(1.) The contagion of constitutional syphilitic sores—*e.g.*, the syphilitic secondary ulceration of the female nipple inoculating the mouth of the healthy infant born of healthy parents; also the frequent infection of nurses through suckling infants, transmission of the disease in kissing, &c. (2.) *Secretion from syphilitic tonsils*, also from *acne pustules* in a child with hereditary syphilis; these have produced by inoculation syphilis in healthy persons. (3.) It is now also established that *secondary* syphilitic inoculation (*e.g.*, the thin fluid discharge from the softening and ulceration of *mucous tubercles*, *flat condylomata* *papules*, and the like) gives rise to a sore which exactly resembles a primary infecting chancre, followed by all the constitutional phenomena. (4.) *The blood* of those suffering from acute syphilis, in the form of general eruptions, inoculates. It is probable that the blood is virulent only during the febrile period, or up to and including the period of cutaneous eruption—as it is not found to be so during what have been called the tertiary phenomena. Experiments at Florence, at the Clinique for Venereal Diseases, show that healthy persons (*i.e.*, those who have not previously had syphilis) may be inoculated with the blood of syphilitic patients. On January 23, 1860, two young doctors were inoculated with the blood of a syphilitic patient, but no result followed; on February 6, 1862, three other doctors (perfectly free from syphilis) were inoculated by venous blood taken from a female suffering from the acute lesions of syphilis. Charpie soaked in the blood was applied to an abrasion in the arm of each. On March 3 (twenty-five days after the operation) a slight itching and elevation was perceptible: a papule formed, which, eight days afterwards, became covered with a crust. This crust increased in thickness day by day: and twelve days after the appearance of the papule two glands in the axilla became enlarged, and the sensibility of the papule increased. Nineteen days after the appearance of the crust it fell off, leaving a funnel-shaped chancre, with elastic resistant borders. On the twenty-third day the chancre had increased in size and induration. On the fortieth day eruption on the skin and glandular

swellings in the neck supervened. The erythema lasted eight days, and pursued a regular course. On the forty-eighth day the glands had increased in size and hardness, the chancre maintaining its specific condition, showing no tendency to heal. On the fiftieth day the colour of the erythema became decidedly coppery, and treatment by mercury was then begun. In these experiments the blood communicated disease to *one* out of *five* who submitted to the experiment. Thus the contagious properties or inoculability of the blood during the acute state is conclusive. Numerous instances are also related by Bäumler. The recognition of this fact explains many occasional cases of syphilitic affection hitherto obscure—*e.g.*, syphilis from vaccination, contamination of a healthy nurse from the sore mouth of an infected infant, and the like. One of the most remarkable and lamentable instances of the inoculation of syphilis through vaccination is that which is now well known as the epidemic at Rivalta. At that place no fewer than *forty-six* children became affected with syphilis, the disease being communicated to each of them through the operation for vaccination (PACHIOILI, SPERINO, *New Syden. Society Year-Book*, 1861-62). These and such like accidents have fully proved both for the acquired and for the inherited forms of syphilis, that the blood may be rich in contagious material even when the patient displays no external symptoms.

(5.) *Propagation through the semen—Hereditary Syphilis and Transmission of Syphilis by Inheritance.*—Several years ago Professor Harvey, of Aberdeen, directed attention to the power of the foetus to inoculate the maternal with the anatomical and physiological peculiarities of the paternal organism; and he then suggested the probability of secondary syphilis being imparted in this way (*Ed. Mon. Journal of Med. Sc.*, Oct., 1849, and Oct. and Nov., 1850). A female, otherwise free of syphilis, may thus become contaminated during the gestation of a foetus begotten of a male who, at the time of the fruitful connection, was himself alone suffering from contamination of the system by syphilis, in some form of active secondary phenomena (HAY, HARVEY, DIDAY, MACLEAN). In such cases no syphilitic sores existed on the genital organs of either party at the period of sexual intercourse; but the begetting of a syphilitic child within the body of the mother contaminates the mother “through the foetus—itself begotten of the father, from him inheriting the *virus*, and having *in utero* the disease resulting from it in such manner and degree as to impart it to its mother through the medium of the placenta” (ROBERT HARVEY). It is also highly probable that the blood continues to be contagious for a certain period after the external phenomena have ceased. How long that period is, and within what limits its duration may vary, are still undetermined questions. Mr. Hutchinson is of opinion that—

“We can produce but little evidence in favour of prolonged contagiousness of the blood. All the accidents occur during the year or eighteen months which we count as the secondary stage, and most of them in the early part of it. There is every reason to believe that, in the tertiary stage, neither the blood, nor even inflammatory secretions produced by sores which still bear the specific type, can reproduce the disease. Our next test is the possibility of transmission to offspring; and I note that almost every speaker has been inclined to assert that the production of a tainted

child must be regarded as proof of blood-poisoning still extant in the parent. This may be so; but I cannot help the conjecture that it may be possible for the germs to still hold possession of cell-structures in the ovary or testis when they no longer exist free in the blood. That such is the case cannot be proved, and must rest for the present as mere conjecture. It becomes, then, of great importance to answer the question—How long after the secondary stage is it possible for syphilis to be transmitted hereditarily? During the last few weeks I have gone through the notes of a great many cases, in order to get data for a safe reply on this head, and with the result of a strong impression that we have much exaggerated our estimates of the time. The cases in which syphilis is transmissible by inheritance for more than a year or two after its secondary stage appear to be very exceptional. I possess notes of a few in which successive children, during a period of seven or even ten years, have presented evidence of taint; but the ordinary course certainly seems to be that the first two or three children suffer, and that the others escape. Unless the risk of hereditary transmission did really cease early in the vast majority of instances, infantile syphilis would be far more common than it is. It will be readily seen that our decision on this point is not a mere matter of speculation or of transcendental pathology, for upon it must rest the advice which we give our patients in reference to marriage. I have for long made it a rule when consulted on this point to insist that, before marriage, a period of two years should elapse from the last of what I have considered blood-symptoms. I have given this opinion to a great many persons, and may confess that it has been a constant source of anxiety, lest some day some one should bring me a snuffling spot-covered baby, and say: 'See here; you said I might marry; just look at this!' Such an occurrence has, however, never yet happened to me. I may strengthen the bearing of this fact by adding that I have been cognisant of not a few cases in which the marriage took place at a much shorter interval than had been advised, and yet healthy children were produced. I repeat, then, that there is reason to believe that the instances of liability to transmit to offspring extending over periods of several years are exceptional, and are by no means to be dealt with as if they illustrated the rule. There are, moreover, numerous fallacies to be carefully kept in mind in investigating cases of the supposed unusual prolongation of this risk. There is the almost certainty, to which I shall have to allude directly, that the mother becomes contaminated by her foetus, and thus, if healthy before, supplies a new starting point for the infection of future children; and there is always the risk that one or other of the parents may have contracted the disease a second time. Thus, then, we have clearly a period of syphilis during which the original sore is contagious; a period during which the blood and tissues are contagious; and a period during which transmission to offspring is possible. It is doubtful whether or not the two latter cease simultaneously, but there is some probability that the last remains the longest; both, however, in almost all instances, end within comparatively short periods" ("Discussion on Pathology of Syphilis," l. c.).

The virulence of the *semen* of a syphilitic father for the child he begets is now an unquestionable fact (BÄUMLER). This may occur without the mother becoming syphilitic. On the other hand, the ovule developed in the ovary of a syphilitic woman gives rise to a foetus which will be syphilitic, although the ovule may have been impregnated by the semen of

a healthy male. A third mode of hereditary contamination is that where the *mother acquires syphilis during the time of gestation*, and infects the developing foetus through her blood. The conclusions recently arrived at are—(a.) That syphilis in the child may and most commonly does come from the father alone (VON ROSEN, JONATHAN HUTCHINSON, E. FRANKEL). (b.) The preponderance of the paternal influence is such that if the husband is healthy, but the wife syphilitic, the probability of the offspring becoming gradually free from syphilis is greater than when, on the other hand, the wife is healthy and the husband syphilitic (HUTCHINSON). (c.) A man who at the time is apparently free from disease, or has been so for a number of years, but at some former time was syphilitic, is still capable of transmitting syphilis to the child he begets. (d.) Syphilis in both parents is more certain to cause disease in the child, than when but one of them is infected; and the disease in the child will probably be severe. (e.) The shorter the space of time since infection, or since the outbreak of constitutional symptoms in the parent, the severer is the infection of the offspring. (f.) Whether syphilis is transmissible into the *third generation* is still an open question. (g.) Persons who have suffered from inherited syphilis in their childhood are in later years protected to a certain extent; and if they become infected, the disease runs a modified course.

(6.) *Muco-purulent, urethral or vaginal discharges* are also vehicles of syphilitic virus; such secondary syphilitic *urethral discharges* sometimes occurring after marriage (H. LEE, *Brit. Med. Journal*, July 24, 1875, page 93).

The “infecting” sore of *syphilis* does not remain merely a local disease. It contaminates the system, giving rise, by a multiplication like that of *small-pox* poison, to one of the most malignant, most lasting, and most destructive forms of a disease poison that affects the human frame. How is this brought about? We do not quite know; but the poison seems to multiply and grow with the physiological processes of life, just as *rheumatism* or *gout* increases and develops; and hence syphilis has been classed among the constitutional diseases in this section. “In all our pathological researches, we are endeavouring to find out the why and wherefore of disease. Here is a wonderful example for us. A healthy man has a small amount of virus introduced into him, perhaps an infinitesimal amount, and the whole of his nature is changed; he breaks out into a rash over the whole surface of his body, and all his viscera are affected. For years and years afterwards any morbid process that may take place is entirely altered in its character. The man brings into the world a number of puny, ill-formed children, and the result is, perhaps, seen in the next generation. There is no example like it in pathology. It is a large experiment made for us, and that is why so many of us take an interest in this affection” (Dr. WILKS, *Brit. Med. Journ.*, Feb. 19, 1876). The only constant index of such contamination seems to be the occurrence of multiple enlargement of related lymphatics and lymphatic glands in whose district the infected part lies. This enlargement begins about ten or twelve days after the indurated papule has made its appearance—*i.e.*, from four to five or six weeks after *contagion*,

implantation, or *inoculation* by sexual intercourse, or otherwise. Such glands do not suppurate. They are congested, and become hypertrophied. The induration is due to a very uniform germination of young cells in all parts of the gland. Every space is crammed with cells; and this condition may remain unaltered for *months* and *years* together. Ultimately fatty degeneration sets in, the *débris* is reabsorbed; and the glands return to their normal state (RINDFLEISCH.) They enlarge slowly, and without pain, in the immediate vicinity of the sore; and can be felt as a group of enlarged glands freely movable beneath the skin. Eventually those in the axilla become similarly affected, and ultimately enlargement of the chain of glands, extending up towards the occiput, behind the sternomastoid muscle, is apparent. A general morbid condition of the whole system is the necessary result of this extensive disease of the lymphatic system. Nutrition becomes defective. The blood is changed; it becomes anæmic. Emaciation is then often rapid. The digestive organs are impaired in function. The muscles lose their hardness, elasticity, and energy; and the later lesions peculiar to syphilis set in.

The *virulence* of the syphilitic poison varies much as to its results. It is most intense in the primary lesions; it is of almost equal intensity in various local affections of constitutional syphilis, such as in the flat condylomata and in the pustular affections of the constitutional disease. So long as these local manifestations of the infection of syphilis are present in a patient, that patient may communicate the disease in any of the ways already mentioned. But whether the contents of the gummy tumors, or the secretions from the ulcerating tubercles of the skin; or whether, in general, the local affections of the (so-called) tertiary period are infectious, are still unsettled questions.

The *susceptibility* to the syphilitic poison also varies much. The mode of life, manners and customs, material prosperity, national temperament, and the grades of civilisation, are the main conditions that influence the spread of syphilis. In some there is an acquired immunity, as in those who have already had the disease. By this fact is explained the circumstance that a child with hereditary syphilis does not infect its mother, though a healthy nurse may contract the disease from the child (COLLES *On the Venereal Disease*, 1837, p. 385); also, the failure of auto-inoculation—i.e., inoculation of the syphilitic poison upon the person from whom the poison is taken. It is not auto-inoculable. The general contamination is of such a kind as to render the system, as a rule, proof against a second invasion of the specific "*infecting*" virus. The disease never repeats itself, except, it may be, after a long interval. Sigmund has seen such a case of repetition, and similar cases (*reinfectio syphilitica*) have since been recorded by Zeissl, Diday, Hutchinson, Lee, and Bäumlér. They are rare, and are often mere relapses, or rather a recrudescence of the original disease. In this respect syphilis resembles other virulent diseases acknowledging a specific virus as their origin; and in them the immunity is usually, but not invariably, complete—e.g., in small-pox, cow-pox, scarlet fever, and the like. After the system is once infected, the specific sore cannot be transplanted by contagion or inoculation to any other part of the body. The "*infecting*" sore is not *auto-inoculable*; and a person suffering from a chancre affecting his system will not be affected by a further inoculation of the same

specific virus. There is, however, a slight qualification to be made here. Mr. Henry Lee has shown that, (1.) There is a *stage* in the existence of an "*infecting*" sore when it is *auto-inoculable*. That period or stage is a very early one in the existence of the sore—namely, before any specific systemic action has begun to develop. If at this period the poison of another "*infecting*" chancre from another person, or from the chancre already existing on the same person, be inoculated, then a second "*infecting*" chancre, accurately representing the original, will result. The period when this event can happen is *before* the gland induration. (2.) Mr. Lee has also shown that there is a *certain condition* of the chancre in which, at any stage, on being inoculated or transplanted, it will produce a sore. It then appears to be auto-inoculable: but this is only in appearance, and not in reality. The condition of the chancre that does this is one of *irritation*. Blister a chancre, or irritate it by an irritating ointment, or by any other means, so as to cause pus to flow—free pus-corpuscles being generated—and then we may have what has been recently termed a "*mixed chancre*," of much more frequent occurrence than has generally been supposed (SIGMUND). Sigmund has produced such chancres by inoculation. The utmost caution, therefore, is necessary before pronouncing a sore to be *non-syphilitic*—i.e., "*non-infecting*." Sigmund inoculated the pus of a soft, contagious, or suppurating sore upon the infiltration or sclerosis of a hard papule on which the skin had remained unbroken. Between twenty-four and forty-eight hours after, a suppurating ulcer was established, which afterwards assumed Hunterian characters. Inoculation of two poisons may thus be in some cases simultaneous or successive. Hence "*mixed chancres*" present two aspects: on the surface is the soft, contagious, pus-producing ulcer; while deeper down is the specific syphilitic infiltration of the true "*infecting*" virus. Local plugging and enlargement of the superficial absorbents take place from such "*mixed chancres*," followed by similar infiltration of the group of lymphatic glands nearest to the sore, spreading gradually to distant and more distant groups. This is the constant series of phenomena after syphilitic "*infection*,"—a regular series of connected events, giving rise to such symptoms as are associated with no other disease-poison except that due to syphilis.

The interval of reinfection is a long one—five to twenty years. In general the second attack is a modified one, and all the milder the sooner it occurs after the first. Persons also who have suffered from inherited syphilis in their childhood, are in later years protected to a certain extent; and if they become affected the disease has a modified course. It is undetermined, however, whether the children of syphilitic parents inherit a certain degree of immunity without having actually had hereditary syphilis. The thorough saturation of the inhabitants of Portugal, and the mild course which the disease follows amongst them, points to some such immunity, either acquired or inherited. A similar effect is observed amongst the common classes, who are peculiarly exposed to syphilitic contagion (Dr. H. LEE).

(c.) *Morbid Anatomy*.—Advances in pathology of late years have not been more marked in any direction than in demonstrating the very remote effects which the poison of syphilis exercises upon the organs and the constitution of man. These advances are due to clinical, experimental,

and *post-mortem* observations. They have shown that a considerable number of doubtful cases of ill-health are in reality due to the specific poison of *syphilis*, whose morbid effects are not fully developed till many days, months, and even years after inoculation. Hitherto surgeons have claimed the subject of *syphilis* as their peculiar field; but after the surgeon had healed the sore, the morbid influence of the poison in many cases still remained, and internal lesions, impaired health, and degenerate constitution, eventually brought the patient to consult the physician as well as the surgeon. The pure surgeon and the pure physician must, therefore, condescend to forget their purity as regards the conventional separation of professional practice into the two departments of the Surgeon and the Physician, if they would comprehend the pathology and morbid anatomy of this disease. The relations of syphilis are so vast and complicated, that both the physician and the surgeon must combine their knowledge and their skill, before the many interesting points in the pathology of syphilis can be fully cleared up. To heal the original sore and obtain a cicatrix is but the beginning of the end. It is partly to the unscientific division of the field of medical practice into Medicine and Surgery, that the phases of opinion regarding the pathology of syphilis have been so remarkably diversified. *The surgeon* alone saw the primary infecting sore or inoculation, and only by chance he might see the development of the future lesions, now so important in pathology. *The physician*, on the other hand, rarely saw the primary sore; and when at last he saw the victim of secondary and tertiary syphilis, the case had often become extremely complicated, "mixed up and overlaid by other constitutional and local diseases," which in their turn were made more serious by the existence of syphilis. It is especially the remote and visceral lesions of syphilis, as demonstrated after death, which have brought this disease within the province of the physician, and which warrants its finding a place in text-books of medicine.

I. *General Anatomy of the Lesions.*—Cellular infiltrations are properly characteristic of the lesions of syphilis. They constitute the distinctive feature of all the local lesions; and the connective tissue is the matrix in which they grow. The cells composing them have usually a single nucleus, and bear a close resemblance to the white blood cells, to the cells of lymphoma—adenoid cells—and to the cell growths in enteric fever. The entire formation resembles granulation tissue (VIRCHOW); but they have no specific microscopic character as peculiar to syphilis. Nevertheless the arrangement, the development and further course of the cell growths, do afford certain marks by means of which it is possible to distinguish the syphilitic new growth from every other (Dr. BAÜMLER, E. WAGNER). The lesions have therefore received special names, such as "*syphiloma*" and "*gummy tumor*." These gummy tumors or gummata are the basis of all the characteristic lesions of syphilis. They form growths which lead to the development of elastic tumors, composed of a well-defined tissue, with elements extremely minute. The growth takes origin from the elements of connective tissue, or the analogues of such tissue, and hence the universality of their site. They are like pus or an abscess in this respect; but their favourite seats are—the subcutaneous cellular tissue, the skin, in and upon the bones, the liver, the testicles, the brain, the kidneys, and,

especially in children, the lungs. When they first attract attention they are small, solid, pale swellings, like a hard kernel, varying in size from that of a pea to the size of a haricot bean. They present the appearance of a greyish-red, soft, homogeneous mass, either without fluid contents or else yielding a scanty juice like mucus. They may be generally first seen in true skin, or subcutaneous or submucous tissue; and where the tissue is lax they grow to a considerable size, and give a sensation to the hand as if filled with gum (*tubercula syphilitica*, *nodular syphiloma*). Repeated examinations of this growth show that in the gelatinous condition it arises from a germination of nuclei in the cells of the connective tissue—like the formation of granulations in a wound (*granulation tumors* of Virchow). The component elements appear as round or oval cells, more or less closely packed together, imbedded in a matrix of fine connective tissue, of a granular character, tending to fibrillation, especially in the presence of spindle-shaped cells. The cells are a little larger than red blood-globules, and contain granules, with a nucleus in their interior when mature. In some respects they resemble tubercle, but differ thus in the mode of growth. The specific anatomical peculiarity consists in the circumscription of a more or less spheroidal nodule in the midst of a larger deposit of newly formed embryonic tissue, more at first in the form of a diffuse infiltration (Fig. 96) than a distinct tumor. Such infiltrations may be scattered through the parenchyma of an organ; and



Fig. 96.*

when they appear as tumors as large as a walnut or more, they are not encysted nor sharply defined, but merge directly into the surrounding tissue. This nodule is still further peculiar in the subsequent changes through which it passes—(1.) as regards the embryonic tissue which surrounds it, and (2.) as regards the nodule itself. The embryonic tissue becomes converted into fibroid tissue, forming a condition resembling a cicatrix, characterised by a tendency to extreme contraction. The component parts of the nodule consist of minute cells, which continue to retain their form, and sometimes constitute a reticular or anastomotic network of corpuscles, until its intercellular substance undergoes a morbid transformation—a softening takes place in the central portions, which become gummous or honey-like; or the mass dries up and becomes firm. This production of mucoid tissue is the commencement by liquefaction of a slow degenerative change. The individual cells become fatty—their place is taken by round or stellate aggregations of fat-granules,

* Syphilitic induration. *a.* Vascular lumina; *b. b.* All clear spaces are bundles of connective tissue pressed asunder by a uniform small-celled infiltrate, 1'300 (after RINDFLEISCH).

which continue for long periods of time without further change. In the end a yellowish-white roundish nodule of soft and elastic consistence is the result, imbedded in a deposit of newly formed connective tissue, which tends to continuous accumulations under the least irritation. On section it is the mingling of greyish-red translucent tissue with scattered irregular caseation, which are so characteristic of syphilitic gummata, especially in the visceral organs. How are we to recognise the specific nature of such gummatous nodules? There is nothing in them so specifically and anatomically distinct that, apart from their history, they can be recognised. The history of the syphilitic case during life is the great guide. The nodes on the shin-bone or clavicles have long been recognised as the product of syphilis. It may almost be said that they have been seen to grow under the eyes of the patient and the observer; and their anatomical characters are found to be such as compose the gummatous nodules just described. In a case of inveterate syphilis, therefore, whose history is fully known, in whom the node on the shin or other bones is characteristic, and has been seen to grow, and in whom also we find similar nodules in the lungs, or in the liver, or in the testicles—symmetrically growing in these latter organs, and consisting of minute cell-elements exactly the same as the node on the shin—it is impossible to overlook the fact, or not to be impressed with the belief, that all of these lesions acknowledge one and the same cause of development—namely, the syphilitic poison, of which they are the expression. The progress of the node is also characteristic and suggestive. Growths of a similar form which result from idiopathic inflammation generally proceed to the formation of an abscess, or to the hypertrophy of fibrous tissue. Abscesses are recognised by their pus; fibrous tumors or hypertrophies, by the fibre-elements which compose them. Growths of a form similar to the nodes which result from cancer, are in general to be recognised by the juice expressed from them. In the gummatous nodule we have no such juice, and the cell-elements seen in cancer are generally so diversified in their form and mode of growth as not to be easily mistaken. The gummatous nodule is uniform as to the size and form of its cell-elements, and is a growth less highly supplied with blood-vessels than a cancer. Cancers tend to infiltrate and involve neighbouring textures; the gummatous nodule remains isolated and distinct, and is eventually surrounded by a dense but clear, semi-transparent greyish vascular fibrous tissue, very resistant to the finger. Thus these nodes appear sometimes as if inclosed in a cyst, from which they may be enucleated. By way of elimination or exclusion, therefore, and by duly observing the history of the case (and even without such history), we are generally able to recognise the nature of such growths, and to assign to them their proper place in pathology. The gummatous nodule has now been recognised and described in almost all the solid viscera of the body. During the growth of the nodule, germination advances slowly, and a gluey-like material forms, which constitutes the inner cell-material of the nodule. If near the surface, such a nodule is apt to melt down, soften, open, and ulcerate; and such a result seems to be associated with other evidence of active constitutional disease. The tumor, however, continues gelatinous and coherent if it is inclosed in a dense part, or is deeply seated, as in

gummata of the periosteum, scalp, brain, liver, testicle, lungs, and heart, if constitutional disease remains latent or inactive. Fatty degeneration may also eventually occur in the gummatus nodule, and eventually lead to its absorption; or its absorption takes place as a natural process of cure, the changes of which are not exactly known. We know only that the node on the shin-bone not seldom disappears from view, and does not return. Thus while nothing specific exists in the elements of the gummy tumors of syphilis, the general course of development which the new-formed tissue pursues is more especially characteristic. Its course, its causes, and even the action of remedies on it must be taken into account in determining its nature.

II. *Primary Lesions.*—The first specific lesion to be inquired into as regards its anatomy is "induration of the sore itself." The hardness of the induration is peculiar. It arises without inflammatory action, and must be distinguished from the hardness caused by irritation or friction, as of clothes rubbing on the sore. A sore after a few weeks may heal up, but if it leaves a "lump," that "lump" is specific induration, and denotes contamination of the system; and so long as hardening continues at the site of the original sore, the contamination may at any time express itself by some specific local lesion distant from the original seat of the sore. The elementary constituents of the induration are constant. It always presents the same anatomical composition. It resembles the development of fibro-plastic tissue in the substance of the true skin. It is an exuberance of growth (a germination) of the elements of tissue, similar to the gummy tumors or node just described. (Fig 96.) There is an infiltration of the connective tissue with the small cells, uniformly packed into all the interstices between the vessels, on the one hand, and the fibres of the connective tissue, on the other—an infiltration which produces an elastic tension, conveying the sensation of hardness to the fingers. The capillaries show numerous cells in their markedly thickened walls, infiltrating far beyond the site of the sore. The calibre of the thickened blood-vessels is diminished, hence the remarkable dryness and rigidity of the tissues in the initial sclerosis of syphilis, as well as the waxy whiteness upon slight pressure (VON BIESIADECKI). But a similar cell infiltration obtains in *lupus* without such hardening; hence something besides cell infiltration is required to account for the hardness in the primary infecting sore. Auspitz therefore describes, in addition, an amorphous substance interspersed between the cells and the normal tissue elements. But, in addition, there is a new formation also of connective tissue fibres, especially in the peripheral portions in the higher grades of induration (VAREN, BIESIADECKI, BAÜMLER). The lymphatic vessels within the morbid growth have been found enlarged. The papillæ of the true skin are thus augmented in volume; and they become infiltrated by a large quantity of new growth, in which they seem imbedded—the embryonic elements of the growing connective tissue. Irritation of the neighbouring sudoriparous glands also adds to the induration, and augments the volume of the parts. This new material does not live long. It soon commences to degenerate—to undergo a change into granules of fat; and to this molecular death is due the formation of the dry ulcer, so characteristic of the true *syphilis* sore. This degeneration of the new elements of

growth is characteristic of all the subsequent lesions of syphilis, whether they be called secondary or tertiary—whether they occur in the true skin, the bones, the liver, the heart, the arteries, or any other part.

(1.) *Gland Lesions.*—Following the induration of the primary sore (the first evidence of constitutional contamination), is the chronic induration of the corresponding lymphatic glands—forming what are called “indolent bubos.” The induration is due to the same anatomical peculiarity as in the gummy tumor and induration of the primary sore itself—namely, a very uniform, but by no means luxuriant, germination of young cells in all parts of the lymphatic glands (RINDFLEISCH)—a *hyperplasia of the cellular gland elements*. Every vacant space is crammed with cells—a condition which may remain unaltered for months and years together. The gland does not tend to proceed to inflammation, nor yet to caseation or necrosis. Ultimately the tendency is towards fatty degeneration of the cells—reabsorption takes place, and the gland returns to its normal state. But a second general infection of the glands also takes place, especially from the local affections of the skin, the mucous membranes, and the bones. The poison is then conveyed in larger amount to the related lymphatic glands, exciting in them changes similar to those which become hard in connection with the primary infecting sore—the indolent bubos of the early period. Thus, in connection with the secondary affections of the mouth, the submaxillary glands enlarge; with eruptions of the hairy scalp, the cervical glands; with papules of the palm, or affections of nails, the cubital glands. On the other hand, the morbid blood is held sufficient to establish directly a general hyperplasia of the glands. Hence the glands at the nape of the neck, those above the internal condyle of the humerus, or the small glands at the side of the breast, often become enlarged before any source of peripheral irritation can be discovered. The lymphatic glands in the interior of the body have also been found enlarged at *post-mortem* examinations by Virchow and Von Barrensprung. They either terminate in resolution, suppuration, caseous degeneration; or follow the course of a *gumma* containing a gummy-like mucous fluid, or become lardaceous.

(2.) *The Blood* is the seat of considerable modifications. These occur mainly in the earlier stages of syphilis, and consist in a diminution of the red blood corpuscles—to the extent of *one-seventh* or *one-half* of the usual number. An increase in the albumen of the serum also takes place, and an increase of white globules. The change is apparent to the naked eye, as, namely, *oligæmia*. But unless the chronic ill-health of syphilis—*sypilitic cachexia*—supervene, the blood begins to improve again. During the secondary stage of syphilis all the tissues are uniformly subjected to the action of the poison, the vehicle of which at this time is the blood, as shown by the experimental inoculations already noticed (p. 901, *ante*). When numerous lymphatic glands suffer hyperplasia in consequence of the irritation of this poison, there is a more abundant production of white corpuscles (*leucocytosis*, VIRCHOW)—changes neither essential to nor characteristic only of syphilis. The belief in bacteria or fungi in the blood as the cause of syphilis has no foundation in fact. Nevertheless the blood contains *a something* specific, the nature of which has not yet been found out.

(3.) *In the Bones* syphilitic lesions may be arranged into three groups—(a.) The primary characteristic growths, or various states of *gummata*, which advance to the formation of ulcers; and the death of parts in little necrotic sloughs or cores of dead bone, imprisoned within cavities. There are usually several points of attack, where swellings form, accompanied by great pain, especially at night. Pains in the bones begin as early as the eruptive fever (to be noticed afterwards, p. 930), causing the patients sleepless nights. Such pains tend to occur in the regions of the joints, and are often considered rheumatic; but the joints themselves are not affected, and the points from which the pains really proceed are “certain” sensitive spots at the articular ends of the bones, especially the bony prominences to which muscles are attached (BURGER, BÄUMLER). The slightest periosteal swelling causes the greatest pain, especially those in the secondary stage. The sensitiveness of the affected part is often so extreme that even the pressure of the bed-clothes cannot be borne. The pain from this source has the peculiarity of occurring with the greatest vehemence at night. During the day it may be totally absent. Dr. Bäumlér gives the best physiological, and therefore the true, interpretation of these pains, from the clinical evidence of the occurrence of an *evening exacerbation of fever*, more or less constant, even only to a slight degree. The result is—(1.) Alterations in the fulness of the blood-vessels, which, gradually dilating, either occasion or aggravate the pains from determination of blood to those parts of the periosteum and bones which lie nearest to the skin—a swelling being thereby produced in the painful parts; (2.) The patients who have fever complain more of periodically recurring pains, than those in whom bone affections run their course without febrile symptoms; (3.) In the bone affections, also, the pains commence gradually to increase from the later part of the afternoon; and, (4.) The fact of the pains ceasing with an attack of sweating in the early morning hours, speaks in favour of such pains being related to the bodily temperature (Dr. BÄUMLER). In the earliest periods of syphilis the very painful, though scarcely perceptible swellings—as early as the eruptive fever—are due to a *simple periostitis*. *Gummy tumors* not unfrequently develop in the periosteum in the later stages of the disease. Their development is slow and painless. They may be small and circumscribed, or (as in the cranial bones) more diffuse. Such gummata or nodes are most apt to appear upon the skull, the shins, sternum, and clavicle; and generally upon such bones as are covered by skin only. The flat circumscribed elastic swellings of the cranial bones may be felt through the scalp. The soft and rapidly germinating tissue of which these tumors are composed is primarily furnished by the innermost layer of the periosteum. The new cell-growths arrange themselves in concentric rings round the blood-vessels involved. These are usually the periosteal vessels, and cross through the interval of new tissue, between the periosteum and the bone, and dip into its superficial portion. The connective tissue round these vessels furnishes the proper matrix of the *syphiloma*. Here the cells and basis substance are produced in successive layers—the younger pushing the older before them; so that the entire structure of the morbid growth is a concentric arrangement. The periosteum is soon separated more and more from the bone, which

wastes under pressure, becomes rough, and finally shows loss of substance. The syphilitic growth in its further progress continues to creep along the Haversian canals into the compact tissue of the bone, destroying territory after territory of its substance. When the bone tissue between any two Haversian systems is wholly destroyed, the masses from either side coalesce. All forms of otitis and periostitis may be set up in the vicinity of *syphiloma of bone*, by which it becomes more and more damaged and distorted. *Caries* and *necrosis* thus set up are not texturally different from the simple varieties of the same lesions. (b.) The virus of syphilis seems to have the power of fostering the growth of simple interstitial inflammatory products, and thus leading to *hyperostosis*, *exostosis*, and *hard nodes*. General thickening of the cranial vault is thus one of the most common lesions in syphilis. Caries and necrosis of the bone follow the eating ulcers of soft parts, such as the roof of the palate, the nasal septum, the laryngeal cartilages; or caries and necrosis may follow periostitis of the long bones. Internal caries and necrosis may also occur, beginning in the marrow of the bone, giving rise to suppurating osteo-myelitis. Most of these forms of syphilitic lesions are to be seen in the skull bones, especially in the forehead and anterior parts of the skull. They may be seen as superficial or deep exfoliations of the outer laminae. The deeper portions become dead in small circles, inclosed by new bone, after sclerosis of the diploë; and then existing like the core of a carbuncle, they cannot be removed without enlarging the aperture through which they make known their existence. (c.) *The dry caries of bone*, or cicatricial forms of lesion in bone, with considerable loss of substance, is recognised by numerous holes, or pores with furrows converging to a centre, where the lesion first began, as the *caries sicca* of Virchow, leading to superficial excavation and to a porous condition of bone; while, at the periphery a hyperostosis is in progress, so that a central depression (with or without a core) comes to be surrounded with a hard bony wall; giving rise to the stellate depressions so characteristic of this cicatricial-like loss of substance. Such cicatrices, which result after absorption or elimination of gummata in bone and loss of substance, have a characteristic appearance, especially denoted by the want of growth in the centre, and activity of growth at the edges. Thus an inflammatory lesion commences as an osteo-periostitis, and forms a swelling of a rounded form, merging insensibly into neighbouring tissue. The bony canals become dilated, and contain a substance like soft callus, into which small blood-vessels pass, so that the whole area of lesion becomes very red, congested, and vascular. Microscopically, the elements of the new growth are embryonic forms of connective tissue, as already described as characteristic of a *node*. These elements are very liable to the granulo-fatty metamorphosis, so that finally such lesions assume a cheesy, yellow, tubercular appearance, and so become almost entirely absorbed. If the new material does not become absorbed, but continues, then the thickness of the bones (such as the cranium) is considerably increased, and the tissue becomes dense and heavy. The periosteum may remain unchanged, except that it is increased in thickness; but generally nodes form upon it, with circumscribed spots of inflammation, disappearing in a week or two, to be

followed by others in the vicinity. These nodes tend to suppurate, and to caries of the bone beneath, in small portions. The ulcer generally cicatrises without spreading, leaving the surface of bone uneven below; and the cicatrix adheres to the bony tissue. The tibia, the clavicle, the elbow, the bones of the cranium, nose, and all superficial bones, are the seats of selection of syphilitic inflammation. Gummy tumors occupy either the periosteum or the bone itself. In the medulla of the bone the changes proceed from hyperplastic cell-growths, as in hereditary syphilis; but while in the medullary cavities of the long bones the osteo-myelitis appears in the form of circumscribed tumors, in the spongy tissue of the flat bones, especially those of the cranium, the medullary gummy growth appears in the diploë as an infiltration, separating the laminæ of the compact substance, and finally causing caries or necrosis of small portions of bone. Hence absorption, suppuration, caries, or necrosis are the various modes of termination: exfoliations occur frequently on the skull, and the sequestrum is marked by great porosity corresponding to the processes which led to its separation (Dr. BÄUMLER). Hence also in the necrosis of gummy lesions of bone, the dead portion has a worm-eaten appearance, with large holes over a plain surface. Atrophic forms of bone lesions—dry caries, bony cicatrices—are usually seen on

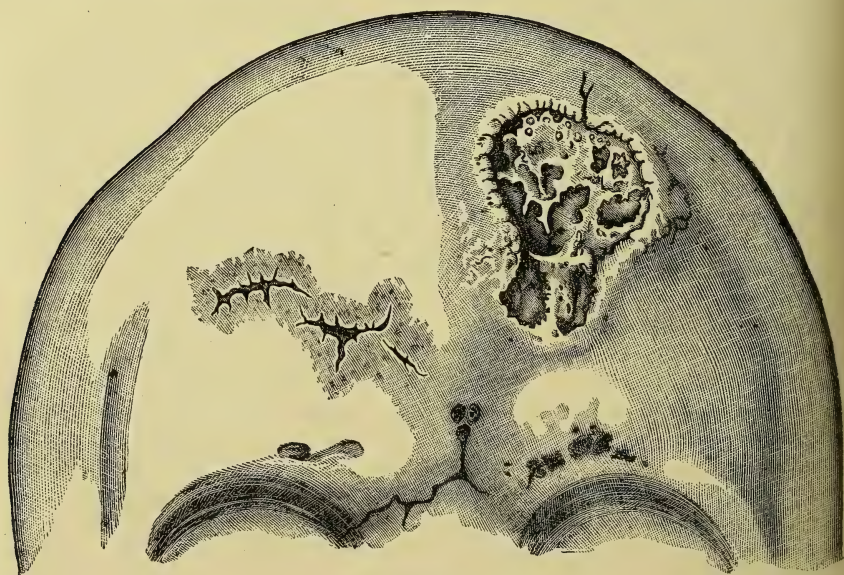


Fig. 97.*

the cranial bones, associated with gummy tumors of the periosteum. The lesion is very slow and insidious in its origin and progress, and is never observed till it has existed for a considerable time. It exists, simultaneously, at several points of foci, characterised at the centre by

* Bone showing syphilitic caries of the *os frontis*. The indented circumference is seen in the larger spot, and the cicatriform character in the smaller (after Dr. WILKS).

a process of thinning, and at the circumference by a process of hypertrophy. The thinning is due to dilatation of the vascular canals, with absorption of bone tissue; large orifices, pores, and furrows converge to the affected point. The depression has a fissured appearance, like some of the cicatrices of the skin and mucous membrane. These stellated depressions increase in circumference and depth, and sometimes the bone is slowly perforated. *Osteophytes* surround the lesion. This form is believed to be the last stage of gummy deposits which have been absorbed.

III. *Visceral Lesions*.—After a period of latency of very variable duration, symptoms may again appear, but which have been so long deferred, that the former attack, particularly if it was a mild one, has long been forgotten. It is only during the last fifteen years (since about 1864) that these later symptoms (to be afterwards described) have been regarded as syphilitic, and referred to their proper source; and even now their true nature is often misapprehended. Many of them have so much the character of local affections, that they are regarded by some as sequelæ of syphilis (JONATHAN HUTCHINSON, Sir JAMES PAGET). The visceral local affections are peculiar on account of the marked tendency to tumor development, not usually shown in the earlier manifestations of syphilis; and the growths do not show that disposition to undergo reabsorption which is characteristic of the inflammatory symptoms of the previous stages of the disease; but a tendency rather to disintegration and necrosis. These growths may occur in any organ. They present at first hard elevations, afterwards gradually softening, and named on account of their gummy-like, semifluid, greyish-yellow contents—“*gummy tumors*” or “*gummata*.” They have been already described in the skin, mucous membrane, subcutaneous areolar tissue, periosteum, and bone, whence their softening and discharge are usually in the direction of the external surface; but in the internal viscera the periphery of the tumors becomes thickened into a fibrous envelope of connective tissue, inclosing the contents of the original *gumma*, which have now become condensed, fatily degenerated, and dry. The internal viscera most commonly attacked are the liver, testicles, brain, and membranes. The *gummata* in these viscera appear to proceed from the connective tissue, and especially from the adventitia of the blood-vessels. They occasion symptoms of great variety and gravity, depending on the organ affected and the seat and extent of the tumor (Dr. BÄUMLER).

Much has been done in the Pathological Society of London, especially by Dr. Samuel Wilks and others, not merely in bringing forward evidence of syphilis in regions where it had not been recognised before, but in contending against the obstructive scepticism of some pathologists who attempted to undervalue the evidence, and to ascribe to other causes such visceral syphilitic affections as were continually being brought forward by Dr. Wilks and others. His monograph on *Syphilitic Affections of the Internal Organs* in the third series of *Guy's Hospital Reports* for 1863, has done much in this country to advance our knowledge regarding the pathology of syphilis.

(1.) *Syphilitic Lesions of the Liver*.—Dittrich and Gubler were the first to give an accurate description of the syphilitic lesions in the liver; and in 1855 Mr. Busk translated Carl Wedl's *Rudiments of Pathological*

Histology, in which he gives (p. 432) an account of the cicatriform tissue on the surface of the liver, which may extend deeply into the parenchyma after inveterate syphilis, as described by Dittrich. From that date the study of visceral syphilis may be said to have begun. Virchow has also described a perihepatic lesion and a simple gummy interstitial hepatitis. The former never occurs alone, but is generally associated with the latter. The hepatic substance atrophies, and the deposit contracting is eventually absorbed, causing a cicatrix-like mark. The liver lesions are usually among the later symptoms of syphilis, and are well described by Wedl, Virchow, Wilks, Frerichs, Bäumler, Moxon, and Grainger Stewart. The anatomical alterations embrace several distinct processes, which are often variously combined with each other:—(1.) An indurative overgrowth of connective tissue, leading to a form of cirrhosis (syphilitic, as distinguished from alcoholic); (2.) The development of the specific gumma; (3.) Extensive fibroid infiltration with gummata; (4.) Gummata with cicatricial patches; (5.) Cicatrices and fibrous bands; (6.) Lardaceous infiltrations. The most frequent morbid appearance is where one or more deposits of tough white connective tissue are found in the parenchyma, in conjunction with analagous changes in other parts, such as cranial bones, pharynx, larynx, skin. These deposits send radiating prolongations into the surrounding parenchyma in various directions, which are followed by fissuring and lobulation of the surface in large masses, corresponding to and caused by the vigorous contraction of the cicatricial-like tissue—*syphilitic cirrhosis*. These appearances are most marked along the attachment of the suspensory ligament. In these regions three or four stellate cicatrices are often seen; or the entire thickness of the liver may be traversed by a single fibroid band of very large size. *Interstitial hepatitis*, or *syphilitic cirrhosis*, requires to be distinguished from *alcoholic cirrhosis*. The *syphilitic cirrhosis* sometimes involves a part only, and sometimes the whole of the liver. It is associated with—(a.) Vascular injection; (b.) In the fibrous capsule and main septa there grows, at various points, the new tissue, which thickens the web, and leads to enlargement; (c.) Contraction ensues as a result of the retraction of the new tissue. Hence furrows, more or less deep, cause the characteristic appearances which stamp the lesions as syphilitic. Very remarkable changes of form are thus to be observed and explained in such livers. Deep cicatricial-like contractions divide the organ into a number of distinct, roundish lobes or prominences, the liver at the same time becoming much diminished in size—reduced, sometimes, to a nodular mass of small dimensions. When cut into dense bands of fibrous connective tissue, that grate under the knife, are found corresponding to the external constrictions, inclosing at some points masses which have undergone cheesy degeneration, similar to those left by gummy tumors; (d.) *Perihepatitis* is invariable; hence adhesions are common to diaphragm and other parts. *Alcoholic cirrhosis* progresses always along the course of the portal veins, and the liver is never ploughed up into large furrows, so characteristic of syphilis. The *syphilitic depressions* and cicatrices are also sometimes due to absorption of gummy nodes, or to partial hepatitis. *Gummatous nodules* of various age and size very often occur in the substance of or near this fibroid tissue. More rarely they are found alone

amid healthy parenchyma. On section, they are white, grey, or yellow, firm or a little soft, having a dry, sapless, and elastic surface. Generally they are surrounded and circumscribed by a thickened retractile zone, less prominent on section than the node itself, on account of this retraction. Sometimes we find them still young and soft, in which the characteristic anatomical products may be recognised. These are corpuscular cells—round, stellate, oval, or spindle-shaped—often undergoing fatty degeneration, and persisting in the form of granule cells. Round cells occupy the centre; the stellate and spindle-shaped ones are round the periphery of the nodules. Hence it is probable that gummata may continue to grow at the periphery, on account of the continued germination of the nucleated, interstitial, and interlobular tissue, while at the centre and intermediate zone degeneration has commenced (J. E. PAYNE, *Trans. Path. Soc.*, Vol. XXI., 1870, p. 207). The cheesiness is a mark of chronicity. Sometimes they soften and are reabsorbed; but more often remain latent, imbedded in the connective tissue. Sometimes these nodules are small, multiple, deep-seated foci (*miliary syphiloma*)—thirty to sixty throughout the whole liver. Sometimes from five to ten gummata, like millet seeds, unite into a small tumor, held together



Fig 98.*

by tough connective tissue, having a basis substance as transparent as cartilage, and sending processes radiating to a variable distance in the neighbourhood. These gummata are always situated in the portal canals, where arteries, veins, bile-ducts, and lymphatics run side by side. When special vessels are capable of being seen involved, they have been found to be lymphatics (RINDFLEISCH). When superficial *gummy tumors* exist, the liver generally adheres to the diaphragm or other parts

* Section of liver showing a large deposit, with a cicatriform appearance of the surface, and with several other smaller nodules round it (after Dr. WILKS, l. c.)

in the neighbourhood of the superficial growths. The surface of the organ is generally traversed by cicatricial furrows and deep depressions. The true gummy tumor of the liver (*large nodular syphiloma* of Wagner) is an irregular circumscribed mass, from which prolongations extend into sound hepatic tissue, with greyish-yellow cheesy deposits scattered through it—more sharply defined the denser the connective tissue. Sometimes it takes the form of a well-defined tumor of a roundish form, inclosing a dry, firm, cheesy matter in a fibrous capsule. *A third variety* of syphilitic liver is that of hereditary cirrhosis—met with in new-born children and infants of syphilitic parents. It is a *diffuse infiltration*, first described by Gubler, marked by an increase in size and weight of the liver. Its colour resembles flint, and its acinous structure is more or less obliterated. In children from ten to fifteen years of age typical syphilitic cirrhosis is sometimes seen; and in these cases there is complete obliteration of the branches of the portal vein in the parts involved. It is presumed that these cases are examples of inherited syphilis. Compensatory arrangements, in such cases, regulate the portal circulation—a circumstance that can only be accounted for, or understood to occur, while the liver was still in process of development. Another form of lesion of the liver is lardaceous disease, which may involve the whole organ or parts of it (see page 129, *ante*); and acute yellow atrophy of the liver may also be consequent on syphilis.

The *symptoms* of syphilitic affections of the liver are especially referrible to the size and form of the organ, to derangements in the portal circulation, the escape of the bile, and to involvement of the peritoneal coat. Increase of size is constant in the diffuse hepatitis, alike of hereditary and acquired syphilis, and in lardaceous disease involving the entire organ. When contraction takes place, the symptoms are those of *cirrhosis*.

(2.) In the *Digestive Tract*, *lesions of the TONGUE* in the later stages of syphilis are to be seen in many cases. They are mostly expressed by ulcerations at the base, often in such positions that the laryngoscope only enables them to be seen. Sometimes they appear as a raw, indolent, abraded-like surface, in circumscribed patches, on the dorsum or edge of the tongue—the aphthous exfoliation and syphilitic tubercles of Erasmus Wilson. (See Plate 3, Fig E, of his work on *Syphilis*.) Gummy nodules may also develop, especially out of an ulcer set up by irritation from the sharp edge of a tooth; or, they originate in the substance of the tongue. The



Fig. 99.*

TEETH suffer, and are subject to various deformities in the upper central incisors—a diagnostic mark of an inherited syphilis (Fig. 99) (JONATHAN HUTCHINSON). The condition is accompanied by *stomatitis* in early infancy. In the LARGE INTESTINES an ulcerative process, attended with dysenteric symptoms, is sometimes a terminal affection (BÄUMLER). The RECTUM is very commonly the seat of a dysenteric ulceration, gradually leading to stricture.

(3.) In the *respiration tract*, the LUNGS have long ago been described as the site of syphilitic lesions by Morton, Sauvage, Portal, Morgagni, and more recently by Graves, Stokes, Walshe, Wilks, Virchow, Lebert, Ricord,

* Upper incisors stunted in growth, with convergent lateral borders (after Mr. JONATHAN HUTCHINSON).

Munk, Varen, Ziemssen, Bäumler, Weber, Moxon, Förster, and others. Two forms of lesions of the lungs are recognisable,—(a.) *Evidences of Inflammation of the Mucous Membrane of the Bronchial Tubes.*—Bronchitis (or at least bronchial irritation), with fever, in many cases precedes the skin lesions, and disappears wholly or partially when this is established; and if the syphilitic eruption suddenly disappears, bronchitis may again ensue. Walshe records well-marked instances of this; and it is a circumstance to be looked for amongst soldiers especially, who, having recovered from a primary syphilitic sore, are apt to be exposed to the risk of bronchitis when mounting guard soon after being discharged to duty. Secondary symptoms and pulmonic lesions are then apt to date their commencement. All the general symptoms of phthisis may supervene, and yet no tubercle in the lung may be developed; but chronic bronchitis remains persistent. On this point Dr. Walshe observes that in the persistence of the general symptoms there “is assuredly enough to create a strong suspicion of the existence of tubercle in the lungs, taken in conjunction with the indubitable tendency of syphilis, *plus* mercury, to induce the outbreak of pulmonary phthisis. How are the cases to be distinguished? By the total want of accordance between the physical signs and the constitutional symptoms; the patient with syphilitic bronchitis has neither consolidation signs nor, *à fortiori*, the evidences of excavation. But there is a source of difficulty which may sometimes arise in these cases and render doubt imperative—the infra-clavicular ribs and clavicle thicken from periostitis, and produce dulness under percussion, which cannot with positiveness be distinguished from that of tubercle within the lung. Here the observer must wait for events to clear up the diagnosis” (*Diseases of the Lungs*, p. 233). (b.) *The Occurrence of Gummatous Nodules in the Pulmonary Substance.*—These are, in the first instance, of the same histological constitution as the well-known node of the shin, or the subcutaneous product described by Ricord, Bärensprung, Virchow, McCarthy, and Bäumler. They form especially towards the periphery and bases of the lungs. In the former site they resemble nodules of lobular pneumonia. They may soften and be eliminated much in the manner of tubercle, although they may have at first a consistence like scirrhus. It is concerning those which soften in this way that Ricord gives the warning “not to confound suppuration of a few syphilitic nodules of the lungs with phthisis.” Regarding those gummatous nodules Dr. Walshe observes:—“I can find no positive answer to the query, Do these gummata ever form independently of other tertiary evidences of syphilis in the bones and cellular tissue? If they do, their diagnosis must be infinitely difficult—difficult, indeed, under all circumstances; for the physical signs can be none other than those of solidification, followed by softening and excavation, while the local and general symptoms closely simulate those of phthisis” (*Diseases of the Lungs*, p. 431). (c.) *The Occurrence of Gummatous Nodules in various Stages of Growth and Degeneration, associated with the Deposit of Miliary Tubercle.*—In such cases the history of events in the illness of the patient may be found to correspond more or less closely with the appearances seen in the lungs—appearances which distinctly indicate the formation of lesions commenced at different dates—appearances which denote the occurrence of lesions in crops, or as a succession of events which may be

illustrated by the history of the symptoms during life. For reasons already stated pulmonary phthisis must be regarded in many cases as the product of syphilis; and I would fully endorse the statement of Dr. Balfour, from what I have seen in the *post-mortem* rooms, when he says that a great cause of pulmonary disease among the Guards is the amount of syphilis which prevails amongst the men, which he has not the least doubt is a very fertile cause of its being called into active operation. The influence of syphilis on the health of the soldier is indeed powerful for evil throughout the whole army.

(4.) *In the substance of the HEART* Virchow describes the syphilitic growth, and refers to cases of a similar kind recorded by Ricord and Lebert. Ricord, in his atlas, gives illustrations of them, and calls them "syphilitic muscular nodes in the substance of the heart" (*Clinique Iconographique*). Firm, yellow, cheese-like masses were found in the substance of the ventricles. There was a history of old chancres and ulcerated tubercles of the skin. In Lebert's case these gummata were seen at a comparatively early stage of development, and were found in the wall of the right ventricle. There were tubercles of the skin, of the subcutaneous tissue, genital organs, and bones of the skull (*Anat. Pathologique*). In Virchow's case there were syphilitic gummata in the testicles. Dr. Haldane of Edinburgh records a case of a similar kind, and gives excellent drawings in *Edin. Monthly Med. Journal* for Nov. 1862. In the Museum of the Army Medical Department at Netley there are two preparations which show such gummata in the substance of the heart. One occurred in the case of a soldier twenty-four years of age, under treatment for venereal ulcers of nine months' duration, on various parts of the body. He had lost his palate, and eventually sunk from exhaustion, with symptoms of phthisis. Sections of the muscular substance of the heart showed several isolated deposits in its substance and beneath its serous covering. Isolated portions of the lungs were also converted into a substance of the consistence of cheese. Gummata are most commonly found in the septum between the ventricles, but they are also found in varying numbers in all parts of the muscular tissue, varying in size from a pea to a pigeon's egg; and if they are situated near the endo- or pericardium, these membranes are thickened and often as hard as cartilage (BÄUMLER). The lesion also often assumes the form of *indurated interstitial myocarditis*. White fibrous tissue abounds in the left ventricular layers of muscle, and the heart is usually larger. Round nuclei abound in the sarcolemma or connective tissue. Vascularity increases, yellow deposits follow, the result of fatty degeneration of the new growth; and degeneration passes ultimately to the muscular fibres of the heart. The lesion is to be distinguished from the *myocarditis of rheumatism*, and from that of *alcoholic myocarditis*. In *rheumatism* the lesion is usually limited to orifices and valves. In *alcoholism* the adipose tissue is in excess at the base of the heart, is of a yellow colour, and fatty degeneration extends from this between the fibres of the heart.

(5.) *Lesions in the common muscles* generally also occur in two forms—namely, *interstitial myositis* and *circumscribed gummy node*. The *interstitial myositis* shows itself most frequently in the flexor muscles of the fore-arm. The gummy nodes appear most often in the *gluteus maximus*, *trapezius*, *sterno-mastoid*, and *vastus externus*.

(6.) Lesions in the BLOOD-VESSELS are of interest as the indirect cause of aneurism from the atheromatous conditions to which they lead. Circumscribed thickenings, with considerable elevation of tissue, occur in the aorta in circular patches as large as a florin. Lardaceous disease of the smaller blood-vessels is also a lesion which, in some way yet unknown, is yet a result of syphilis. The tendency to aneurismal dilatations and cicatrix-like loss of substance in the lining membrane of the great vessels in *young* subjects, who are or have been severely affected with syphilis, is a point in morbid anatomy which requires yet to be investigated.

(7.) Among the *Genital Organs* the syphilitic lesions of the TESTICLES (*syphilitic sarcocoele*) appear in two forms, namely,—(a.) Well-marked induration from interstitial growth of tissue. Where the general substance of the testicle is affected, the deposit is interstitial—a simple indurated interstitial growth; and the free portion of testicle is first attacked; then the *tunica albuginea* thickens, and the inflammation extends along the tubes. The disease generally begins in the *tunica albuginea*, and induces germination of new connective tissue and young cells upon the interior of the tunic and between the seminal tubes. The induration destroys large tracts of the parenchyma. A hyperplastic growth of young connective tissue is followed by its condensation. With the naked eye the fibrous white bands can be easily seen,

often conoidal in shape, as determined by the original lobular segmentation of the organ. The base of each cone lies in a similarly thickened albuginea—its apex directed to the *corpus Highmorianum*, where it blends with the apices of adjoining cones. In extreme cases nothing is to be seen beyond a continuous mass of white fibroid tissue—the *tubuli seminiferi* having gone with every trace of the original segmentation of the gland. The middle cones of the testicle are most frequently affected, and they increase in size from this proliferation of tissue-elements. The interstitial tissue softens and is red, the tubes thicken, fatty epithelium becomes developed, and atrophy results. (b.) Solitary nodules (gummy tumors) sometimes form, varying in size from a millet-seed to a cherry. Several of these, or only one about the size of a cherry-stone, may be present. They look like yellow tubercle, and are analogous to the gummata observed in the scrotum and in the substance of the tongue, which are hard and elastic, about the size of a pea, and easily overlooked. These tumors in the testicles (Fig. 100) and tongue are peculiar in not growing from a centre, like other tumors, but rather as infiltrations of tissue. (WILKS). Thus they are not per-

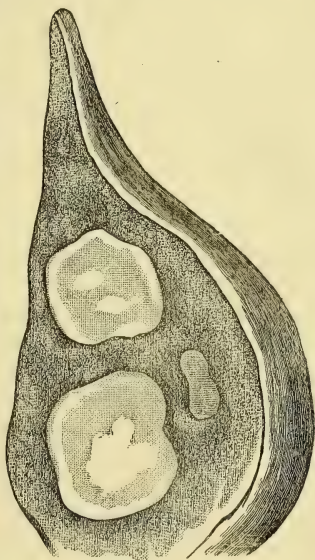


Fig. 100.*

* Testicle, showing several syphilitic deposits (after Dr. WILKS).

fectly circumscribed, but are found mixed up with the adjacent tissues (*gummy orchitis*).

The lesions of the testicle occur at a late stage of acquired syphilis, and syphilitic sarcocele is very rarely seen as a symptom of inherited syphilis. It is more often associated with gummous affections of the bones, and with tubercular or ulcerative diseases of the skin. It does not appear sooner than ten months after syphilitic symptoms, more often many years intervene before it appears. Its progress is slow and insidious, and the patient only becomes aware of it after considerable swelling has taken place. It causes little pain, and the tumor is but slightly sensitive to pressure.

(8.) *Affections of the Nervous System.*—Here also *syphilitic gumma* denote the advanced or tertiary stage of constitutional syphilis. The lesions in the BRAIN are—(a.) Gelatinous degeneration—a myxoma of the perivascular sheaths penetrating into the brain from the *pia mater*, and destroying the nervous parenchyma between the blood-vessels. The thickened vessels and new growth then coalesce to form a pale reddish-grey tumor, which occupies a superficial depression. (b.) Gummata are usually found near the surface of the brain, at its base; and more rarely in the interior. They attain the size of a walnut, or even a hen's egg. Caseation takes place in their interior, often at several points or foci; while the circumference remains of a soft, jelly-like vascular tissue. Sections show a highly corpuscular parenchyma concentrically arranged round blood-vessels. The growth advances by infiltration along the perivascular sheaths in the interspaces which lodge the vessels. The gumma of the brain are generally surrounded by every stage of softening, with inflammatory or apoplectic changes. Such gummatous tumors in the brain have been especially described by Bonet, Ricord, Cullerier, and Lallemand. Ricord describes them under the name of syphilitic tubercle of the brain. Dr. Steenberg (Physician for the Insane at Schleswig) believes that a great proportion of the syphilitic affections of the brain are subsequent to lesions of the arteries, and the organs of circulation, generally, he observes to be the frequent seat of syphilitic localisations. Hence softening of the cerebral substance, and various lesions of the nervous system, are by no means rare in cases of prolonged syphilis. Virchow has also frequently noticed lesions of the great vessels in those who die from syphilis with lesions in the brain. In the cases where cerebral symptoms have long co-existed with syphilis, "a quantity of tough, yellow, fibrous tissue unites together the surface of the brain with the adjacent membrane, and this again is adherent to the bone. The cortical substance of the brain at the affected spot is often partly destroyed, and the adventitious material occupies its place. The question has still to be solved as to what structure is primarily affected. Many have given the authority of their name to the opinion that the disease commences first in the bone, but simply for the reason that the osseous system is that which has so long been recognised as liable to be affected. But since we now know that other structures may be similarly attacked, we are prepared to look for its commencement in other parts, even in the brain structure itself. . . . The cases which are so frequently met with are those where the deposit involves both sides of the *dura mater*, and includes in it the bone on one side and the brain on the other.

The probabilities are in favour of its occurring in the *dura mater* first, as it arises in the periosteum on the exterior of the cranium" (WILKS in *Med. Times and Gazette*, Oct. 25, 1862). But the lesion also occurs in isolated gummatous nodules in the great nervous centres, such as the *thalami optici* or *corpora striata*. I saw, on the 29th May, 1863, a most interesting dissection of such a case in the Middlesex Hospital, which had been under the care of Dr. Goodfellow. There had been a history of syphilis, and some of the children of the man had died of inherited secondary syphilitic lesions. A gummatous tumor occupied the left *optic thalamus*. Numerous cases of syphilitic tumor of the nerves and nervous centres are to be found collected together in the pages of the *Medical Times and Gazette*, and two may be referred to of the intracranial nerves, related in the seventeenth volume for 1858, p. 419, in each of which paralysis was due to such syphilitic *neuromata*. Dr. Thomas Reade, of Belfast, also published in 1871 a most interesting monograph "On Syphilitic Affections of the Nervous System." Lesions are also described in the ganglia of the *sympathetic*—hypertrophy of the interstitial connective tissue, inducing atrophy of the nerve fibres and ganglion cells, with marked pigmentation and colloid degeneration (PETROW, VIRCHOW'S *Archiv.*, 1873, p. 121). The lesions of encephalic syphilis are ushered in by obscure phenomena; but the following may be especially noticed:—*Insomnia* manifests itself at the commencement; and *headache* is characterised by—(1.) Its great severity; (2.) prolonged duration; (3.) nocturnal recurrence or exacerbation. The *general nervous symptoms* are especially obvious in alteration of intelligence, of sensibility, and of motion. These, combined with such obvious local lesions as caries, or necrosis of the facial bones or of the cranium; or tumors on the external surface of the cranium, such as gummata, periostitis, or exostosis, at once point to cerebral syphilitic lesions. They are sometimes also expressed by persistent epilepsy. Unilateral convulsions with fixed pains and paralysis, are regarded by Dr. Hughlings-Jackson to be pathognomonic of syphilis. Any form of syphilitic infection may be followed by nervous affections, from a year old up to old age. Syphilitic brain-disease generally leads to *softening* of the cerebral substance surrounding the nodule; and this softening cannot be distinguished from the softening induced by any other cause. The duration of syphilitic nervous affections averages about one year; and their natural course is characterised by intermissions; and at the outset the intermissions are very distinctly expressed.

(9.) *Lesions* in the organs of the *special senses*:—(a.) In the EYE. The syphilitic lesions capable of affecting, directly or indirectly, the sense of sight are numerous and varied. They involve either the optic nerve itself or the constituent parts of the eye—e.g., *choroiditis*; diffuse exudation throughout the *retina*; atrophy of the *optic nerve* and its *papilla*, with diminution in the calibre of the central vessel; circumscribed abscesses, or partial softenings developed in the course of the optic nerve; opacities in the lens, changes following *choroido-retinitis* (VON GRAEFKE). Of great practical importance is an affection of the *cornea*, which has been shown by Mr. Jonathan Hutchinson to be almost exclusively found in cases of inherited syphilis. It is described under the name of *interstitial* or *parenchymatous keratitis*. It declares itself most frequently

between the ages of nine and fifteen—and in girls oftener than in boys—associated with syphilitic indications in other parts. It may be found in such cases that the mother has had miscarriages or still-born children previous to the birth of the one suffering from *keratitis*; and also that the patient has suffered from other symptoms of syphilis during infancy, such as *snuffles*, or sore mouth, ulcers round the *anus*, skin eruptions, and the like. The physiognomy of such patients is characterised by an old, pale-looking face—a squarish forehead, with the bridge of the nose frequently wide and depressed. The shape, size, and colour of the permanent incisor teeth are often also peculiar (see page 918), being small, narrow, squared, of a yellow colour, and more or less deeply notched in a vertical direction. *Syphilitic keratitis* generally appears in both eyes within an interval of a few days or weeks; and may take a chronic course, continuing for several years—at one time being completely opaque or crimson red, and again recovering its transparency after a few weeks. It is generally on account of impaired sight simply that such patients seek advice; and the corneal changes may be so great as to reduce vision to a mere perception of light from opacities, or from alterations of its curvature. The vascularity of the ocular conjunctiva is but slightly increased, while a more or less broad pink zone in the sclerotic round the edge of the cornea is usually present. The centre and lower half of the cornea are the first parts to lose transparency. Small, ill-defined, grey, yellowish, or brownish opaque dots, in groups or singly, appear in its lamellated structure about these parts. Its surface appears dull or slightly uneven, as if pricked with a pin in numerous places. Within a few weeks transparency may be so impaired that the cornea looks like a piece of ground glass, the depending portions being the more densely opaque, or of a pink or crimson hue, most intense at the surface and periphery, caused by blood-vessels passing from the substance of the sclerotic between the lamellæ of the cornea. The vessels tend to run parallel. If the syphilitic infection is severe, or if health be feeble, lymph is abundantly developed in the cornea and iris, rendering the cornea yellow and the iris opaque. Permanent opacity may follow their adhesion, occupying most frequently the lower half of the cornea. Ulceration never occurs. Usually the cornea begins to clear up gradually, from the circumference towards the centre; and from five to seven months, or perhaps a year, may elapse before it becomes again quite transparent. Cloudy or striped opacities may even then be left, or the curvature of the cornea may remain permanently altered. Inflammation of the sclerotic is also often referable to *syphilis*. It is generally circumscribed, and associated with inflammation of the conjunctiva and subconjunctival tissue. It generally commences in the ciliary region, where it forms patches of a purple tint, covered with enlarged vessels. The textures appear swollen, and in from five to ten weeks the inflammation may subside, leaving the sclerotic slightly discoloured, semi-transparent, and thinner. Such circumscribed inflammations often appear successively in adjoining parts of the ciliary region of the sclerotic. When iritis occurs in the later stages of syphilis, it may be several or many years after first infection; and it is then generally a recurrent attack, to which the synechiæ left render the iris peculiarly liable. Important changes may also occur in the deeper

portions of the eye. (b.) In the EAR, catarrh of the middle portion may lead to deafness by perforation of *membrani tympani* and purulent infiltration of the cells of the mastoid process. Such lesions are especially the result of inherited syphilis (JONATHAN HUTCHINSON). Moist papules, also, sometimes develop in the external auditory meatus.

Symptoms.—After infection with syphilitic virus a *period of incubation* has been determined by experimental inoculation upon persons who have not had syphilis, and by clinical observation. *Generally, from three to four weeks may be taken as the length of this period of incubation for syphilis—i. e., the period elapsing from the infection until the first appearance of local symptoms.* In two cases observed by Dr. Bäumlér, the periods could be precisely determined to the day, and gave twenty-five days and twenty-nine days respectively. Dr. Berkeley Hill's observations gave, as the most common periods, twenty-five and twenty-eight days. In a large number of exact cases (thirty-one), from experimental inoculation calculated by Bäumlér, the shortest period of incubation was ten days—the only instance of a period less than sixteen days. Most frequently the incubation lasted between fifteen and twenty-four days; in a small number of cases from twenty to thirty days; and only in four over thirty. The longest period was forty days. *From clinical observation* Fournier gives the period as oftener over than under three weeks; and not unfrequently it is four, and sometimes six weeks; and in one case it was seventy days. Von Sigmund gives from forty-two to fifty-six days; while in the vast majority it was over two weeks, and not over four weeks.

The forms of primary syphilitic or infecting sore ultimately vary; but the first sign is to be observed at the point where the poison gained entrance, either by contagion through sexual intercourse, or by experimental inoculation. A circumscribed redness may be seen, and shortly afterwards an elevation of the spot into—(1.) a flat papule, the subsequent course of which differs somewhat as it occurs upon skin or upon mucous membrane. *The dry flat papule* is the rarest form seen by the surgeon, because it usually gives no sign of local trouble, for usually that stage has passed before advice is sought. It is a *flat papular protuberance*, varying in size from a *pin's head* to that of a *sixpence*, at the point of entrance of the poison, of a dark-brown, red, or purplish colour, round or oval, *firm and elastic*, sometimes covered in from eight to ten days with *white scales of epithelium* or scurf—hence, sometimes called a *desquamating papule*. The induration and the papule sometimes both disappear by resolution or absorption, without any ulceration whatever, just as gummy tumors or nodes disappear in the same way. The induration loses its resistance and elasticity, diminishes in extent and volume, becomes gelatinous, finally subsides, and leaves behind a slight violet, copper-coloured, or black depression. These are the most insidious cases; and when secondary phenomena appear, the existence of a primary sore is very generally denied, for as a papule and not an open sore existed, the lesion may never have been considered of sufficient importance to attract attention. It may, in fact, never have been seen or noticed in any way by the patient. (2.) *The syphilitic or hard chancre erosion.*—This is the most frequent form in which primary syphilis presents itself. *Patchy excoriation, or superficial ulcer* of primary syphilis, or *parchment-like chancre* (Ricord's *chancre parcheminée*)

are other names by which this form has been described. It commences as a copper-red spot, scarcely raised, papular, and dry. It is covered with a crust or thin scales, after desquamation of which the centre becomes a little moist, and the secretion dries again into a crust, on the removal of which the underlying surface is of a bright red colour, and shining as if it were glazed. A very little pus is present; and when a moist dressing is applied to prevent a crust forming, a very scanty secretion is the result, of a grey shiny deposit, adhering to the centre of the flat papule, which is of a livid colour. The ulceration is circumscribed within the induration, and presents a *flat* livid-coloured surface, projecting on a level with the summit of the swollen part; and it is prominent in proportion to the amount of increasing volume and induration. If it is pinched up between the finger and thumb, it imparts a feeling "as of a hard disk, bevelled at the edges and set in the skin," or as if a bit of parchment had been inserted beneath the surface of the ulcer. The induration is superficial rather than deep. It continues to discharge a small quantity of serous fluid from a diffused base. This sore is often so slight, the discharge so little abundant, cicatrization, when it begins, so rapid that, in the absence of induration of the sore or of the cicatrix, prognosis must be doubtful, till some secondary result demonstrates contamination of the system. If left to itself the ulceration and induration may continue to extend, and then remain without change for a lengthened period, before resolution begins; but usually this lesion lasts about two months, terminating by resolution and cicatrization, and generally leaving a slight induration, with the corresponding ganglia hard and indolent.

Upon the *mucous membrane* the course of the primary papule is somewhat different. In protected spots, such as the *sulcus coronæ glandis*, after the period of incubation has passed, a very small itchy vesicle upon a reddened base is seen, or an erosion due to the bursting of this vesicle, which cannot be distinguished from the erosion resulting from a simple herpetic vesicle (BÄUMLER). It looks so trifling that many persons scarcely notice it, especially if some weeks have elapsed since infection. Moreover, it soon granulates, and may cicatrix completely, with the gradual disappearance of induration; but more commonly the induration remains or continues even to increase.

Similar forms of the primary syphilitic sore are to be observed in *females*; but in women compared with men, the flat papule is still more rarely seen as the primary form of "infecting" sore. A hard, open chancre or sore in them is exceptional; and when it does occur, it remains small, is ill-developed, slightly indurated, has a brief duration, and is readily overlooked, even when searched for with great care, aided by a vaginal examination with the speculum. In them the primary lesion which *infects* the system is generally a papule rarely giving any sign (SIGMUND, CLERC). Another peculiarity connected with the "*infecting*" sore in women is, that such papules are apt to form along the course of the superficial lymphatics; these may afterwards become eroded into a long fissure, from which issues a virulent infectious discharge. By a large number of observers induration in the female genitals is said to be of rare occurrence; or so inconsiderable and obscure in cases followed by constitutional syphilis, that in these the primary lesion may

be difficult to discover. Bäumler does not concur in this opinion. In the vast majority of cases in females he found the primary affection as a dry or ulcerating induration upon one of the *labia majora*, or else as a simple parchment-like thickening upon the borders of the *labia minora*, or in the form of several papules in process of conversion into flat condylomata. The induration in females may not be felt, because in certain places (e.g., *introitus vaginæ*) it is impossible to make the examination by touch in the proper manner (FOURNIER). Mr. Cooper Forster is also of opinion that indurations in women are more common than has been supposed. They are to be looked for in various situations in the external organs, and also upon the *os uteri*. They occur in the form of dry or moist papules (*condylomata lata*), or extensive parchment-like thickenings, or of merely small or superficial erosions, in which a slight degree of hardness may be felt by pressing from the sides. Moreover, in females multiple venereal affections are most apt to occur. A soft chancre may appear upon the vulva, while a syphilitic primary sore may have its seat upon the *collum uteri*, where it might be easily overlooked. Indolent glandular swellings in women may thus often come to be of importance as a guide to the situation of a primary syphilitic lesion.

In the female genitals simple *erosions*, which the patient herself notices, with parchment-like thickening, are of more common occurrence than the tubercular-like indurations—commonly on the edges of the *labia minora*, or upon the *præputium clitoridis*. But where the mucous membrane is kept moist by opposition of surfaces, the parchment-like indurations are gradually transformed into moist hyperplastic growths—*condylomata lata*. The primary affection in women has its seat most commonly on the *labia majora* or *minora*, or in the *posterior commissure*, and extremely seldom in the vagina. (3.) The indurated sore of syphilis—*non-suppurating chancre*—*Hunterian chancre* (*ulcus vallatum*).—Induration is the primary lesion, first as a papule, over which a crust may form, and underneath this crust a cup-shaped ulcer of greater or less depth rapidly develops itself. This sore is *indolent in its progress*, and has the appearance of being scooped out; it presents raised and rounded edges, a glossy iridescent surface, a base generally greyish or lardaceous-like, bathed with a serous or watery-like secretion, not pus, and not reinoculable on the patient who carries it. This is the most characteristic lesion of commencing syphilis. The induration which forms the bed of the lesion and base of the ulcer, extends beyond its circumference, and has been compared to the half of a dried pea as to hardness. It is elastic, resistant, and cartilage-like, quite different from cicatricial hardness or oedema. This condition of ulcer lasts about three to six weeks, when the edges of the chancre begin to empty themselves and to collapse. The granular particles which covered its base become eliminated or absorbed. At any rate the false membrane-like surface disappears, granulations form, and cicatrization commences from circumference to centre. The resulting cicatrix is round and slightly depressed, and is always the seat of induration, sometimes persistent. For a long time it is of a dark brown or bronze colour; finally all colour disappears, and an unnatural whiteness takes its place.

In all forms of the primary syphilitic affection the main characters are

papular development combined with *characteristic induration*; which induration has been long known to be the most essential part of the primary infection, as was known to the physicians of the fifteenth and sixteenth centuries, and specially insisted upon by John Hunter. When left to itself the indurated primary sore, if the induration is extensive, has a very protracted course. Months may elapse before it resolves and softens, even where specific treatment has been pursued. In large flat indurations upon the skin of the penis, the elevation may be seen gradually to decrease; and as the softening goes on, it becomes somewhat hollowed or umbilicated, and as it falls more and more to the level of the surrounding skin, the redness disappears, and is succeeded by a brownish pigmentation, which at the periphery is uniformly and pretty sharply defined; while in the centre, where slight ulceration has been, pale cicatricial lines traverse it in a reticulated form. The pigmentation disappears in the course of time, but may be often seen for years afterwards, notably towards the edges. The turning point in this softening is generally coincident with the outbreak of general constitutional symptoms. When the induration has entirely softened and receded, the primary syphilitic sore is at an end,—often without a vestige of it remaining; but more commonly it leaves a scar and a spot of pigmentation. So long as any hardening remains about the cicatrix, and even when none can be felt, the patient is in danger of a relapse in the same spot. The scar may become inflamed and hardened, and may even ulcerate without a new infection having taken place (J. HUTCHINSON). The subsequent enlargement of the lymphatic glands in the region of the primary infection is one of the most constant, the most characteristic, and most valuable of the symptoms of syphilitic infection; and the route by which the *virus* reaches the glands may sometimes be traced through a portion of its course by a thickening of the lymphatic vessel, which may be felt beneath the skin as a narrow and exceedingly hard cord. When the primary sore is situated upon the genitals, the intumescence of the lymphatic glands takes place very gradually, and is not usually perceptible until several days (five to six) after the primary sore has appeared. Usually only a single gland is at first affected. In cases where the virus was inoculated experimentally, the swelling of the glands was not observed before the eighth to eleventh day after the appearance of the primary papule (VON BARENSPRUNG, HEBRA). The swelling of the glands is seldom very great if they are not irritated. The inguinal glands may become as large as from the size of a filbert to a pigeon's egg or large almond. They are not usually tender on pressure, nor painful, nor do they cause any inconvenience; hence the name of *indolent bubos* given to them. It is not only a single gland which becomes swollen, but gradually those adjacent become swollen; so that after a time a bundle or chain of such swollen lymphatic glands make their appearance, varying in size, hard to the touch, slightly movable under the skin, which thus appears to be elevated above a smooth but somewhat nodulated tumor (BÄUMLER). The superficial lymphatic glands in both groins are often attacked simultaneously, although the primary sore is only on one side; and sometimes also the first gland to swell is on the side opposite the primary sore. *Induration of glands*, with *induration of the primary sore*, is absolutely pathognomonic of

sypilis. There is no inflammation, the enlargement is slow and insidious. They are freely movable upon each other and over the tissues. They may sometimes be slightly tender on pressure, and then the pain is peculiar, but not severe. They never suppurate, or, at any rate, actual suppuration is extremely rare, except when injured by violence, or if the patient be scrofulous, or unless a soft chancroid or suppurative urethritis co-exist. The enlarged ganglia may even point to the original site of the primary sore. Thus, the inguinal ganglia, enlarged and indurated, point to the genital organs, urethra, or hypogastric region; the group of glands near anterior and superior spine of the ileum point to the anal region; the submaxillary group to the lip, mouth, tongue; the axillary group to the elbow, hand, arm, as the original sites of the sore. Where the primary sore is on one of the fingers, the corresponding cubital glands swell; if the nipple has become the seat of the primary affection, certain glands swell on the side of the thorax (as they do in cancer), below the *pectoralis major*, or in the deeper glands of the axilla. These gland swellings may continue to increase for weeks; and the swellings are not merely confined to those glands nearest the primary sore, but generally it is believed that the whole lymphatic system of glands becomes more or less indurated and swollen, although this has only been demonstrated as far as the iliac glands. The lumbar glands are believed to follow next in the order of infection, and so the poison passes to the thoracic duct, and finally arrives in the general circulation. But there is no anatomical proof with regard to the entire and exact route. The body generally becomes primarily affected through the blood-vessels circulating in the primary sore, during the period of induration and absorption of the softening from the primary sore—the swelling of the glands being the earliest evidence of constitutional contamination next to induration of the primary sore.

One of the most interesting and important facts in the pathology of syphilis, is that the poison undergoes a process of multiple elaboration, a so-called second incubation or development in the system, before its full effects are completed; and the lesions it induces demonstrate some of the most interesting points in the pathology of the multiplication or reproduction of morbid poisons. It is this multiplication of the poison which ultimately destroys life, through a degeneration of the tissues and the establishment of a cachexia; or by the induction of grave lesions in important visceral parts, such as the brain, the lungs, the liver, or the kidney. The earliest effects of the syphilitic poison, after the first period of incubation has passed, become established upon the system during the occurrence of the "*hardening process*" or induration of the tissue which has been described, and which ultimately surrounds an infecting venereal sore—the local papule and the subsequent ulcer or primary syphilis sore. This hardening process is peculiar; and although not constant as to the local sore, it is constant as regards the glands or lymphatics which proceed from the vicinity of the part inoculated. It occurs in one or other of the three following conditions:—(1.) Hardening or induration of *sore and glands*; (2.) hardening and induration of the *cicatrix and glands*; (3.) hardening and induration of *lymphatic glands* only—the original local lesion never having been observed to become hard (SIGMUND). It may continue a long time.

Thus the period between the first appearance of the primary syphilitic affection and the eruption of general symptoms, has sometimes been designated as that of the *second incubation* (BÄUMLER). This is the period which is required by the syphilitic virus so to increase as to gradually infect the total mass of fluids and solids throughout the body. Thus it is that from the specific and characteristic local conditions just described, as from a focus, the system eventually becomes contaminated. The steps or sequence of phenomena associated with this contamination are not yet clearly understood; but as the contamination is expressed by very constant and specific characters, it is obvious that the original virus has become *intensified* in its action (as is also the case with the virus of hydrophobia), its pernicious influence more active and obvious, while its remote effects have become more fully and extensively developed. The lymphatic glands become deposits for the accumulation (if not for the elaboration and multiplication of virus), so that the blood gradually becomes more and more poisoned; but the diffusion of the virus through the body takes place very slowly by way of the lymphatics. On an average the duration of the *second period of incubation* is from *six to seven weeks*; and the whole time from the infection to the outbreak of general symptoms from nine to eleven weeks. In Bäumler's cases it amounted to thirty-five, forty-four, and fifty-four days. In the cases of experimental inoculation noted by Bäumler the shortest period is twelve days, the longest one hundred and thirty-nine days. The variation between two and twenty weeks is very great, and the cause of this variation is quite unknown. In hydrophobia it is not less so.

Beyond what has been described, no further derangement of general health may occur. But in many weakly subjects, especially females, a gradually increasing anæmia sets in, the patient begins to grow pale and to feel "out of sorts," indisposed in many ways, so that a condition like *chlorosis* becomes developed. The alteration in the constitution of the blood on which this state depends, is in close relation to the hyperplasia of the lymphatic glands, delivering a larger quantity of white corpuscles into the blood than usual.

In males and females at this period, with or without febrile symptoms, an *eruption* appears upon the skin, without itching, and in the form of red spots or papules. Simultaneously with this, or somewhat later, an inflammation (at first simply catarrhal) begins in the throat, shortly leading in the development of the disease to ulceration, or to the production of circumscribed flat growths on the mucous membrane. With the accession of these general symptoms, it is usual to say that the syphilis has become constitutional (BÄUMLER). The system, at any rate, as to blood and solids, has become affected, and general infection is complete, so that every organ and tissue may thereafter suffer in consequence. In numerous cases, over 20 per cent. (BÄUMLER), the commencement of general symptoms is very sharply defined by the sudden occurrence of the eruptive fever of syphilis—the relation of which is similar to that which obtains in the acute exanthemata where fever precedes the eruption. Even if a primary sore has not been observed, and febrile phenomena appear, such phenomena are constant in syphilis. Hence it is necessary to recognise the *fever of syphilis*. During the early period, the differential

diagnosis of the febrile phenomena lies between "*intermittent fever, rheumatic fever, typhoid fever, gastric derangement, and the cephalalgia of Bright's disease.*" The severity of the general symptoms, and especially of the fever, is sometimes so great that they are apt to divert attention from the investigation of the original and primary syphilitic lesion, even if it were known to exist. This syphilitic fever requires special study. Its relation to the severity of the future lesions is quite undetermined as yet. It is ushered in by symptoms like those which precede eruptive fevers. It usually precedes, by eight or ten days, some of the early eruptions; but the fever continues *after* the eruption appears, and is a true fever, attended by general derangement of the functions, nausea, flying pains, frontal headache, and depression of spirits. Nevertheless it will not come into the same group with well defined fevers. The ultimate (so-called tertiary) phenomena or sequelæ of syphilis are specific; they come from syphilis and from nothing else. Hence they cannot be compared with the sequelæ of other fevers, about which there is nothing specific. The bodily temperature rises to 100° or 102° Fahr. at night, falling in the morning to 98° or 99° Fahr. This alternation may continue for days or even weeks, so long as fresh cutaneous eruptions or other lesions continue (SMITH). Fever is thus "a significant part" of syphilis. In point of fact, the pyrexia in the exanthem stage of syphilis sometimes runs high; and is probably always present more or less. Dr. Buzzard has known a patient almost to die during it. The disease is extremely variable as to its severity in different individuals. A considerable number of those who pass through the syphilitic fever after chancre contagion do so without rash, without sore throat, without indeed any visible symptoms (JONATHAN HUTCHINSON, l.c.) Periodicity of these febrile exacerbations is a frequent phenomenon—a chill, followed by a hot stage and sweating, occurring with great regularity at a certain hour of the day, generally towards evening (ZAMBACO, YVAREN, BOYER). Syphilitic fever may thus be mistaken for "intermittent fever." The ague-like fits are repeated for some time—a week; then the fever becomes continuous, and so persists, in spite of quinine. The influence of mercury alone subdues the febrile exacerbations. Epistaxis and palpitations are also not unfrequent concomitants. It bears a close resemblance to rheumatic fever at first, but later on is more like hectic. The syphilitic fever generally commences between fifty and sixty-five days from the time of infection—rarely before the fortieth day, and may be deferred till the ninetieth day.

Stages of Syphilis.—There is an order in the evolution of syphilitic phenomena, so that "when allowed to develop without the interference of specifics, its stages are fairly regular: but it is hopeless to get correct ideas of the natural course of syphilis if we investigate only cases in which mercury has been used." The *primary symptoms* are those developed at the site of contagion. The so-called *secondary symptoms* are those lesions which affect parts not directly inoculated, and which are generally superficial as to cutaneous and mucous surfaces. The so-called *tertiary affections* are later, and are considered to be those which are separated by an interval of apparent health from the usual secondary symptoms, and the lesions generally attack the visceral organs and deeper structures. But, in point of time, such a classification rarely exists in fact, as it is a

very common occurrence for a patient to have the phenomena of all the three periods present at one and the same time; and at all stages of the disease the histological elements of the lesions are the same. A division, based on the *anatomical* characters of the lesions, seems to be more satisfactory than any arbitrary arrangement into stages of a supposed *primary, secondary, or tertiary order* (HALDANE). The arrangement of the phenomena into "primary" and "secondary" was based upon Hunter's division of the "tissues affected in syphilis" into "*parts first in order*," and "*parts second in order*" of being affected. The secondary he reckoned to begin from the third week to the sixth month; the tertiary, not before the sixth month, and in many not till after several years. This was an artificial and no real distinction; and the sooner these distinctions are forgotten the better.

The constitution of the person also materially influences the phenomena which supervene during syphilis—*e.g., gout, rheumatism, tuberculosis, and cancer* modify the syphilitic lesions and degenerations; while constitutional syphilis in its turn modifies the character of ordinary diseases. For example, persons with a tendency to rheumatism are apt to have the same tissues involved in syphilitic lesions as if they suffered from rheumatic inflammation. Hence syphilis is often set down as a cause of rheumatism. The serous, fibro-serous, white connective tissues are the sites of the lesion in the forms of *periostitis, iritis, corneitis*, and affections of the true skin. In scrofulous patients those tissues are apt to be involved in the syphilitic lesions which are most prone to ulcerate, and to have tubercles grow in them. Hence syphilis is often set down as a cause of phthisis. The mucous membranes are most prone to suffer in such cases. Hence syphilitic growths develop themselves in the lungs, the glands, and brain, pharynx, larynx, tonsils, tongue, and testicles. In the gouty or vascular subjects the arterial or vascular structures and joints are apt to suffer most from the syphilitic virus, and the lesions ultimately assume the form of degenerations. Hence syphilis may be set down as a cause of disease in the great blood-vessels leading to thoracic and abdominal aneurisms at an early period of life; and of the smaller blood-vessels, leading to lardaceous disease of the liver, kidney, spleen, and intestines.

Bäumler suggests such a practical division of the symptoms of syphilis as would separate them into groups according to the order in which they gradually develop, namely:—(1.) *The primary stage*, commencing with the infection, and comprising the gradual development of the local symptoms at the point of entrance of the virus, and the indolent gland swellings in the vicinity. It therefore includes the period of incubation, and terminates at the breaking out of general symptoms. During this stage there is no absolute immunity from new infection. (2.) *The secondary stage*, when blood-poisoning is at its height, begins six or eight weeks from the first appearance of the primary affection, accompanied frequently by an eruptive fever. It is characterised by *superficial* eruptions of the skin and mucous membrane, distributed symmetrically over both halves of the body; falling off of the hair and disease of the nails; anæmia; universal lymphadenitis; simple irritative processes in the periosteum and interstitial areolar tissue of internal organs—all disposed to heal spontaneously without loss of tissue, but tending to permanent derangements of function

through adhesive inflammation of connective tissue, followed by its contraction—syphilitic cirrhosis. During this stage there is immunity from new infection with the virus of syphilis, and the power of transmitting the disease to offspring. This stage may continue from several months to a year, merging into an intermediate stage of indefinite duration, during which the disease remains latent; or from time to time various eruptions appear upon the skin and mucous membranes, more limited, less symmetrical, and not so acute as the first general manifestations of the secondary period. (3.) *The tertiary stage* is characterised by local affections, for the most part symmetrical, often set up by external causes, and consisting in cell-growths having a tendency either to disintegrate or to become encysted with caseous metamorphosis and new formations of connective tissue; gummata in various organs; ulceration; necrosis and caries of skin and bone. The limits of this stage as to time are quite uncertain; and its phenomena are sometimes concurrent with those of the secondary period. As a rule, several years, often twenty or more, may intervene between the time of contagion and the outbreak of tertiary symptoms. At this period the blood-poisoning of syphilis is now either insignificant or entirely absent, so that the offspring generated in this stage usually escape infection: the local manifestations are incapable of conveying the syphilitic poison: and, at the same time, *immunity* from new infection of syphilis is *diminished*. (4.) *Stage of confirmed marasmus induced by syphilis*, characterised by such irremediable changes as lardaceous disease of liver and intestines especially; destructive caseous pneumonias; dysenteric and other ulcerative processes.

The following table exhibits a scheme of the order of evolution or periods of appearance of the phenomena after inoculation or contagion from an infecting sore, estimated from the first appearance of the papule or sore (based on the experience of BASSERAU, SIGMUND, FOURNIER). After examination of the system, specific lesions peculiar to syphilis begin to appear somewhat in the following order of events:—

LESIONS OR SYMPTOMS.	Date of usual Development.	Date of earliest Development.	Date of latest Development.
Roseola,	45th day.	25th day.	12th month.
Papular Eruption,	65th "	28th "	12th "
Mucous Patches,	70th "	30th "	18th "
Secondary Affections of Fauces,	70th "	50th "	18th "
Vesicular Eruptions,	90th "	55th "	6th "
Pustular Eruptions,	80th "	45th "	4 years.
Rupia,	2nd year.	7th month.	4th year.
Iritis,	6th month.	60th day.	13th month.
Sarcocele,	12th "	6th "	34th "
Periostitis,	6th "	4th "	2 years.
Tubercular Eruption,	3 to 5 years.	3 years.	20 "
Serpiginous Eruption,	3 to 5 "	3 "	20 "
Gummy Tumors,	4 to 6 "	4 "	15 "
Onychia,	4 to 6 "	3 "	22 "
Exostosis,	4 to 6 "	2 "	20 "
Ostitis—Changes in Bone and } Cartilages, }	3 to 4 "	2 "	41 "
Perforation of Palate,	3 to 4 "	2 "	20 "

It is therefore natural for syphilis to show itself, not in a continuous, but rather in an interrupted, succession of phenomena; and the reappearance of many syphilitic lesions (such as the lesion of skin and mucous membranes)—*erythema*, *alopecia*—the shedding of the hair differing in the early and late periods. In the early period, one of the earliest general symptoms is shedding of hair, which may grow again, because the hair bulbs are not seriously damaged; in the late periods, the bulbs are damaged, and the scalp becomes permanently bald. So also two forms of *iritis* and *ecthyma* are distinguishable.

These are not to be regarded as relapses of the disease, but as recrudescence phenomena in the natural course of its development.

Affections of the skin are the most apparent among the early manifestations of syphilis; and are now generally comprehended and expressed by the term "*syphilide*" (ALIBERT, BIETT). The condition of the skin still further demonstrates the contamination of the system. In the more insidious form of contamination its colour generally is altered. It becomes pale, white, fawn, yellow, or brown; and is wrinkled, dry, harsh, rough, and hard,—no longer soft, elastic, and of healthy lustre,—particularly in the tertiary stage, or where there is pronounced marasmus a sallow, harsh, and flabby skin; and there are besides a great number of special forms of skin affections which are quite peculiar to syphilis, with certain characters which render them at once recognisable as due to syphilis. The most important of these characteristics are—(1.) *Colour*, which has been generally described as a "*coppery red hue*," its peculiarity being a combination of *yellow* and *brown*, with the more or less dark red of efflorescence. But this colour cannot be regarded as specific or as an exclusive mark of syphilitic eruption; and it does not pertain to all forms of the syphilitic eruptions in an equal degree. It is mostly pronounced in persons of dark complexion; least so in the fair and pale-skinned and anæmic. (2.) In the *polymorphous* character of the eruptions, when *macules*, *papules*, *vesicles*, and *pustules*, may be seen side by side—no doubt explicable from the essentially chronic course of the "*syphilides*." (3.) *The annular form of efflorescence*—i.e., the grouping together of several points to form perfect circles or sequents, similar to *herpes circinatus*. This character belongs to the *papular*, *tubercular* and *vesicular* eruptions, rarely to the *macular*; and a considerable length of time is necessary to its production. (4.) *Absence of itching* is so characteristic that many patients are not aware that they have any eruption. The different forms of the eruptions on the skin and mucous membranes are,—(1.) Exanthematous; (2.) Vesicular; (3.) Pustular; (4.) Papular; (5.) Squamous; (6.) Tubercular. They are often associated together; and they are peculiar in their symmetry of distribution and in the curvilinear character of their grouping. They leave behind them stains of colour, pale cicatrices, or persistent ulcerations of the true skin. Bäumlér has arranged these different forms into groups, each of which include several varieties:—

GROUP I. *Circumscribed Hyperæmias, with but slight infiltration*, including only one variety, the macular syphilide, commonly named, "*Roseola syphilitica*,"—the earliest and most common form of the syphilitic skin affections, making its appearance not unfrequently after an eruptive fever of

several days' duration. It does not then become an abundant eruption at once, but so gradually that fourteen days or more elapse before the eruption is at its height. The eruption (*exanthem*) consists of perfectly even rose or dark-coloured spots, of a size varying usually from that of a lentil to that of a pea, with a roundish or irregular shape. At first it disappears entirely under pressure; but after several days (if the complexion is dark) a yellow discoloration remains behind, thus giving a characteristic coppery hue. The abundance of maculæ varies much—from a few spots upon the sides of the chest and groin, to a crop as abundant over the whole body as in a severe case of measles. If scanty the eruption generally disappears rapidly—*i.e.*, in the course of from one to two weeks under mercurial treatment; but if left to itself it may remain for several weeks. Vesicles and pustules develop very rarely during the existence of roseola. The *roseola syphilitica* may be the sole eruption during the whole course of the disease. It is rare that no eruption occurs at all; but often it is not noticed, beyond trivial affections of the throat and tonsils. It may also occur again and again in the course of the same syphilitic attack.

GROUP II. *Well marked Circumscribed Infiltrations of the Papillary Structure of the True Skin*,—(1.) In the form of papulæ (*papular syphilide*); (2.) in large patches (squamous syphilide); but on mucous surfaces or at moist parts of true skin as moist papules—*i. e.*, *condylomata lata*. (1.) *In the papular form* the papules vary in size from a barley-corn to a split pea, varying also in colour according to their age, from red to brown, hard to the touch, and smooth or slightly desquamating on the surface. The separate points of efflorescence are about the size of a lentil; and the separate papules are sharply circumscribed. If the loose epidermis is rubbed off a thin scab soon covers it, by the oozing from it of a little serum. Where surfaces of skin lie in contact, especially where there is friction, or the irritation of secretions, the papules develop exuberantly, running together in patches of large size, with moisture on the surface. The papillæ become hypertrophied, with maceration of the epidermis and molecular disintegration, so that it is covered with a greyish slimy secretion, which is generally a dirty fetid fluid, extremely irritating to the neighbouring parts, and apt also to develop vegetations or warts. They are soft and rarely painful or itchy. *Syphilitic condylomata* rarely fail to appear as a frequent symptom of general infection of the system. Their appearance is generally the first symptom which succeeds the primary infection and enlargement of the lymphatics. They consist of broad elevations of tissue above the level of the skin or mucous membranes; covered in the skin by a thin coating of epidermis, usually in a state of superficial ulceration, and bathed in a slimy, bad-smelling secretion. The most frequent seat of soft condylomata is between the nates—in women between the *labia*, and in men upon the scrotum and outer surface of the penis. Sometimes they spread over the inner surface of the thighs. They may appear also at the angles of the mouth; more rarely at the commissures of the eyelids, and between the toes. They often become fissured, especially when they have coalesced to form extensive growths; and in this way painful and often obstinate ulcers, fissures, and cracks form, attended by severe pain, and which heal slowly. Fissures (*rhagades*) occupy by preference the cutaneous folds, especially in the neighbourhood

of the anus. The most common seats of election are—(1.) In men, round the anus and within the mouth. (2.) In women, upon the vulva (RICORD, *Iconographie*, Pl. XVII., also XX). The seat of *mucous patches* is mostly about the genito-anal region, and, next to the vulva or anus, they are met with most frequently on the inner and upper part of the thighs, on the tonsils, in the mouth, upon the lips, in the inter-digital spaces, about the nipples (women especially), in the groins and about the ears, on the scrotum, inguinal folds, and between the toes. The colour is peculiar in different places. Within the mouth it is greyish-white, like the result of the application of nitrate of silver—hence named “opaline patches”—described on the internal surface of *lip* or *cheek*, side and dorsum of *tongue*, the *gums*, *tonsils*, and *soft palate*. The laryngoscope will show them on *epiglottis*, *larynx*, and *pharynx*. In this state it has received the various names of *moist* or *mucous patch*, *mucous tubercle*, *fig-wart*, or *flat condyloma*—the secretion from which is *infectious in the highest degree*. They are conditions altogether peculiar to syphilis. This eruption is often attended by violent pains in the bones, and by *iritis*; and when it continues for any length of time it is apt to be followed by *alopecia* and diseases of the nails. It continues much longer than the *macular syphilide*—often for months—and relapses may occur, especially on the palms of the hands and soles of the feet. (2.) *Infiltration of the papillæ in large patches (squamous syphilide)* is generally associated with considerable infiltrations in the cutis, and when desquamation is considerable resembles *psoriasis*. They develop especially on the forehead, back of the neck, hairy scalp, the *mons veneris*, the *labia majora*, and scrotum. It has been observed by Dr. Bäumler as early as the fourth month from infection. This eruption may also become condylomatous.

GROUP III. *Infiltration into the Walls of the Follicles*, when a hair is seen to penetrate each of the several points of efflorescence; or the excretory duct of the follicle may be observed as a minute dark spot in its centre. (1.) In the simplest form (*lichen syphiliticus*) there is scarcely any infiltration; but hard granules, situated beneath the epidermis, which can be excavated with the finger nail, leave little cicatricial depressions. Such papules occur singly, but ranged in groups of a dozen or more. If the process becomes acute an exudation takes place into the follicle, and groups of vesicles form, which become pustules. This acute form is characterised by the grouping, and by the constitution and course of development of the papules. (2.) When acute suppuration develops in the follicle (*acne syphilitica*) in connection with infiltration, acuminate pustules on a red coppery base are produced. The usual location of this eruption is the face, the back (especially between the shoulders), the hairy scalp (particularly at the edge of the hair), the buttocks, and the extensor surfaces of the thighs and legs. If numerous acne pustules develop all at once the general symptoms may be severe. (3.) A form sometimes occurs in which the skin between the follicles also becomes infiltrated at once with the follicular infiltration. Desiccation quickly occurs, and greenish-yellow granular crusts cover the whole surface of the infiltration; and beneath there is loss of substance over an uneven surface (*Impetigo syphilitica*).

GROUP IV. *Infiltration with Subepithelial Suppuration and Superficial Ulcer-*

ation.—In this group the pus is situated under the epidermis. (1.) The pustules (*pustular syphilide*) are small in size, develop rapidly, and may resemble those of *varicella herpes* or *acne*. (2.) The ecthyma pustule (*ecthyma syphilitica*) is the typical form of the group. It occurs as an elevation of the epidermis by a cloudy fluid, which soon becomes purulent, not unfrequently tinged with blood over a reddened base. It appears in isolated spots, with wide interspaces separating the pustules. Its common site is on the legs below the knees. It develops very quickly, following hyperæmia of spots which has existed for a long time before. The scars left are generally round, and remain red for a considerable time, and may ultimately become pigmented or remain perfectly white; or depressed and surrounded by a border of pigment. It is a manifestation of the later periods of syphilis, and denotes a poor constitution—prone to suppurations (see “Pyogenic Fever,” page 796). Affections of the mucous membrane at this time are also prone to suppuration and ulceration—*e. g.* aphthous sores in the mouth, deep ulcers in the tonsils, or destructive ulceration of the soft palate. Suppurative processes in the bones also accompany this form of eruption, with febrile symptoms of a marked remittent type; and with gummata in the subcutaneous cellular tissue. (3.) *Rupia* consists of large, dirty, brownish-green stratified crusts of a conical shape, resting upon a perfectly flat ulcerating surface. The crusts are formed out of purulent bullæ, the contents of which dry into concentric rings, each enlarging in circumference. It may attain the size of a half-crown piece, but is rarely larger. Cicatrisation of the ulcerating surface is very irregular and slow. It may continue to extend for years, converting extensive tracts of skin into cicatricial tissue. The cicatrices are permanent, and become gradually very white, usually quite smooth. *Rupia* sometimes breaks out within the first six months after infection, attended with violent remittent fever; but is oftener a manifestation of the later periods without fever, and associated with marked syphilitic marasmus, gummy tumors, and necrosis in the mucous membranes and bones. It always lasts for many months.

GROUP V. *Deep Infiltrations and Disintegration*.—This is the stage and period of *gummous* formations, characterised by the *tubercular syphilide*, in which gummy tumors may develop in the skin in the form of roundish tubercles, singly or in groups, having a circular or crescentic arrangement. It begins to grow in the deep layers of the cutis, and redness only is present when the tubercle approaches the surface and begins to cause elevation. They tend to reabsorption without ulceration, and are to be found especially on the shoulders, on the face, and on the extremities. There are other forms which tend to ulceration of an irregular progress (*lupus syphiliticus*). It belongs to the tertiary group of phenomena; and similar gummous formations may be at the same time going on in the bones, the testicles, and the various internal viscera, previously described under morbid anatomy, page 906, *ante*. Its cause is very protracted and prone to relapse. The patients are generally cachectic, with co-existent lardaceous disease in the abdominal viscera and intestines—a number of years having elapsed since the acute stage of the disease. Dr. Bäumler has seen it as early as the eighteenth month from the contraction of the disease.

The symptoms of syphilis from the condition of the mucous membranes are analogous to those of the skin; and, next to the skin, they are the most common. They consist of *erythema* of the mouth and pharynx; patches on the palate, velum, pharynx, inner surface of lips and cheeks, with sharp pains and dryness of throat. These symptoms usher in the usual and mildest form of a *syphilitic catarrh*. The early affections of the fauces are often not more than a peculiar colour of the mucous membrane, persistent, however, like the staining of the skin (GAIRDNER), and eventually leading to disorganisation in the uncared for development of the disease. In women the process may cease with a slight follicular swelling of the mucous membrane of the fauces, tonsils, and soft palate (SIGMUND). If the process does not cease, then superficial erosions or deep ulcers of the soft palate supervene. Or still more diffused forms of ulceration may set in, involving great destruction of parts, and spreading in all directions—encroaching on the nasal fossæ and pharynx, eating away the epiglottis, extending down the air-passages, and even causing necrosis and exfoliation of the cartilages of the larynx. Gastro-intestinal derangement leads us to believe that the intestinal tract is also affected by the syphilitic poison at the same time as the throat; and *bronchitis* is a frequent result of the disease. Ulcerative affections have their seat in the nostrils, tongue, anterior pillars of *velum palati*, the tonsils, and internal surface of pharynx. The larynx is subsequently affected.

The symptoms of syphilis from the condition of the HAIR and NAILS are chiefly—(1.) Loss of hair—*alopecia*. The hair loses its lustre, becomes dry, often discoloured, and upon combing easily falls out, with the result of thinning of the hair in certain spots or patches. Total baldness is very rare, and the skin of the scalp is normal—not smooth and shiny, as in ordinary baldness. It is one of the early manifestations of general syphilis, sometimes just preceding the outbreak of the eruptive fever. This loss of hair may continue for months; but in the young its luxuriance is again restored. In the later periods this restoration does not happen again. (2.) Affections of the Nails occur in two forms, namely—(a.) As *onychchia sicca*, and (b.) as a dark-red eruption on the nail, due to congestion of the vascular layer beneath, with numbness, and tingling beneath the surface. The nail atrophies, desquamation from its surface commences, and continues with splitting up of the substance, and pitting. Lastly, it crumbles down from the edges and free margin, point, or tip. It is irregular, and thickened from the under part (psoriasis of the nail), analogous to the “Seedy toe” of horses. (Compare RICHARDSON’S very interesting paper in *Clinical Essays*, Vol. I.)

The symptoms of visceral syphilis have been considered in connection with morbid anatomy (page 915, *ante*).

Diagnosis.—In the previous pages the hard primary infecting sore of syphilis has been the sole subject of consideration as the only one capable of communicating syphilis. At one time another sore in addition to the one already considered was believed capable of communicating syphilis—namely, the *soft sore* or *soft chancre*. Hence also the unity and duality theories of syphilis. Leon Bassereau was the first to show the respective sources whence these two sores spring—the hard and the soft. Clere

confirmed the facts of Bassereau; and Ricord (abandoning the idea of the unity or sameness of the two kinds of sores) adopted the principle that each variety of sore propagates only its own kind—the soft sore taking effect on all alike, both healthy and syphilitic; but the hard syphilitic sore only on the healthy—*i. e.*, those who had not before suffered from syphilis. It is therefore important in diagnosis to distinguish the soft sore or chancre which develops itself in a very short time, generally at the end of two or three days after connection or exposure to its virus. In the first twenty-four hours the point of inoculation becomes red and surrounded by a small circle of inflammation, tumefaction supervenes, a vesicular pustule appears, and finally a pustule-like ecthyma. To this succeeds an ulcer, more or less deep, round in shape, with a tendency to have its edges cleanly cut, and sometimes everted. A magnifying glass shows small indentations on the edges, each surrounded by a red inflamed circle. The floor of the ulcer is uneven, covered with a yellow grey matter, composed mainly of a dirty thick pus, virulent and contagious in the highest degree. The base is as supple as the neighbouring tissue. This soft chancre is always inoculable on the same individual, producing the same kind of local sore as that from which the poison was taken—to an almost unlimited extent; and if at first single, it may multiply to any extent. The glands are, as a rule, affected; and usually only a single gland is painful from the first, and it tends to suppuration. The pus of the bubo has the same properties as that of the soft sore; *but no constitutional syphilis* follows. The contagious principle of the soft sore is thus non-identical with that of the hard infecting sore of syphilis. It may be generated *de novo* under the co-operation of certain influences, and quite independently of syphilis (BÄUMLER). These characters are to be contrasted with the peculiar clinical characteristics of the *syphilitic sore* already described, page 925, *ante*.

The following tabular summary may be given :—

TRUE SYPHILIS.

(From hard infecting sore.)

1. Incubation, of a mean duration of twenty-four days.
2. Lesion mostly single, not reinoculable on the subject of it.
3. Consisting in a papule of greater or less size, which erodes or ulcerates, the ulcer being always superficial, without detachment of the edges and without suppuration, unless it be in the period of cicatrisation.
4. Almost always accompanied by firm, indolent, multiple, non-suppurating enlargements of glands.
5. Influenced by iodide of mercury and iodide of potassium.

PSEUDO SYPHILIS.

(From soft non-infecting sore.)

1. Incubation, none (commences within twelve hours).
2. Lesion generally multiple, indefinitely reinoculable on the subject of it.
3. Showing itself as a vesico-pustule, which terminates in a deep ulcer, with detached perpendicular edges, and furnishing an abundant purulent secretion.
4. Accompanied in some cases only by enlargements of the glands, which furnish inoculable pus.
5. Aggravated by mercury.

It is also of importance to distinguish the specific induration of the primary infecting sore from hardness due to inflammation or irritation.

The following are the characteristics of specific induration, as contrasted with inflammatory engorgement:—

<i>Specific Induration.</i>	<i>Inflammatory Engorgement.</i>
Boundary clearly defined, terminates abruptly.	Boundary unlimited, shades gradually into normal suppleness of parts.
Freely movable.	Adherent to parts below.
Firm, hard, resistant, like a mass of cartilage or a split pea.	Soft, dough-like, and tough.
Continues persistent.	Subsides in a few days.

Both conditions of hardening may co-exist. But when the inflammation subsides, the specific induration is laid bare, which was previously buried or masked by the inflammatory products. The induration is very insidious in its appearance; hence cases of *syphilis*, without chancre or sore, have been recorded simply because the original lesion has never been noticed. The testimony of venereal patients may be influenced by a want of honesty; but quite as often they are influenced by ignorance, want of experience, and absence of medical knowledge as to the nature of their disease. Many vulgar errors prevail on the subject. The surgeon and physician must each judge for himself. Some believe patients implicitly; some, as a rule, always disbelieve them; but the distinction requires, in most cases, to be drawn between honesty and ignorance.

The great diversity of symptoms which syphilis presents at different stages of the disease often renders diagnosis difficult in advanced cases. The appearance of certain typical symptoms may lessen the difficulties, such as—(1.) Well-marked primary indurations on the genitals; (2.) some forms of papular eruptions, especially *condylomata lata*; (3.) symmetrical ulcerations of the tonsils; (4.) gummy iritis; (5.) papules on the palms of the hands; (6.) serpiginous ulcerations on the skin; (7.) tertiary ulcerations in the throat. Particular inquiries should be made in regard to those symptoms which usually occur at an early stage of syphilis, especially,—cutaneous eruptions, falling off of the hair, sore throat, pains of the bones, and iritis. As for the primary sore itself, it may be spoken of by men, but scarcely ever by women. The diagnosis of primary syphilis can then only be made when the initial lesion has become noticeable, and has been the seat of an induration (BÄUMLER). The diagnosis and opinions must sometimes be delayed until the result of the treatment employed or of reinoculation may be seen. When the papule opens and becomes a sore, the fluid discharged from its open surface has been shown by Hubbenet, Lee, and Rollet to furnish a diagnostic test of the kind of disease, and of the sore from which it proceeds. Sigmund does not go so far as this. He does not consider the sores or chancres so different in form or characters as to be at once distinguishable the one from the other. *He waits to see the virus produce PART of its effect upon the system beyond the site of inoculation before he decides as to diagnosis.* He waits to see the lymphatics indurate. He believes that then, and not till then, the distinction can be absolutely drawn between a sore which will infect the system and one which will not. He believes—(1.) That if induration of the lymphatics does *not* take place within six or eight weeks, and (2.) that if repeated

successful auto-inoculations can be made on the bearer of the chancre during this period, then it is probable that the sore will not infect the system. If, on the contrary, the lymphatics indurate, and auto-inoculations cannot be then effected, the sore is assuredly an "infecting" one. The addition to our means of diagnosis from the nature of the discharge—pus from the one, not from the other—is one of great value when it can be made, because the diagnosis as to the probability of subsequent infection may in some cases be made earlier. The distinction, however, is untrustworthy, for the following reasons:—*First*, the syphilitic virus may be present in a patient suffering with local venereal ulcers also, and the pus of those ulcers might be inoculable, notwithstanding the presence of general syphilis. *Secondly*, the thin discharge of the ulcerated papule of syphilis is not inoculable on its bearer; nevertheless, if the papule is made to suppurate by any kind of irritation, the pus from it sometimes becomes freely inoculable on the patient. The test is now therefore considered useless as a guide in diagnosis (BERKELEY HILL).

Another kind of sore frequently seen about the *prepuce* or *glans penis* of men, or the *vulva* of women, is mentioned here to guard against its being confounded with venereal sores—I mean the sore which forms after the eruption of *Herpes preputialis*. The eruption of *Herpes preputialis* commences by itching, which is felt at the base of the *glans*, at the internal surface of the *prepuce*, or at the junction of the *prepuce* and the *glans*. This itching is often so slight as not to attract notice, and it is not of the same kind, nor does it occupy the same site, as the itching of gonorrhoea. The surface of the *glans* or of the *prepuce* may be red; and although nothing may be at first visible with the naked eye, yet, by means of a hand lens, small circular elevations of cuticle are to be seen, raised by limpid serum. Several groups of these small vesicles generally occur, separated from each other by the space of a few lines. These eventually burst, and in their place small circular ulcerations, perfectly distinct, are to be seen, with a red bottom, and measuring scarcely a quarter of a line in diameter. The site of these herpetic ulcers is highly sensitive, and secretes pus and fluid, usually of an offensive and peculiar odour. When the groups of vesicles are situated on the *cutaneous* surface of the prepuce, they are but slightly inflamed, compared with those situated on its internal or mucous surface. Frequently the fluid contained in the vesicles on the cutaneous surface is reabsorbed, when slight desquamation ensues over the lesion. If the fluid is not absorbed, it becomes opaque after a few days, and small scaly incrustations take the place of the group of vesicles. The disease may thus terminate in about *seven* or *eight* days. When the groups of vesicles form on the internal aspect of the prepuce, they increase in size rapidly, and the inflammation is much more active. The walls of the vesicles are so extremely thin and transparent that the red colour of the inflamed tissue may be seen through them. But the fluid soon becomes opaque and sero-purulent, small moist crusts or scabs form, which, being detached naturally or accidentally, expose excoriated spots; and it is important to distinguish these from soft venereal chancres. Venereal sores never commence as vesicles.

The longer the time since primary infection the more difficult is the diagnosis. But certain symptoms may arouse suspicion and lead to a

more searching examination. These are, especially—(1.) Paralysis of cerebral nerves; (2.) persistent neuralgias; (3.) headache and pain in the bones (more intense at night)—the bones covered by skin only are those which should be carefully examined; (4.) loss of hair; (5.) abortion (often repeated); (6.) remittent febrile attacks (rheumatoid especially).

The diagnosis of syphilitic eruptions may in general be arrived at,—(1.) *From the history of the case*: A chancre commencing at least three weeks after exposure to contagion, becoming indurated, and followed within six or eight weeks by induration of the lymphatic glands. (2.) *From the symptoms accompanying the eruption*: A dusky tint of the skin, rheumatic pains in the head and joints, *alopecia*, ulceration of the throat, *iritis*, nodes, *gummata*, disease of the testicle. (3.) *From the eruption appearing in several forms at one time on the body*—e. g., Condylomatous, roseolous, lichenous. (4.) *From the general coppery tint of the eruption*. Strumous inflammations have a dusky red or vinous tint; simple inflammations in a healthy person have a bright red tint; but syphilitic eruptions in the chronic stage have a characteristic coppery colour. (5.) As a rule, there is an *absence of itching* in syphilitic eruptions. (6.) *Syphilitic eruptions tend to assume a more or less circular form*. (7.) When the eruptions of syphilis ulcerate, *the ulcers are generally round, with perpendicular edges and unhealthy bases*. More than one of these characters must guide the diagnosis, and not one only. Ulceration is not a usual characteristic. The diagnosis from enteric fever rose spots may be easily made. The diagnosis of a syphilitic cirrhotic liver, especially in advanced life and in the marasmus of syphilis, is often difficult on account of the projecting swellings upon the surface of the liver having a marked resemblance to cancerous masses. As a rule, however, the syphilitic liver is smaller than normal, while a cancerous liver is apt to be enlarged. The symptoms, especially the marasmus, increase rapidly if the disease is cancerous, but if syphilitic they may remain stationary for a long time. The syphilitic affections of the bones are to be thus distinguished, by—(1.) Their points of predilection; (2.) the nocturnal pains; (3.) the slight tendency to suppuration; (4.) their disposition to hyperostosis; (5.) in necrotic processes the sequestrum is very porous, and the reaction is slight in the surrounding parts.

Summary of hints for the Investigation, Diagnosis, and Description of Syphilitic Ulcers.—(1.) Ascertain as near as possible the date of contagion, keeping in view the media or vehicles of contagion, in addition to virus from a true primary chancre—namely, from ulcers in acute secondary syphilis; from the blood of patients suffering from acute secondary syphilis; from sexual intercourse, followed by conception, with a man who is at the time suffering from syphilis in some of its active secondary forms; from mixed chancres carrying the virus; from sloughing sores carrying the virus. (2.) Examine the patient, keeping in view,—(a.) That the soft, “non-infecting” sore commences almost immediately (*i.e.*, twenty-four hours to within three days after connection). It commences as a red spot, or a point, passing very soon into a pustule and a soft suppurating sore. (b.) That the “infecting” sore does not commence before the end of the second or beginning of the third week (eighteen to twenty-four days); and if the disease has been contracted from a secondary ulceration, not

before the expiration of the third or fourth week (RINECKER). A specific sore results in the formation of a papule, abrasion, fissure, or crack—the formation of pus, or an ulcer discharging pus, being an accidental occurrence. (3.) Examine microscopically the discharge from venereal sores, keeping in view—(a.) That a soft “non-infecting” sore discharges pus cells; (b.) that the fluid discharged from an “infecting” sore is not pus, but a molecular *débris*. (4.) The irritation of an “infecting” sore may cause it to discharge pus along with the “infecting fluid.” Hence “mixed chancres” capable of infecting the system. (5.) The soft, purulent, non-infecting sore may be transplanted at will, and at any time, on the patient’s body. The true “infecting” sore cannot be multiplied after glandular enlargement and general infection becomes developed. It remains a solitary sore. (6.) Look every day for cutaneous eruptions during the existence of a primary sore. (7.) Examine the lymphatic glands, not only in the vicinity of the chancre, but also those in the axilla, and the neck up to the occiput. Note as to the slowness or rapidity of the enlargement—hardness or softness, tendency to supuration, and whether painful or not. (8.) From the account of syphilis and venereal sores which has been given in the preceding pages, it must appear clear that definite nomenclature must be adhered to in describing all venereal and syphilitic sores.

Prognosis.—The course of syphilis must always be regarded as more or less dangerous and treacherous. Except among children who inherit the disease, it is seldom directly a cause of death; but indirectly it often leads to death, and always to more or less ill health. No statistics can give any adequate idea of the number of men lost to the public service from *syphilis*. Venereal diseases cause the greatest number of admissions to Army Hospitals. The loss of strength from them alone in the British service, in 1860, was computed by Dr. Balfour as equal to the loss of more than eight days annually of every soldier in the service. Dr. Balfour relates, in the *Medical, Sanitary, and Statistical Report of the Army Medical Department* for 1860, that “more than one-third of all the admissions into hospital have been on account of venereal diseases (369 per 1,000); and the average number constantly in hospital is equal to 23·73 (reduced to 14·87 in 1869) per 1,000 of strength (2,315 men), each remaining in hospital on an average 23½ days. Thus the inefficiency was then constantly equal to about 2½ regiments.” Dr. Balfour also observed the individual history of 1,126 men of the Grenadier Guards for three years and five months; 536 of these men gave rise to 1,250 admissions; 212 were admitted *once*; 146 *twice*; 70 *three times*; 55 *four times*; 24 *five times*; 19 *six times*; 6 *seven times*; 2 *eight times*; 1 *ten times*; and 1 *fourteen times*. These figures have great practical significance. They do not tell us how many of these men became constitutionally contaminated by syphilis, or finally suffered from syphilitic lesions of internal organs.

Inherited syphilis kills an unknown number of unborn children. It is a common cause of abortion. Numerous children also die at a very early age who inherit syphilis. In London, alone, during twelve years (1854-65) there were 3,370 deaths from syphilis among children, and 2,587 of these were under a year old (*Brit. Med. Journal*, 1868, Vol. II., p. 116). Also, deaths due to syphilis are often recorded in death returns under the

terms of "atrophy" and "marasmus;" and no just idea can be obtained of the mortality due to syphilis from any existing form of statistics.*

A history of syphilis in soldiers is too often the starting-point of a fatal disease. The impairment of the health takes its origin from the date of the *infecting* syphilitic sore. Early implication of the lymphatic glands leads to impoverishment of the blood as an immediate result, and then to degeneration or wasting of tissues, which attends the general cachexia, and which may eventually terminate in death, with complicated and varied lesions, especially implicating the internal viscera.

In individual cases the presence of a very extensive primary lesion, with well-marked induration, forebodes a severe subsequent course of syphilis. There is also some evidence to show that the locality where inoculation has taken place, to some extent, may influence the course of the disease. Some cases of infection on the fingers sustained by medical men, of whom I have had personal knowledge, have followed exceptionally severe courses. This is also the experience of Bäumlér and of Cazenave. The character of the eruptions is a trustworthy guide to prognosis. A pustular and papular eruption is believed to continue and intensify the contamination of the system; whereas, a roseolar eruption is a comparatively light affection—the mildest form of eruption. A pustular eruption

* During the last thirty-six years, venereal diseases have always formed a class by themselves in the Army Medical Returns, at first under that title—namely, "Venereal Diseases," and latterly as "Enthetic Diseases;" but the College of Physicians did not deem it necessary to retain a separate class for these affections. From a military point of view, however, they form so important a class, especially with reference to the amount of inefficiency they cause, and of invaliding to which they give rise, that the Director-General of the Army Medical Department continues to call for a special return of them annually from every corps; and the following instructions for its preparation have been issued:—

"In this Return, under the head of Primary Syphilis are to be included all those cases in which the venereal sore is one of the indications of a constitutional infection, usually attended with more or less hardness of the sore and induration of the inguinal glands, and followed by general constitutional manifestations.

"Under Local Venereal Ulcer are to be included all cases of sores arising from impure sexual intercourse, which are not attended or followed by any constitutional affection. These cases are frequently attended with inflammation and suppuration of the inguinal glands, which must be considered as a complication, and not as a separate disease.

"Care must be taken not to include in the Return cases of inflammation or suppuration of the inguinal glands arising from causes other than venereal affections: all such cases should be entered in the periodical Returns in Class II., Order 6, of the nomenclature. Cases of inflammation of the testicle, when the result of gonorrhœa (formerly returned as hernia humoralis and gonorrhœal orchitis) should be entered as gonorrhœal epididymitis, Class II., Order 10; when arising from other causes, they should be entered in the periodical Returns as orchitis, Class II., Order 11, and carefully excluded from this Return. (See Part II. of this Text-book, pages 339 to 353.)

"Blank lines are left for the entry of any cases which may appear so exceptional as not to justify their being included in any of the groups; but such cases must be specially noticed, and the grounds on which they have been deemed exceptional must invariably be appended to the Return.

"Medical Officers are requested to take particular care that all cases of venereal diseases are included in this Return, that they are grouped in accordance with these instructions, and that none but venereal cases are included."

From these returns the department will be able to give all the necessary information respecting the prevalence of this important class of diseases in the military service of the country, so as to show eventually how many men have become constitutionally contaminated by syphilis, and how many have merely had sores (simple or suppurating soft chancres), with or without glandular complication, and how many gonorrhœa simply.

in a tubercular form is of the most unfavourable omen; and, generally, gummy formations with a tendency to softening rather than resolution are unfavourable cases. On the other hand, cases of severe nervous symptoms due to syphilis often get well under proper treatment, which otherwise do not do so when causes other than syphilis are accountable for them. Dysenteric symptoms due to syphilis are generally speedily fatal; when a high grade of marasmus occurs, with marked pallor and emaciation, such patients die either from anasarca, sometimes with dysenteric affections, or with uræmia, or from a caseous pneumonia; or an erysipelas may be set up from some point of ulceration on the skin or nasal mucous membrane (BÄUMLER).

Treatment.—(a.) *Preventive.*—There are certain facts which must be kept in view in the consideration of prophylaxis. (1.) The disease spreads principally by sexual intercourse; (2.) prostitution is the source whence the syphilitic virus is ever freshly drawn, and the experience of thousands of years has shown prostitution, alike in the ancient and the modern world, to be an unavoidable evil, the suppression of which is impossible; (3.) with the intimate relations existing at the present day between all parts of the world, and the unbounded intercourse between different places at home and abroad which has been developed in recent times, syphilis is constantly being transported from one place to another. In the interest of public health it is the duty of the State not only to limit prostitution, but to exercise a supervision over it.

Keeping these facts in view, there are other facts which suggest the direction in which preventive methods for repressing the extent of syphilis ought to be persistently carried out, namely:—(1.) Official supervision over prostitution has had the most favourable influence in diminishing syphilis. This is shown in many ways:—(a.) Everywhere it is found that secret, unwatched prostitution is far more active in the spread of syphilis than prostitution which is under control; (b.) In 1866, the second Contagious Diseases Act was passed, after long and careful consideration, and is the one now in force. In the Army Medical Department Report for 1875, it appears that during three years immediately preceding the operation of those Acts the average annual ratio of cases of primary and secondary forms of disease was 104·4 per 1,000, whereas in 1875 it did not exceed 45·8, and in the period from 1870 to 1878, the average rate of prevalence of the disease fell to 44 per 1,000 men; and further, it appears that the ratio of primary disease was 53·4 in 1866, but in 1875 it had declined to 33·5. (c.) Through the courtesy of Sir W. M. Muir, K.C.B., the Director-General of the Army Medical Department, I am able to give the results for 1878. At fourteen stations under the Act, the average annual strength of the troops was 53,813, among whom admissions for primary venereal sores averaged 40 per 1,000, and 78 per 1,000 for gonorrhœa. On the other hand, at fourteen other stations not under the Act, the average strength of the troops being 45,316, the ratio of admissions among them was 86 per 1,000 for primary venereal sores, and 121 for gonorrhœa. Thus, at the stations not under the Act, the average rate of admission for both forms of disease was 103·5 per 1,000; as against 59 at the stations under the Act. At these fourteen stations

under the Act, the highest rates of admission for primary sores were 53 and 54 respectively per 1,000, at Aldershot and the Curragh; whereas at the fourteen stations not under the Act the rates of admission for these diseases ranged upwards to 140 in Warley, 154 in Dublin, 205 in Manchester, and 250 in London. So with regard to gonorrhoea, the highest rate among the fourteen stations under the Act was 110 at Winchester per 1,000; whereas at the fourteen stations not under the Act it ranged upwards to 151 in London, 161 in Warley, 163 in Sheffield, and 177 in Manchester, per 1,000. (*d.*) In *unprotected** cities like London, Manchester, Dublin, Sheffield, Preston, the severity of the cases is greater than in the *protected*† places, where the great majority (after the Acts have been in operation for a year or two) suffer from very mild forms of the disease. In 1870 a Royal Commission was appointed to consider the questions raised in the House of Commons on a motion for a repeal of the Act. The report of this Royal Commission speaks most decidedly upon the effects of the Acts in the reduction of the worst forms of disease among the lower classes of prostitutes, and acknowledges that they have both directly and indirectly promoted the objects sought to be attained by those Acts. "They have purged the towns and encampments to which they have been applied of *miserable creatures, who were mere masses of rottenness and vehicles of disease*," and it was agreed that, for the public good, particular districts which are from any cause peculiarly liable to contagious disease should be subjected to special sanitary legislation. (*e.*) In passing these Acts also, Parliament pronounced the strongest protest against the right of an individual suffering from a loathsome disease knowingly to communicate it to another; hence, in the interest of all, the State has the right to limit the personal liberty of such persons as may be considered dangerous to the

* The term *unprotected* district is meant to include the principal cities of the United Kingdom where women can practise prostitution in any public thoroughfare, at any hour of the day or night, without hinderance, and inveigle men to their homes; and this, too, under the very eyes of the authorities, who are powerless to interfere unless the solicitation be to the annoyance of any passenger or householder, who must in that case accompany the police to the station to charge the woman with the offence, which is rarely or never done. Should the authorities have credible evidence that the woman so conducting herself is suffering from a contagious disease, which she may communicate to others having relation with her, they cannot in any way interfere to prevent it. This woman, when unable any longer (in consequence of the severity of her disease) to follow her miserable calling, has no other resource (prostitution failing) to procure her a livelihood than to fall back on the foul wards of a workhouse (ACTON).

† A *protected* district is one where a woman, instead of continuing wholly or in part to gain an honest livelihood, walks the streets, soliciting different men, and consequently comes under cognisance of the police as being a common prostitute. The British Legislature has determined that in certain garrison towns such a woman, after being repeatedly warned and still persisting in a life of prostitution, shall submit to a medical examination, on the ground that, should she be diseased, she would communicate a contagious complaint to those having relation with her. It is further enacted that if the woman be found diseased she shall be at once sent to hospital, and not allowed to leave till she is, in the opinion of the surgeon, perfectly cured. If then desirous of abandoning her evil courses, the girl is relieved from further surveillance, and will be sent to her friends at the Government's expense. If, on the other hand, a common woman, on being discharged from hospital and released altogether from the operation of the Acts, returns to a protected district, and is again found by the police acting as a public prostitute, she must again be registered. In every case the authorities wish it to be clearly understood that they do not sanction or authorise prostitution; they only watch over it, from their anxiety to prevent its worst evils, and to ameliorate the condition of the women, and check disease (ACTON).

common welfare, and to use force, if necessary, to obtain an examination as to the condition of their health. Regular, frequent, and thorough examinations are absolutely necessary.† (*f.*) The beneficial effect of these Acts on the civil population cannot be ignored. During the five years 1870-74 the annual death-rate from syphilis (including congenital syphilis) in the eleven districts or stations where the Acts were then in operation, was 21 per cent. *lower* than it was during the five years 1861-65 before the Acts came into operation in 1866; whereas in the rest of England and Wales the death-rate from this disease was 20 per cent. *higher* in the second than in the earlier period. (*g.*) Up to the present time, it has been everywhere found that just that class of prostitutes who are the most apt to spread syphilis will not subject themselves to treatment except under compulsion. All prostitutes found to be syphilitic should be kept in hospital till they are cured. (*h.*) It may also be fairly urged and acted upon, that entire classes of MEN who notoriously contribute largely to the spread of syphilis, as, for instance, soldiers and sailors, should be subject to periodical examinations; and that sailors especially should be inspected before leaving a port, and again before entering another, and that if found infected they should be placed in quarantine. The workmen in some of the glass-works in the neighbourhood of Lyons have of their own accord demanded such examinations, to do away with the possibility of infection from the tubes used by them in blowing glass, which are passed from mouth to mouth (BÄUMLER). (*i.*) Not only ought the Contagious Diseases Act to be maintained and extended to the whole of this country, but the time has come when, as Dr. Bäumlér observes, it is the duty of all civilised states to combine and adopt some common course of action against these diseases. There is need of universal prophylactic regulations being more and more widely recognised.

A great responsibility rests upon those who oppose the working of such Acts—so fully proven to be preventive of disease, and in every

‡ It seems useless to argue with the opponents of these Acts, who, while loudly denouncing them as immoral because they render vice easier and safer to men, yet wish for the establishment by Government of voluntary lock hospitals and lock wards, which, if they proved equally efficient, must obviously be attended by the same result; who, while stating that the Acts render vice easy and safe, declare almost in the same breath that they are hygienically a failure, and that wherever they have been in operation disease has increased instead of diminished; who, while charging them with all sorts of pernicious and degrading influences, yet deny them any credit for the collateral benefits which they have been shown to have produced; who denounce them as unwarrantably interfering with individual liberty, on account of their compulsory clauses, and yet would not object to the compulsory detention of women in hospital until cured, provided they were left to apply voluntarily for admission. How, they further ask, if the present system is an infringement of personal liberty, is that principle less infringed if the compulsion is removed from one end of the procedure but retained at the other? Surely if compulsion is unjustifiable, it should be abolished altogether; but if it is to be retained at all, it should be retained in the form which has been found most effectual; and the evidence of those most competent to judge shows that the early detection of disease in public women by periodical examination is essential to the efficiency of the system. Even the Royal Commission were constrained to say, "We are satisfied from the evidence that the frequent examination of women is the most efficacious means of controlling the disease." It is not less important in the interest of the unfortunate women themselves, who obtain the immense advantage that their disease is discovered and treated at the earliest possible period, so that while they are prevented communicating it to others, they are themselves saved from all the serious and painful consequences of neglect (*Brit. Med. Journ.*, July 22, 1876).

respect beneficial. "In the eyes of every physician, and indeed in the eyes of most Continental writers who have adverted to the subject, no other feature of English life appears so infamous as the fact that an epidemic which is one of the most dreadful now existing among mankind, which communicates itself from the guilty husband to the innocent wife, and even transmits its taint to the offspring, and which the experience of other nations conclusively proves may be vastly diminished, should be suffered to rage unchecked because the legislature refuses to take official cognisance of its existence, or proper sanitary measures for its repression" (LECKY, *History of Europ. Morals*, Vol. II., p. 301).

(2.) The facts relative to the Pathology of syphilis which have been stated in the text, show that *Prophylaxis* ought to take cognisance of the vehicles by which healthy persons are apt to be inadvertently infected—(a.) From the use of such drinking vessels, plates, spoons, towels, &c., used by those suffering from secondary forms of syphilis; (b.) contamination from surgical instruments which have been employed upon the syphilitic; (c.) the selection of healthy children from whom other children are to be vaccinated—only those children known to be healthy, and whose parents are known to the physician, should be used as the sources of vaccine matter; (d.) the choice of wet nurses for healthy children ought also to be carefully selected, so as to exclude those who may show the least suspicion of having had syphilis; (e.) on the other hand, it is necessary to prevent children with hereditary syphilis being given to healthy wet nurses; (f.) the guardians of syphilitic children ought to be warned of the danger of their infecting healthy children through kissing them, and also by the use of the eating and drinking utensils of such infected children (BÄUMLER). (g.) As to matrimony after syphilis, see page 903, *ante*.

(3.) *Individual Prophylaxis*—From what has been written, it must appear clear that the only chance of preventing infection, alike on the part of the male and the female, is personal cleanliness after sexual intercourse. Much of the good that has resulted from inspection of females to the females themselves, as in Paris, Brussels, and other places, has been, I believe, mainly attributable to the greater attention to personal cleanliness which such inspections have brought about. Numerous lotions have also been used; and if a man will have sexual intercourse with a strange woman, let him wash immediately after the act, taking care to cleanse thoroughly the folds of the *prepuce*, especially near the *frænum*, and in the sulcus of the *corona glandis*. If a woman will have sexual intercourse with a strange man, let her use a syringe with hot water, to wash out the *vaginal passage*, taking care to cleanse thoroughly the folds of the mucous membrane at the orifice of the canal and of the *labia pudendi*. The real preventive remedy after exposure to infection is the most ancient, the most simple, and the most efficient—Wash, and be clean.

(4.) *Syphilisation*.—By this name an operation is meant which has for its object the eradication of syphilis from the system by repeated inoculations of the syphilitic virus itself. Dr. Boeck, of Christiana, is the most persistent advocate of this mode of dealing with syphilitic cases as a remedy against constitutional affections. And it was at one time seriously proposed to introduce syphilisation as a prophylactic, from its analogy

to the inoculation of the virus of variola (AUZIAS TURENNE). The origin and doctrine of syphilisation having been fully described in previous editions, will not here be repeated, as the method is altogether unjustifiable.

(b.) *Therapeutic treatment*.—As in the case of other specific diseases that are implanted by inoculation, the effect of the virus begins at once at the point of inoculation, and may not be destroyed as to its contaminating powers by any agency we know of. Experience shows that we must not conclude that, even by an early destruction of the sore, the occurrence of constitutional infection will be prevented; and the application of caustics to abraded surfaces after their exposure to contagion is quite useless to prevent infection. The poison is absorbed in a very short time—within a few hours (BERKELEY HILL). Still, however, in some cases positive results have been obtained which encourage attempts to arrest the *local* progress of the sores with escharotics, if they are applied at an early period, and before contamination of the system is evinced by indurations. Ricord and Sigmund have found that sores destroyed by the more powerful caustics, within *from three to five days*, have not been followed by syphilitic symptoms. But these may have been cases of soft chancre, which would not infect. The only efficient caustics for this purpose are—(1.) The *strong nitric acid*; or, (2.) The *potassa cum calce* (most conveniently used in the form of small sticks). *Nitrate of silver* is useless, from its limited action and deficiency of penetration.

If the sore threatens to slough, the parts should be wiped dry, and fusing *nitric acid* applied, and afterwards a lotion of the *potassio-tartrate of iron*, while the same drug is given internally. *Chloride of zinc* paste (Fell's) is a useful escharotic to excite a healthy action round the periphery of a sore. If great pain attends the local progress of the chancre, *morphea* in *liquor ammonia acetatis* is highly beneficial. "*Black wash*" of the strength of 8 grains of *calomel* to the fluid ounce of *lime water*, or weaker, as 5 or even 3 grains to the ounce, is of use in the treatment of large ulcerating indurations. The experience of 300 years has shown that *mercury* is a remedy which acts as a direct antagonist to the virus of syphilis; and no unprejudiced person can deny that its action upon syphilis is quite evident, especially upon the cutaneous manifestations of the disease. *Mercury*, administered during a primary sore, seems to be one of those agents which are able to break the regular order of the manifestation of symptoms, as it does to several other diseases; although it may not seem to possess any certain prophylactic power.*

* There are remarkable variations in opinion as to the influence of mercury in curing syphilis. At one time discussion ran high regarding its use; and, of course, extreme statements were made on both sides, while the facts adduced never warranted the extreme conclusions. Consequently, at one time mercury has been regarded as capable of absolutely preventing the constitutional affection; at another time it has been accused of giving to the syphilitic virus the impulse which sets up its worst results. It is now quite certain, however, that mercury administered continuously to the extent of salivation, or approaching it, exerts a poisonous influence, and produces constitutional effects of a very dangerous kind (GRAVES); and Hunter himself says "new diseases arise from mercury alone;" while it cannot be doubted that in cases in which mercury has been freely given, we are never certain that secondary symptoms may not supervene. Bärensprung, of Berlin, during his most extensive experience, came to the conclusion that syphilis not only can be cured without mercury, but, under its use, he believed the disease is often rendered latent for months and years, and its complete cure delayed. Starvation and Zitmann's weaker decoction were the means

The present position of opinion with regard to mercury in the cure of venereal sores seems to be this, namely,—That it is a very valuable and essential remedy in cases of syphilis, but not in cases of soft chancre; and the difficulty is to express always the nature of the cases for which it is

he employed for cure (*Ann. de Berlin Charité*, ix., 1, 1860; *Syden. Society Year-Book*, 1861). Hermann came to similar conclusions, from his experience in the syphilitic wards of the Vienna Infirmary. The experience of Diday is not less decided. He states that mercury cannot now be said to cure syphilis radically, so as to render all relapse impossible. He does not, however, as is the case with the others, practically withhold mercury in every case. If the primary lesion becomes an indurated, *woody* chancre, mercury is given. If the chancre is a doubtful one, he recommends waiting till some of the early constitutional phenomena render the nature of the case evident, and indicate the probable gravity of the syphilis with which he has to deal. He employs *iodine*, *iron*, and *quinine*, on the appearance of slight relapses, with a tonic and supporting regimen. He recommends *iodides*, to combat the *chloro-anæmia*, and to relieve the pain of tertiary ulcerations.

Numerous examples may be seen in museums, which are placed there to show that the poisonous effects of mercury produce worse lesions than those of syphilis; and, when combined with the *syphilitic virus* in a *strumous person*, the worst lesions of all. In the extreme of syphilitic infection, it ought never to be forgotten that a specific chlorosis results from syphilis, amounting to *anæmia*; and that mercury will bring about a similar *anæmia*. But while numerous instances are quoted by authors of the poisonous effects of mercury inducing lesions similar to those of syphilis, the investigations of Kussmaul, Professor of Medicine at Erlangen, show that mercurial poison produces no single affection or symptom that is identical with, or not easily distinguishable from, those of syphilis. His experience was among the manufacturers of mirrors using mercury in the town of Fürth; and he shows that mercury has but one influence over syphilis—that is, the power of controlling many of its symptoms.

Both kinds of treatment (mercurial and non-mercurial) have been extensively tried since 1816, and formal experiments have been organised on the subject, namely,—*First*, In 1822, in Sweden, by Royal command, when reports were annually furnished from civil and military hospitals as to trials of the two methods; *Second*, Dr. Fricke experimented in the Hamburg General Hospital, and published his results in 1828; *Third*, In 1833 the French Council of Health published a report on the subject. Long before any of these reports were initiated, however, the surgeons of the British Army had the boldness to declare themselves against the system of treatment with mercury, as it was then carried out, and, running into the opposite extreme, introduced measures of non-mercurial treatment altogether. This change was mainly due—(1.) To Mr. Fergusson, who practised it during the Peninsular Wars (*Med.-Chir. Trans.*, Vol. IV.); (2.) To Mr. Rose, of the Coldstream Guards, at the same time, but independently of Mr. Fergusson; (3.) To Dr. John Thomson, the first Professor of Military Surgery; who by lectures in the University and College of Surgeons in Edinburgh, and by his published writings, was mainly influential in convincing Scotch medical men of the evil effects of mercury in venereal diseases. The inquiry, begun in 1816 by these military surgeons, requires to be re-investigated with all the present advanced knowledge of the nature of the disease which we now possess, and with a better prospect of detecting the fallacies which surround the investigations. Under the simple treatment of those eminent men, there can be no doubt that mixed cases of soft, as well as of indurated chancres, and specific or syphilitic sores, were allowed spontaneously, as it were, to develop their distinctive characters. No confidence can now be placed in the results thus published, as derived from clinical observation, because the cure of *soft*, *suppurating*, and *mixed sores*, *gonorrhœa*, *vegetations*, *suppurating bubos*, are all indiscriminately given as evidence of the cure of *syphilis*. Moreover, cases cannot be accepted as cured at the time they are simply discharged from present treatment, because they may seem to be progressing to a favourable termination, but not absolutely cured, as is the case with the records of Fricke's experiments.

The great benefit obtained by the experiments, observations, and discussions regarding mercury in syphilis, carried on during the first quarter of the present century, as Dr. Berkeley Hill justly observes, has been to show—(1.) That all venereal ulcers can be healed without mercury; (2.) That this drug is not the cause of the relapses so frequent in syphilis; and, (3.) That very much less mercury is required to control syphilis than had been previously supposed necessary. Some interesting cases of syphilis cured without the use of mercury, are related by Surgeon-Major Dr. Peter Boileau, in the *Brit. Med. Journ.*, for July, 1879.

most suitable. Even those who believe most fully in its virtues acknowledge that in primary affections, as when given in the treatment of the local sore, its administration will not always *prevent the occurrence* of constitutional symptoms; nevertheless the value of mercury in the cure of THE INDURATION of the true *infecting chancre* is now fully recognised. The local lesion, if it appears after the usual prolonged period of incubation, is as much a manifestation that the constitution is already affected as is the developed vesicle of *variola vaccina* a manifestation that the constitution is affected with variolous poison. Looking, also, to the nature of the virus of syphilis, as expounded in the text (especially at page 904, *et seq.*), the *excision* of the *primary lump* or sore—the *specific induration*—as practised by Dr. Veale, assistant to the Professor of Medicine in the Army Medical School at Netley (*Edinburgh Monthly Journal*, July, 1864), and by Dr. Humphrey, of Cambridge (*British Medical Journal*, August 13, 1864), is a justifiable operation; for the original sore, when it has become a “lump” (as in its state of “woody-like” induration), is an undoubted maintainer of infection and of contamination of the system. If, therefore, it can be easily and completely insulated, as when on the *prepuce*, the cure of the constitutional symptoms may be facilitated and shortened. There are also certain forms of secondary syphilis for which the administration of mercury is unsuitable. These are the pustular eruptions, or ecchymatous states in *rupia* and in syphilitic anæmia. For the cure of other secondary symptoms mercury is certainly of service. If given to the extent to which I have limited its use in the text, I believe that secondary symptoms disappear more rapidly under its regulated use than by any other plan of treatment. All our treatment of syphilis rests on that evidence which must always guide the hand of the physician, namely, —practical experience. That has certainly taught us that the mercurialism of John Hunter's time was an error; and that its regulated administration in cases of *syphilis* is undoubtedly beneficial, and especially during the evolution of the specific symptoms of infection. Dr. Jeffrey Marston, late of the Royal Artillery, has given an admirable summary (*British Medical Journal* of Feb. 21, 1863) of the means and indications of treatment by mercury which he has found most useful. His experience shows that the system ought to be affected *as slowly as possible*; and there ought to be a remission of the remedy for a time as soon as that effect has been attained. As soon as the symptoms for which the *mercury* was given have disappeared, *steel* and other *mineral tonic* remedies ought to be given; and in three cases where the general health seemed to have suffered, *podophyllin* in small doses (*one-sixth* of a grain), with extract of *belladonna*, was given with marked benefit. If the system is too early brought under the influence of mercury, and the symptoms are not benefited, *chlorate of potash* in *compound tincture of cinchona* may be given with advantage; and in strumous subjects the *bichloride of mercury*, dissolved in *ether* and added to *cod-liver oil*, is found to be most useful. Some of the more intractable forms of syphilitic *squama* are best treated by a combination of *liquor arsenicalis*, solution of *bichloride of mercury* in very small doses, and tincture of *sesquichloride* of iron; while the use of soap in ablution ought to be avoided (STARTIN). In the administration of mercury for the cure of syphilis, *salivation*, or anything approaching to that condition, should never be

induced. *Tenderness of the gums* should be the utmost physiological effect, very gradually and gently brought about. As soon as the evolution of constitutional symptoms has commenced, such as the specific induration of the sore or glands, cutaneous, scaly, tubercular, condylomatous affection, or iritis, the sooner mercurial treatment (to the extent indicated) is commenced the greater will be the benefit. The good effects of mercury are known by the following results:—"Given early, it promotes the dispersion of the induration at the point of contagion or inoculation, and the enlargement of the glands; it delays and lessens the severity of the cutaneous eruptions, and of all the symptoms which accompany the early skin eruptions" (BERKELEY HILL). When induration is apparent, mercury ought to be at once administered; and all the useful effects of the remedy are generally obtained when the slightest possible sign of its influence is betrayed by the gums and breath. The most convenient form of administration is either a pill or fluid solution of corrosive sublimate (*perchloride*). The dose should always be small, and combined so as not to irritate the bowels. Grey powder or blue pill may be compounded in the following forms, namely:—*Two grains of grey powder with two grains of compound ipecacuanha powder*, made into a pill with glycerine, two of which are to be taken daily; or *two or three grains of blue pill* combined with a *quarter of a grain of opium powder*, with sufficient glycerine to make a pill, and taken every night at bedtime. Either of these forms is sufficient for women and young men. In adult men, *three grains of blue pill* or *grey powder* twice or thrice a day; or *one grain of calomel* every night and morning, with a *third of a grain of opium*, are most suitable. When the mercury is obviously producing its physiological results, it may be omitted for a day, and then again carried on in smaller or less often repeated doses. Dr. Berkeley Hill recommends that the *perchloride* of mercury be mixed with *iodide of potass*, so that a freshly formed *biniodide of mercury* is kept suspended in a solution of *iodide of potass*, in the following formula:—

R. *Perchloride of Mercury*, gr. 3; *Iodide of Potass.*, grs. 96; *Compound Tincture of Bark*, 4 ounces; *Sesquicarbonate of Ammonia*, grs. 60. Water sufficient to make up 8 ounces.

The dose of this compound ought to be *two teaspoonfuls*, half an hour before meals, three times a day. *Two-thirds* of a grain of *perchloride of mercury* may be taken daily; but except in such a solution as the above its action is uncertain. In Germany, Zitmann's decoction is much used. It is of two kinds—the stronger and the weaker. The former is compounded as follows:—

R. Rad. Sarsaparillæ, ℥xii.; Aquæ, lbxxiv.; *Coque per horas duas et adde* Aluminis, ℥iss.; Hydrarg. Chloridi, mitis (calomel), ℥ss.; Antimonii Oxy. sulphureti, ℥i.; *misc.* Coque ad $\frac{3}{4}$, et adde Fol. Sennæ, ℥iii.; Rad. Glycyrrhizæ, ℥iss.; Sem. Anisi, ℥ss. *Infunde per horam et cola.* The dose of this decoction is half a pint to a pint morning and evening

The weaker decoction is compounded as follows:—

Capiat residuum decocti fortioris et adde Radicis Sarsaparillæ, ℥ii.; Aquæ, lbxxiv.; *Coque per horas duas et adde* Cort. Canellæ, Cort. Lim-

onem, Sem. Cardamomi, ā ā ʒiii. *Infunde per horam et cola.* The dose of this decoction is one pint at intervals during the day.

Another mode of preparing it with somewhat different proportions of ingredients is given by Dr. Berkeley Hill, as follows:—

R. *Sarsaparilla*, ʒxii.; *Water*, three gallons. Macerate twenty-four hours, and put in a linen bag; *White Sugar*, ʒvi.; *Alum*, ʒvi.; *Calomel*, ʒiv.; *prepared Cinnabar*, ʒi.

Hang the linen bag in the liquor and boil down, while adding four gallons more water, till the liquor is reduced to two gallons; remove the bag, and add to the decoction the following ingredients, namely:—*Anise Seeds*, ʒiv.; *Fennel Seeds*, ʒiv.; *Senna Leaves*, ʒi½; *Liquorice Root*, ʒiss. Press and strain.

About half a pint to a pint is the daily quantity to be consumed of the compound. While the amount of *mercury* it contains is rarely enough to produce any unpleasant effects, it is enough to control the disease. A weaker decoction of the woods is free from *mercury*, but is of no value beyond a diet drink.

Iodide of potassium, in doses adjusted to the individual case, appears to act with rapid benefit in some of the syphilitic diseases of the interior of the cranium giving rise to extreme pain. Its administration often causes intense suffering in patients who have been treated previously by mercurials. Two distinct effects are produced:—*First*, the compounds of mercury fixed in the body are rendered soluble and active; and, *secondly* a form is given to them which allows of their elimination, with more or less rapidity, in a state of combination with one of the elements of the *iodide*; and thus the patient is subjected anew to a mercurial treatment by the compounds of mercury already present in his body (MELSENS, in *Brit. and For. Med.-Chir. Review*, 1853). The dose of *iodide of potassium* should at first be small—not more than *fifteen grains* in the twenty-four hours—increasing the dose if the patient bears it well (MELSENS, GUILLOT). Its action is aided by a blister over some portion of the shaven scalp, and by having the blistered surface dressed with mercurial ointment; and generally, it may be said that local treatment gives very valuable aid. For example, cutaneous or mucous *raised papules* remaining persistent, an ointment composed of *oxide of zinc*, *calomel*, and *simple cerate*, hastens their absorption. Eruptions of lichen, acne, and herpes are similarly benefited by the application of *oxide of zinc lotion* or *ointment*; and if *prurigo* and *urticaria* be also present, *diacetate of lead lotion* will expedite the cure. Vesiculo-crustaceous spots will cease to reappear if the affected parts are painted for a few days with a solution of *nitrate of silver* (gr. x.-xx. to ʒi.), and *oxide of zinc lotion* applied afterwards.

In the more advanced stages of the suppurative affections, the use of pyogenic counter-irritants ought not to be neglected, such as *tartar-emetic ointment*. They tend to keep up just so much of a discharge as may be consistent with the strength of the patient; and are worthy of a trial on the principle explained under *syphilisation* in previous editions.

In the dry forms of syphilitic cutaneous diseases, and in *chronic eczema* of the extremities, *tar ointment*, or an *alcoholic solution of tar*, is an excellent

application; and the disappearance of indolent glandular swellings is greatly aided by the use of strong solutions of iodine. Superficial forms of ulceration attending the pustules of *ecthyma* are benefited by the use of solutions of *nitrate of silver* or *sulphate of copper*, and generally by caustics and local stimulants. If a sloughing condition threaten ulcerating sores, lotions of the *potassio-tartrate of iron* will generally improve their aspect. In *psoriasis palmaris*, and similarly fissured conditions of the skin, *glycerine lotions* are most useful. But all these local remedies, it must be remembered, are only aids to the constitutional treatment, whether by *mercury* or *iodide of potassium*, or simply by a well-regulated hygiene. The patient, during the whole of the treatment, should be warmly clad—should be fed upon a good but plain diet—should take plenty of exercise in the open air in fine weather—should use occasionally (once or twice a week) warm baths—and avoid stimulants, unless specially indicated and prescribed. During the febrile stage and the exanthem stage he ought to be confined to his bed.

The administration of mercury, to affect the system rapidly, is best effected through the agency of the mercurial vapour bath. It is a mode of administration not liable to affect the digestion, and it permits other remedies to be given by the mouth at the same time, if they be considered necessary. It is also mild, slow, and equable in its action. The mercurial vapour bath is to be managed in either of the following ways. The first method is best adapted for the practice of a large institution; the method recommended by Mr. Lee is better suited for private practice:—

“The patient is seated on a chair, and covered with an oil-cloth lined with flannel, which is supported by a proper framework. Under the chair are placed a copper bath, containing water, and a metallic plate, on which is placed from one to three drachms of the bisulphuret of mercury, or the same quantity of the grey oxide, or the binocide of this metal. From five to thirty grains of the iodide of mercury may be employed, or a scruple of the iodide, with a drachm and a half of the bisulphuret. Under the bath and plate spirit-lamps are lighted. The patient is thus exposed to the influence of three agents,—heated air, steam, and the vapour of mercury. At the end of five to ten minutes perspiration commences, which becomes excessive in ten or fifteen minutes longer. The lamps are now to be extinguished; and when the patient has become moderately cool, he is to be rubbed dry. He should then drink a cup of warm decoction of guaiacum or sarsaparilla, and repose for a short time” (LANGSTON PARKER).

Mr. Henry Lee's mode of proceeding is more simple:—“A special and convenient apparatus is used (made by Savigny & Co.), which consists of a kind of tin case, containing a spirit-lamp. In the centre, over the flame, is a small tin plate, upon which from fifteen to thirty grains of calomel is placed, while around this is a sort of saucer, filled with boiling water. The lamp having been lighted, the apparatus is placed under a common cane-bottomed chair, upon which the patient sits. He is then enveloped, chair and all, in one or more double blankets, and so he remains well covered up, for about twenty minutes, when the water and mercury will be found to have disappeared.”

With regard to *sarsaparilla* as a remedy, Sigmund, Syme, and many

other acute observers, have come to the conclusion, after long and careful trials of the best sarsaparilla, that it does not, *per se*, exercise the slightest perceptible influence on the course and termination of syphilitic diseases. It is usually given in combination with some mercurial preparation, as in the decoction of Zitmann, already mentioned at p. 952.

As to the original local sore of syphilis, it may be dressed simply with lint soaked in warm or cold water, and renewed every three or four hours. The indurated glands, if they are painful, may be fomented, and the horizontal posture maintained. Moderate exercise may be taken if all local irritation is subdued; and then slightly stimulating lotions may be used to the sore, such as *sulphate of zinc*, in the proportion of *one or two grains* to the ounce; or the *liquor plumbi diacet. dil.* of the pharmacopeia; or the *black or yellow wash* if the surface is indolent.

CANCER—*Syn.*, MALIGNANT DISEASE.

LATIN EQ., *Carcinoma*—Idem valet, *Morbus malignus*; FRENCH EQ., *Cancer*; GERMAN EQ., *Krebs*—*Syn.*, *Cancer*; ITALIAN EQ., *Cancro*.

Definition.—*A growth consisting of a delicate fibroid stroma, containing within its meshes aggregated but not coherent cell-elements—cells, nuclei, or granules—generally UN-uniform, though often similar to natural cell-elements. It tends not only to spread continuously irrespective of the tissue invaded, and to multiply its elements indefinitely, and so to infiltrate into the surrounding structures, especially in the course of the lymphatics and nerve sheaths of the part affected; but it also tends to infect or reproduce itself in more remote and internal parts of the body: and finally, it tends to progressive softening and ulceration, with ultimate poisoning and exhaustion of the system. It is in this dynamical specific force of growth, spread, infectiveness, and multiplication, that the “malignancy” of “cancer” is expressed; and by virtue of which it is a disease sui generis.*

Pathology.—(a.) *History.*—The history of the development of cancers makes up some of the dark pages of Pathology. In many respects the origin of cancers is confessedly mysterious. Their first beginnings are generally hidden—deeply buried in the tissues; so that when a swelling manifests the possible existence of a tumor, it is regarded with *doubt* in the first instance, and then by astonishment as well as dismay at the rapidity of its growth. In such doubt, astonishment, and dismay, we recognise and acknowledge our ignorance. The normal course of cancer we now know to be that of steady increase—a steady and certain progress towards a fatal end. No radical cure for the disease is yet known. The hand of death is most unmistakably stamped upon those who are the subjects of cancerous growths; and we seek, in all humility, to learn something from an examination of the history of such a disease. But, to learn anything from pathological growths, they require to be studied and examined in *stages*, from time to time, in the course of their development. There are *certain stages* in pathological as in physiological growths, at which periods alone the actual mode of origin and development can be traced. A large cancerous mass does not look very promising; and

we might as well expect to be able to discover, by an examination of the mature foetus, the different steps by which its organs and parts had been formed, as hope to determine in what way a tumor, which at first was scarcely visible, had been converted into a mass of obviously malignant growth. Walshe, Bennett, Paget, De Morgan, Simon, Wedl, Rokitansky, Virchow, and others, have explained many of the phenomena of the growth of such tumors, while there is much that is still dark and mysterious in their history. On the 7th of March, 1874, a most valuable discussion on cancer was commenced by the late Mr. De Morgan at the Pathological Society of London—the results of which are recorded in the *Transactions* of that Society, Vol. XXV., for that year.

(b.) *Causation and Propagation of Cancer.*—The theories or hypotheses regarding the cause of cancer may be reduced to three:—

I. *The Constitutional Theory.*—That cancer is originally of constitutional origin—a specific state of the system existing with a true blood-cause and primary cancerous cachexia, the cancer-tumor being only the local manifestation—is mainly based on the following grounds:—

(a.) Its final universal diffusion throughout the body, all parts being liable to infiltration with the peculiar and specific growths which constitute “cancers or malignant tumors,” and that without any direct communication with the place where the first growth took place. (b.) Its occasional commencement in several primary tumors simultaneously. (c.) Its capacity to grow in various textures. (d.) Its local recurrence after operation or on removal of the primary tumor; so that the removal of the locally diseased part does not arrest the progress of the constitutional disease. (e.) Its appearance in internal organs, notwithstanding the complete extirpation of the primary tumors. (f.) Its repetition in families by inheritance. (g.) Its relation to tubercle.

There is thus some circumstantial evidence to show that malignant tumors are of *constitutional* origin—that they are local manifestations of a *constitutional* disease—that there is something specific, probably elaborated from the blood, which accumulates in their elements of structure. This specific material which composes cancers is different from all the natural constituents of the body; is different from all the materials formed in other processes of disease; and is associated in the malignant tumor with peculiar structural elements. There are also certain things in the history of cancer in which it is impossible not to discern a very large predominance of the constitutional element—especially, (1.) as to the *manner* in which cancer is inherited. It is a constitutional disease because it is inherited in a particular way—namely, without the inheritance being attached to any particular organ. “Cancerous disease, or a tendency to it, is prone to pass by inheritance from parent to offspring, and to occur (probably by inheritance of common properties) in many members of the same family and generation” (Sir JAMES PAGET). We cannot overestimate the importance of inheritance in the origination of cancer. On this point Sir James Paget thus records his more recent, varied, and extensive experience:—

“The more I have seen of cancer as occurring among families whose family history is known, the larger becomes the number of cases in which

well marked inheritance can be traced. When I was engaged almost solely in hospital practice, I thought the proportion was about one in six. When I came to know something more of private practice, I found I could count one in four. Now I can without difficulty count as actual facts, not less than one in three of the patients with cancer in whose families the occurrence of cancer is well known. But this number does not nearly represent what we may very safely assume to be the predominance of inheritance in cancer. A large number of persons die of internal cancer, and convey it to their offspring, though it is never known that they themselves have been the subjects of cancer, or, at least, is never recorded. A large number more die before they have manifested the cancerous disposition which is in themselves; for, paradoxical as it may seem, if a man have not outlived the utmost age of man, we are bound not to believe; but he might not have been the subject of cancer; for cancer is eminently a disease of degeneracy, a disease of which the frequency increases as years increase—i.e., in proportion to the number of persons living at each period of life, the number of cancer cases increases as age increases. So that unless a man have lived to the full age of life, he may have died of some other disease than cancer, and never have manifested the cancerous tendency which he yet conveys in *pre-disposition* to his offspring. The cases are very far from rare in which offspring die of cancer long before their parents. The parent lives and maintains that cancer was never known in the family; but a few years elapse, and then the parent dies of the very same disease as the offspring died of, having been quite ignorant of the conveyance of the disease of which the offspring died" (*Path. Soc. Trans.*, Vol. XXV., 1874, p. 317).

It is therefore of the very greatest importance to study the inheritance of cancer as indicating its origin. And, as to the *manner of its inheritance*, it must be contrasted with the inheritance of purely local growths and malformations. Fatty tumors, cartilaginous tumors, the ordinary cutaneous cysts and malformations, are often by inheritance, when they pass from progenitor to offspring, *in the same tissue if not in the same place*. It is not so as to the *manner of the inheritance of cancer*. The cancer of the breast in the parent appears as cancer of the lip in the child; the cancer of the cheek in the parent becomes cancer of the bone in the child. There is no rule in two cases, and absolutely no relation at all of place or texture. Sir James Paget gave a striking instance of this in the case of a lady who died with cancer of the stomach. One of her daughters died with cancer of the stomach, another died with cancer of the breast; and of her grandchildren, two died of cancer of the breast, two of cancer of the uterus, one of cancer of the bladder, one of cancer of the axillary glands, one of cancer of the stomach, one of cancer of the rectum. Hence he argues that it is inconceivable in such cases that we can speak of the transmission of *anything local*. That which was transmitted must have been *something constitutional*; and the locality of the cancer in each person was determined by some other circumstance than that of inheritance. This constitutional transmission of cancer accords with the transmission of all other hereditary constitutional diseases—with *gout*, with *syphilis*, with *tuberculosis*, and with *scrofula*—and not with what we see in local diseases. (2.) As to the effects of injury in the production of cancer, the same rule holds as with the influence of injury in the production of other constitu-

tional diseases. Cancer sometimes appears to come "of itself;" but in a large number of cases it follows injury sometimes so quickly, that it is impossible to disconnect it from the changes which the injury has produced. We know the limits of such changes in healthy persons. Either there is complete clearing up without change of texture; or there is some inflammatory formation, some thickening and hardening, or overgrowth of some texture like to the natural textures of the body; or, at the most, the production of some tumor which increases and grows on the place of injury, but holds the character of the texture in which it is seated. Any deviation from these ordinary results of injury, we usually regard as due to some constitutional defect or peculiarity in the person in whom it occurs; and when a cancer comes after an injury inflicted upon a part, it is because there was a constitutional predisposition to cancer in the patient—a fact which is still more strongly brought out in those cases in which we know of a cancerous inheritance in addition to the local injury; such, for example, as a cancerous mother having a son whose lip is incessantly irritated, and the lip becomes cancerous. (3.) The constitutional element in the origination of cancer is strongly marked in the constancy and in the method of its recurrence after operations—after complete excision. The number of cases in which it does not recur is not more than 1 in 500; whereas the ordinary non-cancerous tumor does not reappear in more than 1 case in 500. The *method* of reappearance in the one and in the other class of tumors is also very striking.

II. *Theories relating to the Blood*,—namely, (a.) That a cancer-tumor is the expression of a specific blood condition; standing in the relation to this blood condition of a secretion to its gland. (b.) That a morbid material is present in the blood, which, coming into relation with an appropriate tissue, enters into combination with, and causes the growth of, the tumor. Assuming the disease to be *constitutional* as to origin, we may not for that reason assume that there must be a morbid material present in the blood which incorporates itself in any form in the structure of a cancer; although by all the analogies of other constitutional diseases cancer ought to have a special material in the blood to which it owes, at least, a part of its origin. The morbid condition of the body, the ill-health or cachexia, which is associated with cancers, cannot be referred to the blood alone. Indeed, we have no direct proofs that the blood is peculiarly affected. We cannot find in it anything distinctive or peculiar to the cancerous diathesis, either by microscopic or chemical investigation. But while none can doubt the existence of a cancerous cachexia, we have no positive knowledge concerning the state of the blood in cancerous states of the body. It is rather inferred, from various considerations, that a peculiar material is separated from the blood, and is constantly being renewed in the formation of cancers. There are no germs in the blood previous to the development of a cancer-tumor which can be recognised as a cancer-structure. Nevertheless in some sense, however vaguely, cancer may be called a blood-disease; and in so far as it may be a constitutional disease, it is a disease of the solid tissues rather than of the blood.

III. *Theory of Local Origin*.—That it arises as a local disorder, independently of a constitutional cause, was the belief of the late Mr. C. R. Moore,

of the Middlesex Hospital, London, based on the following grounds:—
 (a.) Its invariable origin as a single tumor. (b.) The manifest dependence of later tumors upon the first, especially confirmed by the observed similarity of the morbid substance in the later growths to that in the first; by the order in which subsequent growths are disseminated; by the interruption to subsequent growth and dispersion of the disease, if the first and primary tumor be effectually removed. (c.) Its inheritance as a local and not as a constitutional peculiarity. (d.) Its preference for healthy-like persons. Mr. Moore concluded from these data, “that cancer has no dependence on any malady anterior to the appearance of the first tumor, but that it originates in persons otherwise healthy and strong. If this conclusion is inconsistent with prevailing opinions as to the cause and nature of the disease, the collision of the facts proves the need for more satisfactory evidence on behalf of these opinions than is at present in our possession. The existence of an antecedent general malady is, as far as I can perceive, pure conjecture, being entirely destitute of proof, or even of reasonable support. The idea sprang up in error; and it has been perpetuated mainly by the erroneous conclusions drawn from repeated want of success in surgical operations” (*The Antecedents of Cancer*, by C. R. MOORE).

Notwithstanding the strong statements contained in the last-quoted passage, each of the hypotheses I have mentioned has plausible grounds to rest upon. The evidence is also becoming greater which teaches us to look at the same time for a *local* and for a *constitutional* element as a necessary condition in the origin of cancers: for all that we see in the life of cancer naturally leads to the belief that the disease must from the first be more than a mere local tissue change; and that though it may appear to be altogether local in its origin, there is in some, possibly in all cases, a predisposition to the disease, which may be distributed through the system, but which more probably has its seat in some of the tissues of the body (Mr. DE MORGAN, Sir JAMES PAGET). There are some cases in which it is very hard to discover anything like a constitutional origin; such as cancers which occur in the scars of old burns, after many years of fretting and ulcerating, and at last becoming cancers. Also the perpetual irritation of a lip, which only after years becomes the seat of cancer. But even in these cases it is often observed that they suddenly change and become, not gradually cancerous, but the seats of cancer, indicating that there may have been some condition besides the mere local irritation out of which the cancer has been generated. Morbid anatomy also shows a series of morbid growths, from the simplest forms of fatty or fibrous tissue up to the most marked forms of cancer, in which it may be hard to indicate the point at which we could maintain that a constitutional element or predisposition has had to be introduced. On the other hand, we find growths unlike any others that are produced in the body—rapidly produced, rapidly multiplying, rapidly destroying—things so unlike any that we can call local diseases purely and simply, that we must seek for them another pathology. We are thus bound to believe that in the long groups of diseases which we call cancers, there may be at one end diseases in which it is hard to discern any constitutional element at all, and at the other end diseases in which the constitutional element very far predominates over

anything that we can suppose to belong to locality (Sir JAMES PAGET). In all cases of cancer the *constitutional* and the *local* element both exist; and in different cases the one or the other is predominant, the constitutional element, however, being the one which predominates most in the largest portion of cancers.

On the other hand, there are those who nevertheless contend for an entirely local origin of cancers; and that if the local origin of cancer be accepted it quite excludes a general or constitutional origin (MOXON, HUTCHINSON, ERICHSEN). It is a disease which ultimately kills by becoming general, but which at first is local. Hence the practical question in pathology put by Dr. Moxon is a crucial one—Does the general condition precede and cause the local? or, Does the local condition precede and cause the general? Both views cannot be true. Cancers appear earliest in a certain locality, and the danger to life consists in the extension from that locality. The disease becomes general and fatal through the influence of a part first cancerous. It spreads from that part; it runs along by the lymphatics to the glands; it makes its way along the blood-vessels to organs remote, through the medium of the circulation. It is also the same cancer-structure which diffuses itself—*e.g.*, cancer arising in bone passes to the lungs, and therefore arises in the lungs, by a sort of spermatic-like influence (SIMON), from the cancer bone. Hence secondary cancer has a likeness to the first. When liver becomes cancerous, the liver-cells may be seen turning to cancer-cells. The point of departure for the liver cancer-tumor is the liver-cell itself: and that not in the way of germination or multiplication of nuclei, but in the way of endogenous cell formation—*i.e.*, by vacuolation of the protoplasm of the liver-cell and the differentiation of the solid product that survived the vacuolation (Dr. CREIGHTON). Cancer, syphilis, and tubercle are thus shown—(1.) to arise locally; and, (2.) to become more or less rapidly generalised through an infective process or spermatic-like influence (Dr. CREIGHTON, Mr. SIMON, Dr. MOXON). But, whether it goes by lymphatics or by veins, it would not develop into cancer unless a primary condition existed in the patient—namely, a disposition under irritation to form cancer (Sir WILLIAM JENNER).

Another hypothesis may be here noticed—namely, the idea that local cancers were of the nature of entozoa, originally advanced by Adams and older writers. But it has never met with any proof, although it is entertained as probable by some; and there are many facts in the history of malignant tumors which indicate that their existence is attended with consequences very analogous to those of the existence of parasites. For—(1.) Every new formation which contributes to the body no serviceable structure must be regarded as a parasitical element in that body, alike foreign and injurious to it. (2.) The elements of malignant growths withdraw nutrition from the body, and in some instances elaborate specific secretions, to be stored up in their own structures. (3.) The malignant growth, whatever it may be, in the body or constitution, is the result of long-continued elaboration, and passes through a life of continual change before it attains its highest degree of malignant influence on the constitution. The alternate generation of the malignant growths is also exemplified in the frequent hereditary transmission of the cancerous

constitution. (4.) In active growth and partially independent existence we have another remarkable analogy to parasites. Malignant tumors grow and thrive, while the normal tissues far and near them are only able to maintain existence.

It has not been proved that the inoculation of cancerous matter is followed by even the growth of cancers, far less by the development of cancerous cachexia; and there is every reason to believe that cancers cannot be communicated by the secretions from a cancerous ulcer. Women, having the neck of the uterus destroyed by carcinoma, have conceived and borne children, and yet neither the husband nor the child have appeared to suffer in consequence. Alibert and others have inoculated themselves with cancerous matter, and yet no contagious effect has followed. Neither has this disease at any time been known to result from accidents incident to the examination either of the living or the dead person so affected. The experiments of Harley and Lawrence upon dogs have similarly failed to inoculate cancer. There are cases known, however, in which inoculation of cancers have failed at the time, yet, nevertheless, the subjects of inoculation have died of cancer at an advanced period of life; but the value of the observation does not as yet extend beyond a mere coincidence. Women appear to be more liable than men, and the increase is mainly due to cancers of the breast and uterus; while in man it is chiefly the skin, the bones, and the digestive organs which suffer. Age has also much influence in determining the forms of cancer and the part affected. Hard cancers are rarely observed till after forty, and from that period the liability increases with age. The most usual seats of cancers are also those on which age and functional activity have left their marks upon the organs. Thus cancer seldom occurs in the mammæ, uterus, or in the ovaries, till after the cessation of menstruation, nor in the organs of generation of the male till towards old age, nor in the different portions of the alimentary canal till after forty. Soft cancers are most common in the earlier periods of life; but then also they are observed to involve textures whose functional activity has been ever active, such as the glandular parts—for example, the lymphatic and lachrymal glands.

(c.) *Morbid Anatomy*.—The local exudations which constitute the tumors of cancers consist in general of two parts, namely,—(1.) The material peculiar to cancer, consisting of very varied forms of nuclei, nucleated cells, and juice, all of peculiar natures, distributed through an intercellular medium; (2.) Areolar tissue, which constitutes the stroma or skeleton portion of the new growth. The first is the essential and heterologous part of cancer. The relative quantity of these crude materials gives the most marked and obvious ground—that of consistence—on which cancers have been classified—namely, into *hard* and *soft* cancers; but the grounds of classification are by no means fixed. The nature of the substance affords a ground of classification. When the fibrous stroma is predominant, the new growth is *hard*, and has received the name of *scirrhus*. When the cellular elements predominate, the new growths are *soft*, and have received the name of *encephaloid* or *medullary* tumors. Sometimes the supposed nature of the substance is a ground for classification,—e.g., *meleceris*, *atheromata*, *steatomata*. Many names given to cancers are only

stop-gaps, tending to retard inquiry,—e.g., *colloid*, *alveolar*, and the like. The true nature, rather than form, ought to be the ground of classification. Cancers, again, are subdivided into a considerable number of varieties. For instance, the stroma takes various forms, like net or trellis-work, with large interspaces, or it grows papillary or villous in ramifications or vegetations. Sometimes it undergoes ossification, and then the skeleton of the cancer is formed of a network of true bony texture. This stroma part of the cancer is to be distinguished from the common binding tissue of the organ or texture into which the new growth is infiltrated. It is really and truly part of the cancer-exudations, and forms their basis. In the soft cancers it is more deficient, and then the cellular element predominates. The more distinctive elements of cancer-exudation have been said to exist in the cells, rather than in the stroma. The questions are, indeed, often asked, What are the characters of the true cancer-cell? Has the microscope discovered any structure which is decisive *per se* of cancer-growths wherever found? It may be said that it is significant of cancers that the forms of the elements of the tumors are after the pattern of those elements whose office is to separate whatever is refuse or abnormal from the blood—*i.e.*, the glands. Cancer-cells are formed on the types of the excretory gland-cells; and although they have no special anatomical characters, it is highly probable that malignant tumors eliminate something specific—something peculiar to each of them—which may yet be discovered. Much importance has therefore been assigned, and with justice, to the character of the cell-elements, as affording a specific distinction between malignant and benign forms of growth. Viewed, however, with reference to single isolated cells, it is now agreed that they offer in themselves nothing anatomically characteristic. It is the *grouping of cells and their varied forms* which, anatomically most of all, characterise cancers; and therefore I will select the best representations of the different cancers, and combine them in woodcuts, from Paget, Wedl, and Rokitsansky—with the understanding that cancer includes all forms of growth that can be classed as malignant diseases. (For the definition of “*malignancy*,” see p. 955.)

Malignant tumors are indicated by the following characters:—(1.) *Elements of structure and mode of growth*: (a.) Continuous development of simple cells, largely supplied with very fine blood-vessels, so that the tumor is very succulent. (b.) The growth is mainly a growth of cell-elements, to the exclusion of connective or fibrous tissue. (c.) In the first instance the cell-elements resemble the cells which compose the blastodermic membrane of embryos. (d.) If a new growth is composed mainly of cells, each containing growing matter within it, in the form of another cell, or cells, or granules, it must be regarded as of malignant tendency if it is supplied with blood-vessels. (e.) Generally a constant or continuous repetition of the same structural element is evidence of malignancy. (f.) A tumor consisting entirely of nuclei or of minute cells abundantly supplied with fine ramifying blood-vessels is perhaps the most malignant type of all. (2.) *The grouping of their elements*: (a.) The elements are heaped together disorderly. (b.) Multiplicity of elementary forms are sometimes seen in the mass, due to development or overgrowth, nodulation being an important feature in such tumors; or to degeneration,

calcareous, yellow or fatty; and to germination of cells. (3.) *Infiltration by juice through parts abounding in anastomosing tissue*: Nerve-sheaths and sheaths generally are proven to give facilities for the extension of cancers by continuity—an important point for the surgeon to recognise. The more free the anastomosis and the greater the facilities for the passage of juices, the more readily do the surrounding parts become diseased. Cartilage is slow to become cancerous by contiguity, so also are white and elastic fibrous tissues. Hence joints are often unaffected in the midst of cancerous tumors. The more readily the morbid juices are transferred from the original seat of disease, the anastomosing elements will be found more numerous. Thus nerves are often the best conductors for the spread of cancers, not because they are nerves, but because they are parts with soft interstitial tissue. Diffusion also takes place readily by means of vessels. (4.) *A peculiar tendency to ulcerate*, preceded by softening, with no disposition to heal, but a constant tendency to spread. A peculiar softness of texture precedes ulceration, which simulates the slow fluctuation of a thick pulp, and appears to be more liquid in some places than in others. The softening is a continuous process of secretion of fluid in many respects peculiar, and is a physiological property of their structure. (5.) *Malignant tumors constantly grow and progress to a fatal end*, and at the same time tend to multiply or propagate themselves. (6.) *There is scarcely a tissue they will not invade*.

It is in the comparative appearance of the multitude of cells, and especially in their relative size, that most distinguishing features may be observed. While the cellular cancer-mass often shows merely small pus-like or large lymph-like cells of an oval form with many nuclei, yet when all kinds of cancers are compared, "*typical cancer cell-elements*" in quantity may be described by the following general characters:—While the external cell-wall presents the greatest multiformity of shape, the dimensions of the cell are comparatively fixed to a mean of about $\frac{1}{1200}$ to $\frac{1}{1000}$ of an inch in diameter. Its nuclei, however, are more constant in appearance and more characteristic than the cells (LEBERT, BENNETT, PAGET, LAWRENCE). They are always voluminous in themselves, as well as in relation to the area of the cell in which they are inclosed; of a more regular form, being generally oval, or nearly rounded, clear and well defined, with a distinct single nucleus, and rarely two. Their average mean long diameter is from $\frac{1}{2500}$ to $\frac{1}{2000}$ of an inch. While Lebert attached too exclusive an importance to "a specific cancer-cell," he fully admitted the possibility that the most active and baneful cancers may vegetate through the system without exhibiting any of the cells now described, corroborating in this respect the general statement previously made by Dr. Walshe, "that a tumor may present to the naked eye the characters of encephaloid, and be the seat of interstitial hæmorrhage, affecting the communicating lymphatic glands, run in all respects the course of cancer, and nevertheless contain no cells but such as are undistinguishable from common exudation-cells." Muller, Bennett, Paget, and Lawrence have also since stated the impossibility of distinguishing the cell-element of cancer, as now described, in all cases, from the cells in certain other abnormal and even normal tissues, so that no *single* element can be considered as characteristic of cancer. What is supposed to be the

blastema or mother-fluid amongst which the cells and stroma of cancers grow, consists of an albuminous synovial-like, colourless, or pale yellowish fluid. It gives rise to the materials which compose the cancer-juice, and to the essential cancer-mass now described—namely, the stroma and the cellular elements. The development of cancer, therefore, proceeds *pari passu* with this fluid as amongst the interstices of textures, or on the free surfaces of membranes; and lastly, even by endosmosis of the specific fluid into the natural cells of parts (such as into cartilage or bone-cartilage cells), the characteristic cancer-mass may commence to grow by endogenous growth within such cells hitherto healthy. The late Dr. J. H. Bennett, of Edinburgh, and Dr. Van der Kolk, of Utrecht, have made most interesting observations on the extension of cancers by means of the parenchymatous fluid; and from all that has been observed on this subject, the latter author draws the following conclusions:—

(1.) Through an interchange of material taking place between cancer-cells and intercellular fluid, the latter acquires the property of forming new nuclei and cells of a similar nature. (2.) This intercellular fluid passes, along with the parenchymatous fluid pervading the sound parts, into the textures adjoining the tumor. The parenchymatous fluid thus acquires the same constituents and tendency to form similar cells, which now become developed among the healthy surrounding tissue in the course of the areolar membrane. (3.) On account of the minuteness and small number of the last-mentioned cells, their presence cannot be detected with the naked eye; so that the surrounding parts may appear to be perfectly sound, notwithstanding that they contain the germs of the advancing formation of cancer. (4.) It is therefore of importance, in removing cancer by operation, not only to take away at the same time a large quantity of the adjacent sound parts, but also to examine the innermost sectional edges under the microscope, in order to ascertain whether any trace of cancer-cells in process of formation is to be discovered in them. (5.) The existence of burning or shooting pains in carcinoma may be taken as a proof that the cancer-cells have reached the neighbouring nerves, and the disease can then scarcely be looked upon as a local one, in which an operation might be permanently successful. (6.) By the absorption of the infected parenchymatous fluid through the lymphatics and veins, the whole body seems to become more or less tainted, so that secondary cancer ensues in distant situations, when, as is self-evident, operation can no longer be thought of. (7.) This altered parenchymatous fluid penetrates the organic tissues which are washed by it, the sarcolemma of the muscular fibres, the tubes of the nerves, and the like. These membranes, too (both the sarcolemma and the walls of the nervous tubes), appear to take up the altered nutritive fluid; the consequence of which is, that both within the sarcolemma and the nervous tubes similar nuclei and cells arise, accompanied with an absorption of the muscular fibre and of the contents of the nerve, and attended with the deposition of fat, by which these parts waste and are destroyed, while the surrounding membranes (sarcolemma and walls of the nervous tubes) remain" (*Brit. and For. Med.-Chir. Review*, April, 1855).

The cancer-juice is a most important element of the new growth. To the naked eye it appears as a viscid, whitish, creamy, yellowish fluid,

which may be squeezed or scraped in considerable abundance from the surface of a section.

It is from the performance of the vital functions by all these elements that we are to draw our conclusions regarding the innocency or malignancy of the new growths in which they form a part. If we find adjacent textures are being infiltrated, poisoning the lymphatic current which passes from them; inducing by infection new growths of a like nature in the lymphatic glands through which these currents pass; affecting the general system with a peculiar cachexia, marked by languor, debility, emaciation, and a peculiar sallow, leaden-like colour of the skin; and if after removal such growths return, then there is no doubt that the cachexia of cancer is made manifest by such local lesions. These present so many elements in common, that all these new growths are but manifestations of one disease, which has been named "cancer," and attributable to the constitutional cachexia already described. The varieties of this disease also run into one another by characters which are so insensible that definite lines cannot be drawn between them, and thus many species of cancers are described under various synonyms, as shown in the following nomenclature:—

I.	II.	III.	IV.
WALSHE AND BENNETT (1846-49).	PAGET (1853).	ROKITANSKY (1855).	COLLEGE OF PHYSICIANS (1869).
Scirrhus, or hard cancer.	Scirrhus, or hard cancer.	1. Fibrous carcinoma.	(a.) Scirrhus.
Encephaloma, or soft cancer.	Medullary, or soft cancer.	2. Medullary.	(b.) Medullary cancer.
Colloid, or jelly-like cancer.	Epithelial.	(a.) Villous cancer.	(c.) Epithelial cancer.
All other forms of cancerous new growths are described by Bennett under the name of <i>canceroid</i> growths.	Colloid.	(b.) Cancer melanoides.	(d.) Melanotic cancer.
	Osteoid.	3. Epithelial cancer.	(e.) Osteoid cancer.
	Melanotic.	4. Gelatinous cancer.	
	Villous.	5. Carcinoma fasciculatum.	
	Hæmatoid.	6. Cystic carcinoma.	

Of these particular kinds of cancer some tend to affect certain organs rather than others; for instance, *alveolar cancer* is more frequently found in the stomach or intestines; *epithelial cancer*, in the skin and mucous membrane. The malignancy of such morbid overgrowths consists in the tumor threatening life in one particular way—namely, by exciting a definite constitutional malady incompatible with the healthy nutrition of the organism. Such constitutional ill-health (cachexia) is indicated by increasing prostration of strength, diminution in the amount of blood, its watery character, emaciation, earthy tint of the skin, profuse sweating, diarrhoea, hæmorrhages. These are clinical evidences of malignancy which are always *secondary to the local morbid growth*, and generally in direct proportion to its size, to any discharge from it, and to the involvement of lymphatics and the digestive organs. Primary growths of this nature seem also to possess the property of exciting the development of similar growths elsewhere, and so gradually to extend their influence over the entire organism—a property and influence called "*constitutional infection*." This constitutional infection proceeds by three modes or stages to affect the system; namely—(1.) By the continual production of new foci of growth at the periphery of the central growth, with which they subsequently coalesce by infiltration of the nutrient fluids of the growth into the surrounding tissues. The

migration of specific cell-elements thus takes place from the original growths, which then proceed to germinate and form secondary growths or foci in the neighbourhood of the original tumor, by which it is more or less rapidly increased. (2.) By implication of lymphatic glands receiving lymph directly from the affected part through the lymphatics, which convey some material elements to these glands directly infecting them, and causing in them a similar development to that of the tumor. Such material elements are, no doubt, the tumor-elements themselves, or such young germs as have acquired infecting powers, or even fluid conveyed from the malignant growth. Implication of the lymphatic glands is therefore an unfavourable sign in connection with a tumor, because it is a proof of the infecting property of that tumor, and because these infected lymphatics and glands become new centres of infection. (3.) By the production of secondary similar growths in other regions of the body, which are not in any direct anatomical continuity with each other. To this the name of "metastasis" has been given. It is believed that this mode of extension takes place mainly through infection of the blood, and it is usually the last process in the history of constitutional infection from malignant growths. The general current of the blood may so become contaminated through the lymphatics, or more directly by the passage into the blood-vessels of specific elements from the primary tumor, which has ulcerated through the walls of vessels, laying open their interior, which may thus receive, carry off, and disseminate through the body the specific element of the malignant growth. At the first set of capillaries sufficiently small in any solid organ to arrest these elements, there the secondary growths will develop themselves. In malignant growth of the stomach and mesenteric glands, the blood, returning by the portal vein, infects the liver, and ultimately the lungs, with similar secondary growths; or the liver may be passed by and the lungs alone infected. Each and all of these modes of extension of primary growths infect the constitution, and are evidences of *malignancy*. It may be further stated, that the *anatomical signs of malignancy* in a tumor are mainly based on their richness in blood-vessels and lymphatics; on their comparative vascularity, and the greater amount of juice-carrying quantities of amœboid active growing cells. *Malignancy* is thus a clinical, and therefore a practical, rather than a theoretical character. It is not applicable to all tumors which endanger life. Any tumor may do so by mere position, size, or weight. A tumor or new growth, otherwise innocent as to anatomical constituents and infecting or physiological properties—such as fibroid growth of prostate or of uterus—may cause death; the former from retention of urine, the latter by uterine hæmorrhage. The mere multiple character of tumors is no sure sign of their malignancy. The primary and independent origin of each may be due to the same cause: such are lipomata, fibromata, sebaceous growths, and warts. The multiplicity of cancers is a secondary phenomenon, and due to infection from the original growth. The growths termed *cancerous* (the *carcinomata*, namely *scirrhus*, *encephaloid*, *epithelioma*, *colloid*) are pre-eminently malignant; but some *sarcomata*, *enchondromata*, and *recurrent fibroid* tumors are in some cases not less malignant than cancers—a property not always assignable to their being

combined with cancer. The recurrence of malignant growths at the seat of operation after their removal is another clinical characteristic of them. To a certain extent this circumstance may be explained by the fact that although the main bulk of the growth is removed by the knife, yet, from its *infiltration* of the neighbouring tissue, and the growth of multiple foci of microscopic dimensions at its periphery, the removal has been *incomplete*. Some germs of specific growth have been left from which subsequent recurrence proceeds. These germs of growth extend to an indefinite and indeterminable extent beyond the original and obvious tumor. But there are also some growths which have this tendency without being malignant. *The primary cancer-growth* commences in the textures of some organs rather than others; for instance, in the uterus and female breasts, in the stomach, the colon, the liver, the bones, and the brain. *Secondary cancers*, on the other hand, are most frequently developed in the lungs, the spleen, the salivary and lymphatic glands, in the small intestines, and in the serous membranes. But the local aptitude for cancer-growths is a subject unknown. We have no knowledge why one part rather than another should be the seat of cancer; but certain organs seem more liable than others at certain periods of life. For example, before thirty years of age the eye and the orbit are the parts first most liable to be affected, next the bones, testicles, and areolar tissue of limbs and trunk. Between thirty to fifty years of age the penis, uterus, external sexual parts, and breasts; after fifty years of age the integuments and digestive organs are more apt to suffer. Simple tumors may also become the seat of cancers, just as any part of the body may be the seat of a cancer, although the event is rare. About one-fifth of cancers are ascribed to the effects of injury; and although in many cases the statement may be fallacious, yet the consequences of injury are sometimes too obvious to admit of doubt. The following is a description of the typical cancer growths:—

(a.) SCIRRHUS, or HARD CANCER, has two stages—a hard stage and a stage of softening. It is a “*cancer characterised by hardness of the primary tumor, and by a tendency to draw to itself the neighbouring soft structures. When ulcerated the sore is commonly deep, uneven, and bounded by a thick everted edge.*” The local hard or scirrhus state constitutes the first stage. It may grow in *masses*, or may be *infiltrated* into the tissues of the organ or part affected—the latter being by far the most common form. When in masses, they are generally lobulated, dense, and often contained in a cyst. When these masses are cut into, we find them to consist of two substances—the one is the cancerous growth, and the other is areolar tissue; so that the appearance of the divided surface in general is that of a hard, white, semi-cartilaginous substance, streaked by fibres radiating from what appears to be the centres of connection to the circumference. They are of considerable density and firmness, and in hardness of texture vary from hard-boiled white of egg to cartilage—the knife making a grating noise as it cuts through them. The specific weight of these tumors is extremely great; and in those parts which are external and more or less pendulous, as in the mammae, this fact is, by some distinguished surgeons, regarded as an element in diagnosis: but exact information relative to the specific gravity of cancerous growths is still very limited

—1.040 to 1.160 is the very wide range which I have observed such tumors to indicate. This cancerous growth, however, is much more



Fig. 101.*

an inch, or perhaps even more. On the contrary, when infiltrated into the cutaneous tissue, the layer is often so attenuated as to be scarcely appreciable, and the disease

frequently infiltrated among the areolar tissue of the different organs or tissues it affects (Fig. 101). The affected tissue becomes gradually increased in thickness and in density by a slow growth of the morbid matter, so that the part, if divided, presents the same hard, semi-transparent character as in the mass, but it is more interspersed with areolar tissue, the diseased portion being gradually shaded off into the healthy structures. In the mucous tissues, as those of the stomach or uterus, the infiltrated growth has often a considerable thickness, measuring from a quarter of an inch to

a small hard pimple, or a small erysipelatous tumor, or even by a slight fissure or crack in the skin.

The minute elementary structures of scirrhus are chiefly two:—(1.) Certain cells and corpuscles; (2.) a nearly homogeneous intercellular substance, in which they lie imbedded. As to the cells, multiformity of shape is their chief malignant characteristic (Fig. 102). After a certain but indefinite period, which varies from a few months to a few years, the scirrhus stage of *hard cancer* terminates, and the second stage, or that of *softening*, begins. In mucous membranes this softening usually takes place at the *surface*, or superficially,—as at the mucous surface of the neck of the uterus, or at the mucous surface of the stomach. An ulcer is the consequence of this softened state, and is at first superficial, presenting many remarkable varieties, such as an inverted or everted edge, and an irregular form, while its base may be granulating at one part and sloughing at another. Its course is burrowing, often penetrating between the cancerous lobules, and ultimately may perforate any limiting serous covering, such as the peri-

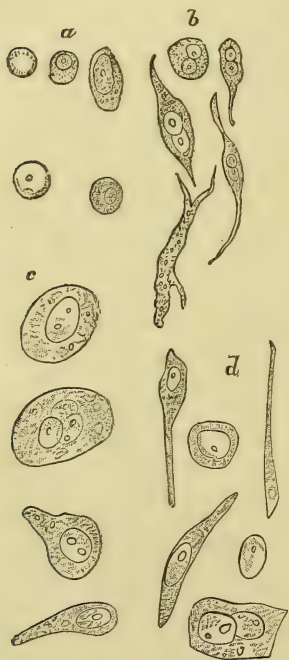


Fig. 102.+

* Cancer cells of scirrhus, filling interstices among bundles of connective tissue, in the skin of the breast—magnified about 200 times. (After PAGET, Fig. 99, p. 615, 3rd Ed., 1870.)

+ Elementary cells of scirrhus cancer from the rib. *a*. Rounded cells with single nucleus; *b*. Cells with two nuclei and fibre-cells (WEDL, Fig. 161, p. 573); *c*. and *d*. Cells and free nuclei of scirrhus cancer from cancerous breasts—magnified about 500 times. (After PAGET, Fig. 100, 3rd Ed., 1870.)

toneum. The pus secreted by this sore is fetid; often a mere ichor, or pus mixed with blood, and so acrid as to inflame the parts over which it flows. In a few instances the large vessels participating in the disease ulcerate, and the patient dies of hæmorrhage. A cancerous mammary gland has been known to remain indolent for fourteen years, and has at the end of that time been removed by an operation. This indolent character of cancer is limited, however, to the hard stage; for, after the growth has commenced to soften, its course is rapid, and a few weeks or a few months generally terminate the patient's life, the part affected in no instance cicatrising, or being again restored to a healthy condition.

(b.) MEDULLARY CANCER, or SOFT CANCER, affects more especially the solid visceral organs. It is a "*cancer characterised by a smoothly lobed surface, soft, irregular consistence, great vascularity, and usually rapid growth and reproduction. When ulcerated, it protrudes in large masses, which bleed copiously.*" Its cell-products are most profuse, and its course much shorter than hard cancers, the disease generally terminating in a few months. While hard cancer for the most part affects persons in the decline of life, soft cancer is most common in its earlier period, or adult age, from twenty-five to forty. Although generally found in masses, it may be infiltrated: the former is the more common form, the latter the more rare. In whichever form it grows, however, it has two stages—namely, one of induration and one of softening. In the first stage, we find it composed, as in hard cancer, of very delicate stroma tissue and a peculiar morbid growth. The areolar stroma is of various densities, often extremely fine or of considerable consistency and tenacity, and in either case radiating through the tumor and dividing it into lobules. The morbid substance or growth is of many degrees of hardness, varying from lard to cartilage, but is generally softer than in hard cancer: it is of a bluish semi-transparent whiteness. The duration of this hard stage is from a few weeks to two, three, or four months, and only in a few instances does it exceed that latter period. "*Hard encephaloid* is a designation sometimes applied to medullary cancers of unusually firm consistence." The woodcut (Fig. 103) brings together representations of the experience of Paget and Wedl, as to the varied microscopic forms which the new growth of medullary cancer assumes. The firmness of cancers seems to be in proportion to the abundance and fineness of the elongated cells figured in *c* and *d*. The SOFT cancers are mainly made up of cells and stroma, as in *a* and *b*, more or less mixed with *c* in the figure. The first stage passed, the process of softening, or of *ramollissement*, takes place. This is evident on cutting into the tumor and passing the handle of the scalpel over the divided surface, a milky white substance being expressed. As the disease proceeds, the parenchymatous substance of the new growth is changed into the consistence of soft cerebral matter, or of thickened pus; it is consequently opaque, and varies in colour from white to red, and even black. These variations of colour appear to be owing to the different quantities of blood or of melanic matter which are effused, and with which the cancerous matter is commixed. When bloodless and white, the product is so peculiar that it has been termed *cerebriform*, and when mixed with blood, *medullary sarcoma*, *fungus hæmatodes*, and many other terms,

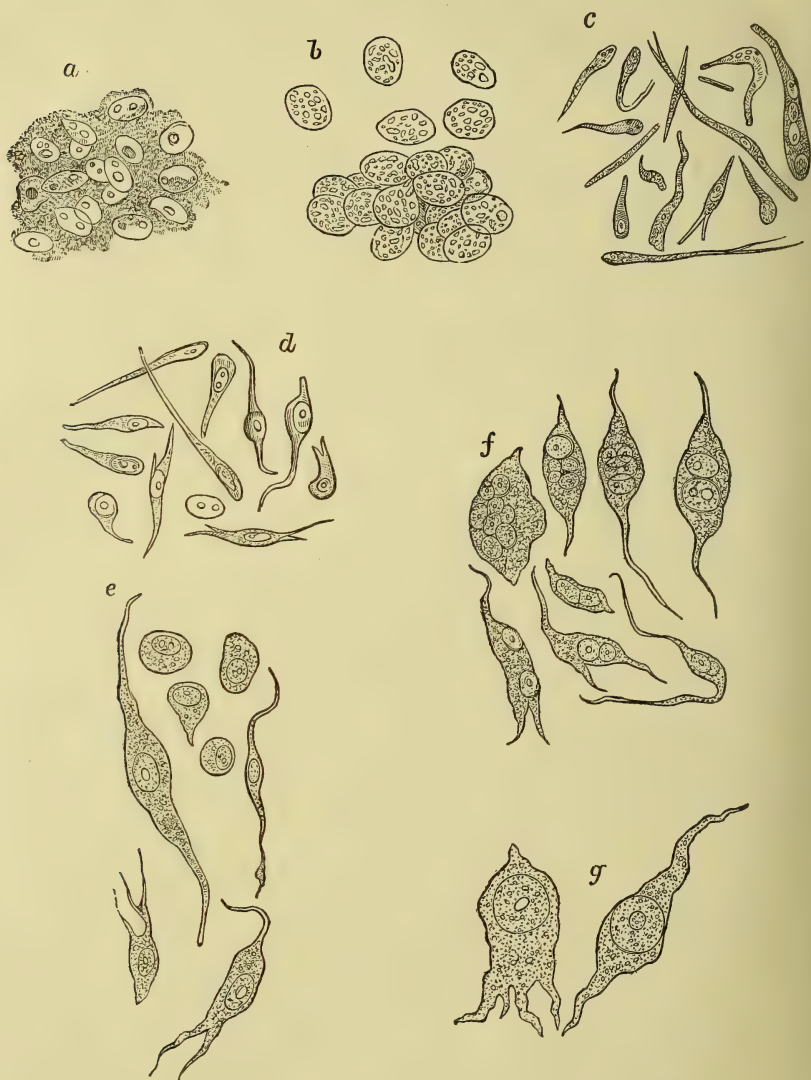


Fig. 103.

(a.) Nuclei of Soft Medullary Cancer imbedded in a molecular basis stroma, and without multiform cells—magnified 500 diameters. (After PAGET, Fig. 105, p. 668, 3rd Ed., 1870.)

(b.) Dotted nuclei of Medullary Cancer, containing several shining granules. (PAGET, *loc. cit.*)

(c.) Developed nuclei of Medullary Cancer, (PAGET, l.c.)

(d.) Caudate and elongated cells of FIRM Medullary Cancer—magnified 450 times, (PAGET, l.c.)

(e.) Medullary cancer-cells of multiform shape, with nucleus and prominent nucleoli—cancer of breast. (After WEDL, Fig. 166, p. 592.)

(f.) Medullary cancer-cells with two to eight nuclei (WEDL.)

(g.) Medullary cancer-cells with large vesicular nuclei and granular nucleoli (WEDL.)

according to the different quantities of that fluid effused, which is often so abundant that the cyst or cavity at length contains little else than fibrine. "*Fungus hæmatodes* is a term applied to some cases of medullary cancer which are more than usually vascular." The process of softening seems to commence indifferently in any part of the medullary tumor, at its centre or towards its circumference; and if the tumor communicates externally, the quantity of softened matter discharged often amounts to many ounces in the course of the day. This profuseness of discharge appears to be owing to the great vascularity of the growth; for although in the hard stage only a few blood-vessels, with coats of great tenuity and delicacy, can be traced between the lobules, yet, in the softened state, a successful injection shows the growth to be made up almost entirely of blood-vessels. The duration of the second stage is generally a few weeks, and very rarely extends to months. It appears, however, that anything which greatly irritates the part accelerates the process of softening. Thus, if a cancerous limb or tumor be amputated, the cancerous matter primarily in a hardened state appears to grow in new sites, subsequent to the operation in a softened condition, no previous hard stage occurring. The minute organic structure of this form of disease in its hard state, is probably not dissimilar to that of hard cancer; and of the vital organic characters there can be no doubt of their similarity. There is scarcely any organ or tissue in which soft cancer has not been found; and by some pathologists the frequency of its occurrence in certain parts is believed to be in the following order:—the liver, epiploica, the mesentery, the lymphatic glands, the brain and nerves, the spleen, the testicles, the uterus and ovaries, the eye, the bones, the heart, and lastly, the blood-vessels. It has been stated that soft cancerous matter far more frequently grows in masses than infiltrates into these parts. In general there is only one tumor; but there may be, as is often seen in the liver, three or four, and in some cases they are extremely numerous. Dupuytren has met with a carcinomatous heart which contained more than 600. In size they commonly vary from a millet-seed to a large egg; but when they form in loose cellular tissue, as between the folds of the mesentery or of the *epiploica*, or in the substance of the lungs or testicle, they have been known to weigh, in extreme cases, 20, 30, 40, and even more pounds. These tumors may also be encysted or non-encysted. One of the most constant features of this disease, which distinguishes it from hard cancer, is, that it often appears in many organs or tissues at the same time in the same patient. Thus, it has been met with in the coats of the bladder, in the liver, and in the lungs of the same individual. It has also a greater tendency to be reproduced after an operation for its extirpation than any other kind of cancer. This reproduction may take place either at the part operated on, or in some organ or tissue distant from the primary seat of the disease.

(c.) EPITHELIAL CANCER, OR CANCROID EPITHELIOMA.—Of late years, under these names, or that of *Cancroid* only, Lebert, Bennett, Hannover, and Paget, have described a form of tumor which has all the vital and malignant qualities of cancer already described, but its minute elements consist of cells resembling those of epithelium or epidermis. It is therefore defined as "*cancer characterised by its occurrence chiefly in parts naturally*

supplied with epithelium, and by the resemblance of its cells to those of the epithelium." It occurs almost solely on the skin or mucous membranes, being frequently seen on the lips or cheeks. Rokitansky has observed it in the liver; Bennett has described it as commencing primarily in the lymphatic glands; and in two cases of what has been termed acute hypertrophy of the mamma, both of which proved fatal with secondary lesions in internal organs, which I examined after death, I believe that this form of cancer was the primary local lesion in the gland (*Med. Times and Gazette* for 1857). It only seems to occur where there is pavement or spheroidal epithelium. Lebert has seen it on the serous layer of the arachnoid, and Robin has described it on the interior of a vein in a horse. The principal sites are in the vicinity of the great orifices of the body; but the lips, and more particularly the upper lip, are most liable to the development of epithelial cancer. The cancer commences first as a small induration, afterwards a pustule or excoriation forms, and subsequently the deeper parts participate, and determine the form of the growth, which may be mulberry-shaped, villous, cauliflower-like, or nodulated. The surface is apt to become ulcerated at an early period, and an actual loss of substance takes place from the central parts. The resulting open sore has an irregular, grey, and often a bloody base; or it is covered with crusts, from below the edges of which a fetid and ichorous discharge may be caused to exude by pressure. When the tumor is cut into at an early stage, the section presents a well-defined border of epidermis, sometimes several lines thick, while the rete between the surface-layers and the chorion is much hypertrophied. The substance is friable, and is easily separated from the surface of the chorion. The tumor soon tends to take deep root in the substance of the chorion, presenting a white and grey speckled surface, with a basement substance in which a variable quantity of whitish bodies may be seen, varying in size from a visible point to the size of a millet-seed. The fluid which exudes is milky and granular, and does not mix with water, so as to form such an opalescent, semi-transparent emulsion as the mixture of other cancer-juices with water. It mixes as if it contained fat. The tumor tends to infect secondarily the neighbouring lymphatic glands; and the fatal termination is generally by exhaustion or by putrid infection, when the progress of ulceration is not arrested. The mean duration of cases of epithelial cancer is about six and a half years; and the duration seems to vary with the part as follows:—Nearly three and a half years for the lower lip; three and two-third years for the penis; nearly nine years for the neck and limbs; and nine and a half for the vulva and face (LEBERT, HANNOVER). Not a few pathologists doubt the propriety of classifying *epitheliomata* with cancers. Mr. Collis and others do not consider that the affinities of epithelioma with cancers are sufficiently strong to warrant their being included in the canceroid group of new growths. Epitheliomata originate in the deeper layers of the epidermis or epithelium of mucous membranes, where their development and growth may be recognised in three stages:—(1.) Simple cellular hypertrophy. (2.) Hypertrophy with ulceration, in which the number of cells has so increased as to press down on the papillæ, and cause ulcerative absorption of them by pressure. (3.) Hypertrophy with infiltration, in which enlarged papillæ overtop the masses of cells, which not only

press on and ulcerate the papillæ beside them, but also push the new-formed cells downward between the papillæ. It is this continuous cell formation which accounts for the widespread destruction of parts induced by epitheliomata. The growth gradually presses on before it, and destroys whatever tissue it meets with. Great irritation is also given to the blood-vessels by the germination of the cells, and the pushings of them along between the separated papillæ, producing an interstitial effusion of plastic lymph. In this lymph the epithelial cells develop fresh nuclei and cells like themselves, and thus become a fresh centre of irritation, the disease advancing by fresh development of cells in the interstitial effusion. "Its superficial origin, its slow progress, its indisposition to infiltrate the deeper structures, or to contaminate the glands, the certainty of cure which follows its timely removal, and the different appearance when occupying similar localities, are of sufficient importance to outweigh the points of resemblance which it undoubtedly bears to cancer in its advanced and secondary stages. In its early stage it is strictly an hypertrophy, and in this condition it may remain for an indefinite period. Its second stage is one of hypertrophy and ulceration combined. This stage also, as far as external or cutaneous epithelioma is concerned, is slow to advance into the third or destructive stage—that of infiltration and secondary deposit" (COLLIS). "It remains for a very long time local, so that the nearest lymphatic glands often do not become affected until after the lapse of years; and then again the process is for a long time confined to the disease of the lymphatic glands, so that a general outbreak in all parts of the body does not take place until late, and only in rare instances" (VIRCHOW). But admitting all these points in the descriptions of Collis and Virchow, neither of them are individually nor collectively incompatible with *constitutional* or physiological malignancy. For, whether the growth takes its origin from connective tissue, as Virchow, Förster, and Webber hold, or from epidermis or epithelium, as Collis, Hannover, and Thirsh maintain, and grow as they are described to do, the *vital* properties of the growth are really those of malignancy. The anatomical form is no essential part of that specific character.

The essential characteristic anatomical elements of epithelial cancers are—(1.) Cells which bear a close resemblance to flat, paved, scaly, or tessellated epithelium; (2.) the insertion or infiltration of these into the proper structures of the skin or other textures (PAGET). Partaking of these characters, the mass of new growth becomes so condensed by the pressure of rapid growth, often within limited space, that peculiar whorls of growing matter constitute the forms seen on section of microscopic specimens. The explanation of this is to be learned by the combined representations of Paget and Rokitansky, as shown in the following woodcut (Fig. 104).

(d.) MELANOTIC CANCER, OR MELANOSIS, is a "*cancer characterised by the presence of pigment,*" which may be found distributed very generally in every organ and every structure of one and the same body—often becoming first manifest in the soles of the feet or in the axillæ. The lymphatic glands subsequently become affected. Such growths are also found in the ovaries, in the mucous membrane of the bladder and intestines, in the kidneys, supra-renal capsules, heart, and brain. The



Fig. 104.*

Various typical epithelial cancer-cells and their arrangement—A to P (after PAGET and ROKITANSKY). Old, dry, and filmy, B; wrinkled-like fibres, C; very elongated, D (PAGET).

E, F, G, H.—Epithelial cancer-cells, with endogenous development of nuclei—brood cells—magnified 350 diameters (PAGET).

I, K, L, M, N.—Laminated epithelial capsules, whorls, pearls, requiring maceration and separation by *liquor potassæ* to separate and distinguish them (PAGET).

O, P.—Epithelial cancer of the calf of the leg progressing into the fibula: O, Single cells, vexed vesicle, with concentric laminæ; P, Alveolus, containing fresh breeding-cells, one or more of which growing with rapid growth, the others become compressed about it, within the capsule or alveolus, so producing appearances as shown in I, K, L, M, and N (ROKITANSKY).

"*fungus hæmatodes*" has generally been considered related to *melanotic cancer*; and the relationship is more than ever established in the connection which subsists between pigments and the changes which take place in extravasated blood. Frequent hæmorrhages are common in melanotic growths of a cancerous nature, due to extreme dilatation and thinning of the blood-vessels, and the red colour of such tumors must be referred to such kind of vascularity. Pigmentation of such tumors is therefore to be considered as of secondary formation; and in the majority of instances melanotic cancer consists of encephaloid or soft cancer, with the addition of black or brownish pigment. The pigment deposit, *per se*, is not necessarily malignant. The pigment of cancers is readily decomposed by nitric and other acids, while the *spurious melanosis* or *carbon* is not. The true melanic deposit exists with the soft cancer-cells either as an infiltration into them or in the form of isolated granules or small corpuscles. The pigmentation may be seen on microscopic sections to take place along the course of dilated blood-vessels. In an early stage the pigment is first contained in cells, whilst later it is found in free granular masses, through the dissolution and disappearance of the cells which contained it, and a gradual transition may in general be traced from the affected part into the normal structure of tissue, where it will ultimately become developed, and which is freely traversed by blood-vessels. Cells of various forms are to be seen in such tumors which have first a yellow appearance and then a black, according to the amount of pigment deposited in them. The cell-walls subsequently disintegrate, leaving the pigment free. The cells composing the greater part of the structures are such as might belong to uncoloured medullary cancer, so that a small proportion of pigment seems to blacken the whole lump. The pigment is generally in granules or molecules.

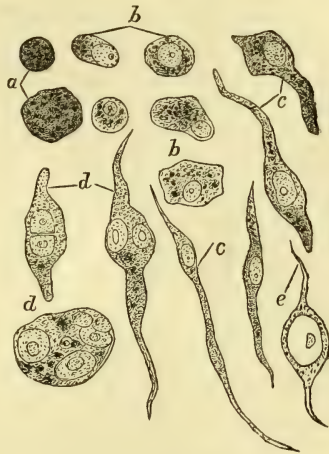


Fig. 105.*

Osteoid Cancer is a "tumor usually commencing in the bones, consisting almost entirely of bone, and followed by similar growths in the glands and viscera."

Villous Cancer is a name given to "cancer in mucous membrane, when covered by a villous growth."

Diagnosis.—Cancerous affections of internal parts may be simulated by many nervous disorders, and also by chronic inflammation of the respective parts, scrofulous enlargements of glands, with deposit of tubercle (*tyroma*); but the long continuance of the symptoms, their gradual augmentation, the severe pain, which admits of no permanent relief, together with the loss of health and slow emaciation of the patient,

* Melanotic cancer from the orbit :—(a.) Two deeply pigmented cells; (b,b.) Five cells containing larger and smaller pigment molecules; (c,c.) Four pigmented fibre-cells; (d,d.) Multinuclear pigmented cells; (e.) Pigmented fibre-cell, with a disintegrated nucleus—350 diameters. (After WEDL, Fig. 107, p. 602.)

at last give a moral conviction that it must be cancerous growth in some organ from which the patient suffers.

The practical questions to solve when a growth is first apparent are—(1.) Is it a mere hypertrophy—a growth from a spot, of the same nature as the structure of the spot, or is it different? (2.) Will it produce a fluid, or germinate elements which will spread and contaminate neighbouring parts? In other words, Is it simple and innocent? or, Is it complex and malignant? To judge of these things, it is necessary to remember that all tumors are composed of a conglomerate mass, made up of numerous little lobules. The first development takes place at a definite point or points. Round the original rudiments of the tumor (that is to be), and which are produced by the germination of a limited group of cells, little new *foci* are formed; which, increasing in size, group themselves round the first, and thus gradually give rise to a continuously progressing enlargement of the original *tuber*. At the peripheral portions of tumors, therefore, the most recently formed portions of the new growth are to be found; while, in the centre of these tumors the elements are disintegrating. Thus the last zone of new growth may extend a considerable distance beyond any line of degeneration or alteration of structure visible to the naked eye: hence, in some tumors, there may exist sources of local recurrence after extirpation. The question of morbid anatomy and malignancy in cancers implies a knowledge of the whole subject of “tumors;” such as are the products of morbid growth, which remain permanent after having obtained a certain size, or which continuously grow. They are broadly classified into—(a.) non-malignant, benign or “good by nature;” and, (b.) malignant; but no hard-and-fast line of separation can be drawn between these two classes. The morbid overgrowths are characterised by internal continuity of constituent tissue-elements, and hence the name “Histoid tumors,” because the majority of such tumors are made up of a single tissue. Sometimes, however, they consist of more tissues than one; and then the component tissues are never isolated, never marked off, as epithelium or muscle is marked off from connective tissue, but the component and varied tissues are in organic continuity with one another. The mode of origin of histoid tumors is the same for all—the same as in foetal development—*i. e.*, the material of which the tumor is built up is *embryonic tissue*, from which by subsequent processes of differentiation, there grow all those varied tissues of a higher type which enter into the composition of tumors. An adequate amount of blood-vessels and blood-supply (*vascularisation*) maintains them in connection with the life of the body, and secures their further independent development and growth. Thus they grow and increase in size sometimes to enormous dimensions, without affecting the integrity of the body, and independent of surrounding parts. The tissues from which tumors originate are the connective tissues and lymphadenoid tissue, of which they are “morbid overgrowths.” Wherever vessels extend, connective tissue extends, and even beyond them; hence it is so extensively distributed throughout the body, that it is impossible to make a cut at any point without exposing connective tissue at numberless parts of the cut surface. Hence also the universality of new morbid overgrowths—as tumors growing from and composed mainly of connective

tissue—less so tumors of cartilage and bone; while, from muscles, nerves, and blood-vessels the development of new growths is comparatively rare. Whence do tumors arise? Migration of cells, followed by their fission, underlies all formative activity. Emigration of such cells as the colourless blood corpuscle brings them into contact with living tissue, and places them at rest, there to undergo fissiparous multiplication—the proximate and efficient cause of the growth of histoid tumors. Such fission in recent living cells may be seen to be the work of a few seconds; but for its activity and continuance, there is required an increase in the amount of protoplasm of the cells—a proportionate increase in the bulk of the original cell, which, with fission of the nucleus, thus makes up the first stage in the process of development of tumors. The next stage is fission of the cell itself—by simple division, gemmation, or endogenous cell formation—the parent cell containing a brood of daughter cells. The embryonic tissue which composes the earliest elements of tumors consists of nucleated particles of naked protoplasm, forming, when in quantity, a very soft and elastic substance, of a pale grey hue. These masses of protoplasm have no limiting membrane or cell-wall, hence they are termed "naked;" but they envelop a round, large, ill-defined nucleus, often only visible after the addition of water or acetic acid. The diameter of each mass is about $\frac{1}{2500}$ of an inch. They are identical with the "granulation" corpuscle, the "plastic exudation" corpuscle, the "proliferation or germination of connective tissue" corpuscles, and the "accumulation of emigrant leucocytes." The subsequent growth of such tissue into a tumor is the history of a differentiating process, by which these elementary embryonic cells are converted into the permanent continuous tissue of the tumor—sometimes of the same tissue throughout; or, more complex, as when they are made up of the elements of several kinds of tissue. The mode of growth and the nature of tumors determine their anatomical relations to the surrounding parts. The malignant growths have no well-defined boundaries, because they increase at the periphery by infiltration of their tissue elements. In tumors of the connective tissue element, where growth mainly takes place at the circumference, such elements of new growth also infiltrate the tissues; and in this respect are similar in their character to cancers, and hence they are often apt to recur in the cicatrix after removal. These include *sarcomata* and some *fibromata*. In the *lipomata*, some *fibromata*, and the *enchondromata*, a capsule of condensed fibrous tissue usually includes the tumor and isolates it from the neighbouring tissue. In such cases growth takes place by continuous multiplication of the cells of which the original growth is composed.

The following are the products of morbid overgrowth which constitute the clinical varieties of histoid tumors, as described by Rindfleisch:—

1. SARCOMATA.—These are mainly composed of the round cell embryonic tissue of granulations, small lymphadenoid tissue, spindle-cell tissue, or densely fibrous connective tissue. As a rule, several of these tissues are in combination; but one of them generally constitutes the main bulk of the growth, the others being present in smaller proportions; and, as results of secondary processes, cartilage, bone, mucous and adipose tissue are also found in them. There are many varieties of sarcomatous tumors. The following are described:—

(a.) *The granulation-like or round-cell sarcoma* is on section of yellowish or reddish colour, homogeneous throughout, like fish-roe, soft, yet elastic. A scanty juice may be scraped from the surface, nearly clear, and containing few cells, small in size, round, and with relatively large, sharply-defined nuclei, carrying nucleoli. These large nuclei are imbedded in very scanty naked protoplasm, as shown by hardening and carmine staining of the tissue. They most commonly grow from membranous expansions of connective tissue, such as periosteum and the coverings of the nerve-centres, as also from skin, mucous and serous membrane, and glands. By development it may proceed to the small-cell, spindle-cell structure, and through that to fibroma—all of which forms are comparatively benign.

(b.) *The lymphadenoid round-cell sarcoma* has its cell elements much less closely knit together than the granulation tissue; and its cut surface readily yields a juice loaded with round cells, having large oval nuclei, feebly refracting light, and carrying each a point-like nucleolus. A delicate inter-cellular network resembling the reticulum of lymphatic glands pervades this form of sarcoma, giving consistence and support to its structure, permitting fluid to collect round the cells, and so favouring their isolation. This form of sarcoma generally grows from the subcutaneous, subfascial, and intermuscular connective tissue of the thigh, or the lymphatic glands; and clinically it is extremely malignant. There are two varieties—one the result of fatty degeneration, the other the result of mucous softening or degeneration, and so named respectively "*lipomatous sarcoma*" and "*myxomatous sarcoma*;" and sometimes these two forms co-exist, such tumors attaining a very great size. They occur chiefly in the lax connective tissue of the extremities, or in the subperiosteal connective tissue. This lymphadenoid round-cell sarcoma is very soft, brain-like, encephaloid, or medullary—by all which names it has been described; and it resembles closely—

(c.) *The alveolar round-cell sarcoma, including melanotic cancer*, sometimes described as *medullary sarcoma*, and regarded as a cancerous degeneration of a sarcoma. It is characterised by the occurrence of large cells, provided with round vesicular nuclei, carrying lustrous nucleoli, grouped in globular masses, and occupying gaps of corresponding size in the continuity of the connective tissue, or amongst spindle-celled sarcomatous texture. This form of sarcoma occurs most frequently in the marrow of bone, in the eyeball, and in the subcutaneous tissue; and a peculiarly malignant variety is characterised by the pigmentation of its cells—"pigmentary cancer"—a very soft and dark coloured tumor—"melanotic cancer."

(d.) *The small-celled spindle-cell sarcoma* resembles in its constituents the spindle-cell elements of recent cicatrices—a short, narrow spindle-cell, containing an oval nucleus, with or without a nucleolus. The protoplasm of the cell is finely granular, and thickest immediately round the nucleus. A limitary membrane to the full grown cells is of uncertain existence; the smaller cells are undoubtedly naked. These constituent spindle-cells are regularly dove-tailed into each other. No interstitial substance exists, beyond an amorphous glutinous cement keeping the cells together, often in bands or bundles of variable thickness, and radiating from numerous centres, so as to be interwoven in all directions. Tumors of this kind

grow by preference from fibrous membranes, fascial sheaths of vessels and nerves, the subcutaneous and submucous connective tissues. Such sarcomata may develop into fibrous connective tissue—*fibroma*. In tumors of the uterus the two forms are frequently combined.

(e.) *The large-celled spindle-cell sarcoma* differs from the preceding four in the disproportionate development of its cells, as compared with all the surrounding elements of its structure. Its spindle-cells may attain a thickness of $\cdot 015$ of a line, and a length so great that, under a magnifying power of 200 diameters, the extreme ends of the cells are separated by three times the diameter of the field. The thickest part of the cell corresponds to the roundly oval nucleus, which carries a lustrous nucleolus. The protoplasm is finely granular and soft in the neighbourhood of the nucleus; elsewhere it becomes homogeneous, firm, tenacious, and rigid, but still with no limiting membrane. Sometimes the cells are stellate. These cells unite to form bands of considerable size, radiating in straight lines in every direction from a common base, or elaborately interwoven with each other. Such tumors grow to a very considerable size, without undergoing further change of structure or composition; and they generally start from fasciæ and membranes, rarely from the interstitial tissue of glandular organs; and they seldom recur after timely extirpation. These fusiform or spindle-shaped cells are the same as "*fibro-plastic cells*," constituting *fibro-plastic* or *fibro-nucleated* tumors. These are rapidly growing tumors; and when they slowly involve the adjacent soft structures, and return after removal, they have received the name of *Recurrent fibroid tumors*.

(f.) *The pigmentary or melanotic sarcoma* usually grows from the choroid coat of the eye, and next in frequency from the skin, the cell elements being infiltrated in both cases with black pigment, and appearing earliest in the epithelial cells lining the vessels. Such tumors are possessed of well-marked malignant characters; and the secondary metastatic deposits are usually also pigmentary, although developed in parts where no physiological pigmentation is known to occur.

(g.) *Fibrous sarcomata, fibroid tumor* or *fibromata*, are represented in structure by the formed tissue of cicatrices, consisting of a fibrous, reddish-white, stiffly-elastic substance, so dense, tough, and even hard, that it creaks or "crys" under the knife. It is made up of extremely fine fibrillæ. These fibrillæ are composed of a gelatinous material, and inclose fine cells between, usually small, roundly oval, and carrying lustrous nuclei. In addition, bands of spindle-shaped cells traverse fibroid tumors in every direction; and round deposits of embryonic cells lie imbedded here and there amongst the continuity of the fibrous bundles. These are the transitional structures from which the fibres are developed. They are the most benign of all morbid growths, and the uterus is their favourite seat. They are circumscribed, and do not invade surrounding structures. When such tumors contain cysts, they are named *fibro-cystic*; when they contain earthy matter, *fibro-calcareous*; when they grow from bone, and are partly ossified, they constitute the non-malignant form of disease known as *osteo-sarcoma*; when they contain involuntary muscle, as when growing in the uterus, they have been called *fibro-muscular*; when they contain fat, *fibro-fatty*.

(h.) *The cavernous tumor* is a tumor of erectile tissue, like that of the *corpora cavernosa penis*. It consists of a network of white glistening trabeculæ of connective tissue, in whose white meshes the blood is contained as in a sponge. It constitutes the *vascular tumor*, and also *nævous*. It originates in a fibroid degeneration of the capillary portion of the blood-vessels, while the arteries show an enormous thickening of their walls, a tortuous course, and a capacity for dilatation such as no healthy vessels ever exhibit. Such tumors grow by preference in the adipose tissue, and are not unfrequently multiple. They are occasionally malignant; but such malignity usually depends on a manifest complication with melanotic sarcoma, whose nodular foci have become developed in the trabeculæ of the cavernous tumor.

2. **LIPOMATA, OR FATTY TUMORS.**—These are principally made up of the elements of adipose tissue—the fat cells—and agree in structure with the normal fat cells of the human body and their relation to the septa of connective tissue. Such tumors are made up of lobules of a globular shape. They are examples of central growth, and are usually connected with the neighbouring parts at one point only by a stout vascular pedicle, while its external surface is limited by a large-meshed connective-tissue smooth capsule. They originate in the subcutaneous, submucous, sub-synovial, subserous, subfascial, intermuscular, and intraorbital connective tissue. At first they grow very slowly, then faster, and may attain a very considerable size—as large as a man's head, or larger. In such large tumors chronic inflammatory changes and fibroid transformation and mucous softening are apt to take place, as well as calcification of the fibrous matrix. The fatty tumors are decidedly benign, and once thoroughly extirpated never recur.

3. **ENCHONDROMA, OR CARTILAGINOUS TUMORS.**—They occur where no cartilage normally exists, and present the peculiar elastic hardness, the milk-white colour and translucency of their layers which are characters of cartilage. All the minute anatomical forms of cartilage tissue may co-exist in an enchondroma; but as a rule hyaline cartilage predominates. The capsules are not always distinct; and, when entirely absent, are evidence that the matrix is beginning to soften. They generally grow from bone, the medulla being the source of the new growth, or from common connective tissue. The protoplasm is of variable forms, such as stellate, and contains a nucleus. The tumor is usually made up of small lobules, not larger than pease, and hence capable of nourishment from their periphery. The formation of vessels and of true bone has been seen in enchondroma. In tumors of small size blood-vessels are abundant round the lobules, and in those of large size, round the periphery; but in the interior of the latter the blood-vessels are compressed by weight and growth, even to obliteration, so that degeneration changes are apt to be set up. Calcified patches are common. These tumors are frequently complicated with alveolar or soft sarcoma, and as such are malignant—otherwise, they are not so. But pure enchondroma are also liable to recur; gradual extension of the growth along the lymphatic takes place, metastasis to the nearest lymphatic glands, and even to internal organs, have been observed. The lung is the favoured site for such secondary enchondromata, but they are rare, and of small size. Four-fifths of all

such tumors grow from the osseous system, more particularly the shafts of the long tubular bones, either from the medulla or the periosteum, especially of the bones of fingers and toes, where they are often multiple. They occur most frequently in early life; and, next to the bones, they grow in the parotid gland and testicle. They usually grow slowly, except those which proceed from the medulla of bone. These are rapid in growth, soft in texture, generally malignant, and not limited by a fibrous capsule.

4. MYXOMATA, OR MUCOUS TUMORS.—These generally originate by a secondary metamorphosis of other connective-tissue substances; and tumors exclusively made up of "mucous tissue" are not common. They consist of a basis-substance containing mucus, or of one which has undergone mucous softening. They are colourless, transparent, and soft, like a jelly; and if cells predominate, the tumor is white, reddish-white, or pale grey colour. Their juice is a tenacious mucilaginous fluid, containing the cell-elements of the tumor. They are lobulated, and but sparsely provided with capillary vessels. They form nodular swellings, which may increase rapidly in size (although growth is usually slow), and this property, with their great softness, causes them to be mistaken for soft cancer. The favourite sites of myxomata are the subcutaneous areolar tissue of the thighs and back, and the external genitals in the female; the interlobular connective tissue of the neck and face; and next, the medullary tissue of bones and connective tissue of the nervous system; they also grow from the placenta as *hydatid degeneration* of that organ. In the nervous system the tumor is often multiple. In the submucous tissue of the nose they constitute one form of nasal polypus. They are benign, and do not recur after complete extirpation. They occur generally late in life.

5. OSTEOMATA, OR BONY TUMORS.—They occur usually in the form of *exostosis*, and vary as to hardness, being ivory-like or cancellated. They are benign, and their growth is slow. They are often hereditary and multiple, usually then occurring in early life.

6. MYOMATA.—These include all tumors mainly composed of true muscular fibre; and in them transitional forms between striped and smooth muscular fibre are often to be met with. To display the muscular tissue it is necessary to dissolve away connective tissue and fibres by maceration in dilute nitric acid, which will thus leave the muscular fibres isolated. The uterus is a frequent seat of these tumors. I have seen one as a large pedunculated tumor or polypus growing from the inner wall of the small intestines—ultimately inducing invagination of the bowels and death, by intestinal obstruction. They also occur in the prostate gland, œsophagus, and stomach.

7. NEUROMATA.—These are of two kinds—one represented by the well-known "painful subcutaneous nervous tubercle." This is essentially a fibrous tissue tumor, growing from the neurilemma of a superficial nerve. The nerve-fibres become expanded over them as they increase in size, and are the source of great pain. The other form is that of a tumor consisting entirely of nerve tissue, and it is exceedingly rare. Such tumors always originate from nerve-fibres, either cranial or spinal, and resemble them in structure, consisting of tubular nerve-fibres, with intertubular connective tissue, and a few grey nerve-fibres. They grow slowly, and never enlarge

beyond a small nodule, which is generally solitary. Their most common site is at the divided end of a nerve, as in stumps after amputation; and are then often in connection with the tissue of the cicatrix—an involvement which is often the source of much pain. Clinically, however, they are benign tumors.

8. COMPOUND HISTOID TUMORS.—These are such as have a number of different tissues entering into their composition; and, regarding these, it is to be observed that their prognosis is less favourable than that of simple tumors of any one of the several species of tissue which have been described. They usually recur as sarcomata after extirpation.

9. LYMPHATIC TUMORS.—These are made up of the basis-substance of lymphatic tissue—now called *adenoid* tissue. This tissue is known to be very widely diffused. It enters into the composition of the general lymphatic and mesenteric glands—the Malpighian corpuscles of the spleen and the glands of Peyer; the solitary glands of the large and small intestines, and the follicles of the pharynx and tonsils. It forms a definite plexus of tissue (perivascular canals) round minute blood-vessels, like those of the pia mater, and amongst the connective tissue round the bronchial tubes. It exists immediately beneath the epithelium of the pleuræ, the peritoneum, the mucous membrane of the alimentary canal, and in the medulla of bone. It is one of the special varieties of connective tissue—the *retiform* of Sharpey, the reticular or cytogenous tissue of Kölliker, the *adenoid* of His. Within the meshes of its reticulum lymph-cells are inclosed, and these constitute the greater part of adenoid tumors, which are morbid overgrowths of lymphatic tissue. The adenoid tumors—*lymphomata*—are generally the result of some specific irritation.

10. GLANDULAR TUMORS.—These are also named *adenocoeles* or *adenomata*. They are tumors growing in or near a gland, and more or less perfectly resembling it in structure. They consist of epithelial cells, whose arrangement suggests the epithelial lining of the tubular or acinous glands, the cells being for the most part grouped round a central axis, as though they included a tubular lumen, but which is generally plugged with mucous or colloid matter, and having no outlet. It is an unlimited reproduction of glandular tubuli. Thus a tumor grows extravagantly rich in cells, and very scantily provided with vascular connective tissue. Thus it carries within itself the elements of destruction. They always originate from a gland structure, especially the liver, the mammary gland, sebaceous glands, pineal gland, ovaries, and mucous follicles of the rectum, and the gland structures of mucous membranes generally, as in nose, pharynx, stomach, intestines, vagina, uterus, where they form *mucous polypi*. *Adenomata* form globular, sharply-circumscribed nodules, which replace a comparatively small portion of the gland from which they spring. Each nodule increases by central proliferation, and tends to push aside, and not to infiltrate the neighbouring parts. *Adenomata* of the liver are encapsuled. Clinically, they are believed to be benign tumors; but they are very apt to be confounded with growths having malignant properties. Much has still to be learned regarding them. A simple adenoma may become cancerous. It may also become caseous from fatty degeneration of the inclosed epithelium. The gland tubes may also dilate into cysts, and mucoid softening is common.

While simple tumors are *overgrowths*, in addition to being *outgrowths* or new growths, they imply a continuous reproduction of one or of several tissues taking place to an excessive extent in any limited territory, ultimately giving rise to a tumor by coalescence and growth. But to cancers or malignant tumors there is *something more specific superadded*. Their intimate structure is not like that of any fully developed natural part of the body, nor like that found in a natural process of repair or of degeneration.

Prognosis.—Cancer, though long latent, and its course slow, pursues its destructive progress unimpeded. It is only in rare instances that amendment or a return to health awaits the patient, who ultimately falls an inevitable victim to the disease. The mortality from cancers goes on steadily increasing with each successive decade until the eightieth year (WALSHE).

Treatment.—No remedy has yet been found which can in any degree be considered curative of the constitutional state associated with cancer, and the efforts of the practitioner are consequently limited to relieving symptoms, and to the adoption of such palliative measures as may prolong life. It has generally been believed that to remove a cancerous growth where it is practicable must, on theoretical grounds merely, be attended with as much benefit to the constitutional disease as would attend the removal of a leg for acute rheumatism chiefly expressed in the knee-joint. The statistics of cancer show, so far as they go, and as Dr. Walshe long ago showed, that "excision of a cancerous tumor seems to awaken a dormant force. Cancers spring up in all directions, and enlarge with a power of vegetation almost incredible." Nevertheless, there are good reasons for removing cancers in some cases, especially mammary cancers and others accessible to the knife. The greatest measure of good may be done, as Sir James Paget has clearly shown, "by making a careful selection of cases fit for operation, and rejecting all the rest as unsuited for operation" (*Med. Times and Gazette*, September 27, 1872, p. 319). With regard to excision of the breast for cancer, the main objection in the first instance is, that even of cases selected with some care, 10 per cent. (PAGET) to 16 per cent. (LEBERT) die of pyæmia, or erysipelas, or tetanus, or secondary hæmorrhage, or some calamity subsequent to the operation. On the whole, however, taking the results of some hundreds of cases, it is certain that the average duration of life of those operated on is not less than those in whom the disease runs its course. In well-selected cases it will be found always greater. A recent tabulation of hospital and private cases by Sir James Paget showed that 85 cases operated on lived an average of 55·6 months; and 62 cases not operated on lived an average of three months. It has also been said that the recurrent disease is more painful than the primary one; but in very many cases Sir James Paget has found that the recurrent disease was much less severe than the continued disease. Considering, therefore, the danger of the operation, and the fact that in every case a recurrence of the disease may be expected, is it reasonable to submit a patient to the risk of dying from the primary operation for the sake of that interval of health between the operation and the recurrence of the disease, for the good probability of adding a year to life, and for the chance of having

a less severe disease? The average length of the interval before recurrence is little more than *thirteen* months; more than half return within twelve months; and two-fifths return within six months. The extremes between which the average is drawn are very wide. In some cases the return may be within *three* months; in others not for *ten, twelve*, or more years. It is of great importance to determine in what cases the risk of life is greater; and in what cases the probability of a speedy return of the disease is greater than the average. The old after sixty; the very large-breasted in cases of mammary cancer; the fat and plethoric; the cachectic; the overfed on animal food; the drunkards; the gouty; the habitually bronchitic; the albuminuric; the very dejected—not timid merely; in short, those with any organic disease of the internal organs—all such cases are “doubly hazardous” to interfere with by operation (PAGET).

The probability of rapid recurrence is great in acute cancers—*i. e.*, all those that are rapid in their progress—and in those which are observed to increase very quickly before the operation. Great pain, however, is often saved by performing the operation even under such circumstances. In illustration, Sir James Paget records the case of a lady “whose breast he removed when she was five months advanced in pregnancy. She recovered well from the operation, and the benefit procured by its performance was very great. She went to her full term, bore her child, and was able to suckle it for a year before she died, with her most anxious wish fulfilled in comparative comfort.” Another condition unfavourable to operation is a brawny skin, with firm œdema and wide-open hair follicles, or wide adhesion of skin, or in which the skin is cancerous, or where there are scattered tubercles of cancer in the glands and skin; or where there is considerable affection of the lymph-glands in the vicinity, and especially *numerous* diseased glands. A moderate amount of lymphatic disease is not a serious obstacle to an operation (Sir JAMES PAGET). In very chronic cases the operation is needless—where the *breast* is small, shrivelled, knotty, and sunk down on the pectoral muscle. By thus selecting with care, on the one hand, cases fit for operation, and refusing to interfere in those cases in which the operation would be attended with more than a proper share of danger, Sir James Paget believes that the life of a large number of those who suffer from cancer may be considerably prolonged. In whatever part the disease may be situated, one great rule is to endeavour to restore the healthy functions of that part, and to alleviate by opiates, chloroform, or chlorodyne internally, the distressing pains the patient endures. These remedies are for a time successful, but make no impression on the disease, which silently proceeds, and the patient finally limits himself altogether to opiates. The quantity of opium or other narcotic known to have been taken in such cases is something enormous—five, ten, fifteen, or twenty grains of opium at a dose, or a proportionate quantity of *hyoscyamos* or of *conium*, three, four, or more times in the twenty-four hours. But although these large doses have occasionally been given, yet it may be questioned whether they are not more hurtful than beneficial; for usually they produce headache, delirium, loss of appetite, and narcotism, so that the patient is only the more rapidly exhausted. In general, therefore, the patient does better under moderate doses of opiates, as one or two grains, or its equivalent of

morphine or other narcotic, every four, six, or eight hours, than when more excessive doses are given—a larger dose producing headache and much cerebral disturbance, without in any sensible degree mitigating the sufferings. When the disease is seated in the colon or intestine, the tumor is in general difficult to make out. One loop of intestine may open into another; and death by hæmorrhage may terminate the case. The symptoms vary greatly according to the position of the cancer and the part of the intestinal canal affected. Great sickness and vomiting generally attend cancer in the duodenum. When the stomach is so irritable that it rejects everything, it is our duty to support the patient by nutritive injections, as of strong broth, egg-flip, sago, or other fluid substances. It has been attempted to impart strength to the patient by means of milk baths, or baths of strong broths; but the skin has not generally sufficient power of absorption, and it has been found that the heat of the bath has exhausted the patient in a far greater ratio than its nutriment supported him. As a general principle, diet has little or no influence over the course of the disease when once established, except it may be that total abstinence from *nitrogenous* food may starve out the growth, and most likely also the patient; so that whatever agrees with the patient's digestion may be safely indulged in. Preventive treatment must be directed to the infant life of those who are hereditarily predisposed.

COLLOID—*Syn.*, COLLOID CANCER, ALVEOLAR CANCER.

Definition.—*A new growth, a great part of which is formed of transparent or gelatinous substance.*

Pathology.—*Colloid, Colloid Cancer, or Alveolar Cancer*, is sometimes also called *gelatinous* or *gum-cancer*. In this form of disease the meshes of the new growth are filled up by a glue-like or gelatinous substance, like half-dissolved gum-arabic. It is “*a new growth, a great part of which is formed of transparent or gelatinous substance.*” The fibrous stroma are arranged in the form of alveolar spaces, the fibroid tissue being extremely delicate and transparent, and the spaces are occupied by the glue-like matter in greater or less quantity. These growths are not now regarded (and their position in the nomenclature as a substantive disease would also indicate that they are not to be regarded) as cancers. They are limited growths, and not infiltrations; but they expand or grow to a very great extent, as in a case related by Dr. Ballard, where the greater part of the parietal, and much of the visceral peritoneum, were involved in colloid cysts. Six quarts of colloid matter were removed from the peritoneal cavity after death; and Sir James Paget remembers a case where the colloid matter was still greater. He acknowledges that “it seems difficult to believe that such a structure [colloid] can have any close affinity with the cancers already described;” but he considers it has, as to its clinical history, all the other distinctive features of cancer, namely:—(1.) Its seats of election are those where medullary cancers are apt to occur; (2.) Sometimes it infiltrates, replaces, and supersedes the natural tissues of the part; (3.) It is prone to extend and repeat itself in lymphatic glands; (4.) It is often associated with other forms of cancer in the same mass, or in different tumors in the

same person; (5.) It recurs after removal; (6.) It is derived hereditarily from a parent having scirrhus cancer, and a parent having colloid may have children with medullary cancer. Mr. Collis regards colloid as structurally and clinically removed from cancer. In structure they are aggregations of the smallest lymph-cells, or of mucous-tissue like the umbilical cord, and held together by the finest connective material, and never of large cells like cancer. Nevertheless, such tumors sometimes recur after removal, and, in other respects, manifest the worst features of malignancy. It generally corresponds with the history of scirrhus and medullary cancers; and there can be no doubt that the clinical course of the disease often resembles that of cancer. The characteristics of its structure consist of a very delicate translucent membranous stroma, out of the spaces of which there comes a glue-like substance. Its growth is rapid, and it may reach an enormous bulk. The spaces or alveolar loculi vary from a very minute size to that of a pea. The contained substance is of a yellowish or greenish-yellow colour, but yields no gelatine on boiling. It is "a clear, flickering, or viscid substance, like soft gelatine." The most common places where colloid cancers form are the *stomach*, the *ovaries*, the *bones*, the *kidneys*, the *intestinal canal*, *uterus*, *mammary gland*, *peritoneum*, and *spleen*, and they may be combined with the *hard* or *soft* cancers. The reader is referred to Sir James Paget's classic work for an account of the various combinations of colloid cysts with cancers of all kinds.

LUPUS.

LATIN EQ., *Lupus*; FRENCH EQ., *Lupus*; GERMAN EQ., *Lupus*—Syn., *Fressende Flechte*; ITALIAN EQ., *Lupus*.

Definition.—*A spreading inflammation of the skin or mucous membrane, usually of the face, characterised by infective cell-growth, tending to destructive ulceration, but only by accident involving textures deeper than the skin.*

Pathology.—*Lupus* has been considered as allied to malignant disease; but its most evident affinities are with scrofula, especially to that state which produces diseases of the psoriasis type, and to the conditions of feeble circulation which favour the occurrence of chilblains. It depends upon a *neoplasm*—upon a development of new growth in the tissue of the dermis, classed by Virchow among "*granulation growths*," the elements of which have been already described, under the head of *gummata*, in syphilis. But it is not here implied that *lupus* has necessarily any connection with *syphilis*. It is wholly independent of syphilitic taint. The nodules of *lupus*, which indicate its existence, are either isolated or diffused, causing considerable thickening of the skin; and the new growth advances from the superficial layers to the deeper parts. Crusts of epidermis next form over the lesions, under which the new growth begins to soften and ulcerate. But sometimes the new growth subsides, the cell elements undergo fatty degeneration, followed by absorption, when a deep cicatricial-like loss of substance remains.

Symptoms.—Two varieties of the disease have been distinguished by the College of Physicians, namely:—(a.) *Chronic lupus*; (b.) *Lupus*

exedens, defined as a variety "characterised by the rapidity, depth, and extent of the ulceration, and by appearing in rare cases on other parts than on the face." The most frequent seat of *lupus* is upon the face, especially the nose; but in the *exedens* variety it may attack other regions, such as the neck, shoulders, chest, and extremities, especially over the joints. The disease first appears as small, painless, brownish-red specks; and although they feel quite hard nodules, they are very easily made to bleed, and are very easily penetrated. In this form of lesion the disease may remain for years, making no advance. When the disease is about to progress, the nodules multiply and grow larger, their surface becomes tense, shining, and covered with exfoliated scales of epidermis (*lupus exfoliatus* of Hebra). As hardness subsides, the lesion also subsides beneath the level of the skin, and the place is converted into a white, hard, shining cicatrix, without any ulceration having occurred. But in other cases, where new nodules continue forming, the surrounding skin becomes hyperæmic, red, and shining; a superficial erosion forms under a scab, which becomes thicker and broader from the accretion of fresh matter at the base. The most destructive form begins at the tip of the nose and *alæ nasi*. Thus, either ulceration or interstitial absorption (the cuticle remaining intact) is invariably present in *lupus*, and constitutes the most marked feature of the complaint (PARKES). The second variety is sometimes known as *lupus hypertrophicus*, and attacks other parts of the body besides the face.

Diagnosis.—The disease is rare before the tenth year of life, and still more rare after the fortieth; being most common between the ages of ten and twenty. It is less frequent among the rich than among the poorer classes (NIEMEYER). *Lupus* may be confounded with *syphilitic tubercles*, *true leprosy*, or *elephantiasis*, some forms of *acne*, of *cancer* of the skin, and *impetigo*. It is to be distinguished from syphilis by the history, by the absence of induration and of coppery hue before ulceration, and the absence of syphilitic characters after ulceration. It is never pustular.

The **Prognosis** is always unfavourable to the extent of local destruction; and there is continual tendency to relapse and renewal of new growth, especially if the cicatrices remain soft, bluish, and surrounded by soft tumors, which yield to the fingers, and if the tumors do not disappear after cicatrization (BIETT).

Treatment.—The new formation must be removed or destroyed; but opinions differ as to how this is best effected. Mr Jonathan Hutchinson prefers Volkman's erosion method. Cazenave recommends chloride of zinc, arsenical paste, or the powder of "Côme," Vienna paste (equal parts of chalk and quicklime), and other powerful applications of the like kind. Hebra also recommends cauterisation, and employs usually strong nitric acid over the unulcerated, and nitrate of silver over the ulcerated surface. It is a remarkable fact in the history of *lupus*, affirmed by Hebra, that the *lupus* disappears during acute febrile diseases, and especially during typhus. It seems to recur with convalescence. (See Dr. A. T. THOMSON on *Diseases of the Skin*, edited by Dr. PARKES, where numerous interesting cases are detailed, p. 310.)

The medical treatment has in view the prevention of the formation of more similar deposits in the substance of the skin by improving the general health. *Cod-liver oil*, *phosphorus*, *quinine*, and *bitter vegetable tonics*,

good food, bracing air, and the judicious use of stimulants, are all useful in this respect. *Iodides* and preparations of *mercury* are to be avoided; but *arsenic* may be given with advantage (HUTCHINSON). On the other hand, Dr. M'Call Anderson recommends the use of *iodide* of starch.* In using it, care must be taken, first, that the cases are really undoubted cases of *lupus erythematodes* and not *lupus vulgaris*; and second, that the medicine is freshly prepared, and in accordance with the directions above mentioned. Dr. Balmanno Squire has devised an improvement upon the treatment of lupus by pricking the skin at short distances with a pointed instrument, as long since advocated by Dubini, of Milan, and subsequently by Volkman, of Halle. By means of a many-bladed instrument which Dr. Balmanno Squire has contrived, and which has been constructed by Messrs. Weiss, of London, he cuts the skin into thin strips. The Dubini-Volkman method (known as punctiform scarification) has been highly successful. Dr. Squire's method (termed multiple linear scarification) has proved still more successful. It has been tested and commended in this country by Dr. Walter Smith, of Dublin, and Mr. Malcolm Morris, of London; while in Paris, at the Hôpital St. Louis, Dr. Vidal and his assistants treat every Wednesday as many as from forty to fifty patients by this method. The advantages are the same as those which attend punctiform scarification—namely, the setting up of a traumatic inflammation, under which the disease heals; but the superiority claimed for treatment by closely and regularly spaced incisions is a more rapid and at the same time a more complete cure of the disease than can be effected by means of irregularly distributed punctures. Dr. Vidal had observed, in sections of the skin thus healed, the gradual conversion of the lupus cells into fibrous tissue. The advantages of treatment of the disease by the excitement of traumatic inflammation as compared with other means of treatment, are described as being less painful, as causing less loss of substance, and as leaving less permanent trace of its performance than the customary methods of local treatment, namely, the application of caustics or semi-caustics (*Brit. Med. Journ.*, August 23, 1879, p. 293).

TRUE LEPROSY—*Syn.*, ELEPHANTIASIS GRÆCORUM.

LATIN EQ., *Lepre Veræ*—*Syn.*, *Elephantiasis Græcorum*; FRENCH EQ., *Lèpre Vraie*—*Syn.*, *Eléphantiasis des Grecs*; GERMAN EQ., *Aussatz*—*Syn.*, *Elephantiasi Græcorum*; ITALIAN EQ., *Lepra*—*Syn.*, *Elefantiasi deis Greci*.

Definition.—*A constitutional, non-contagious, hereditary affection, essentially chronic in its nature, expressing itself mainly by shining tubercles of different*

* The following is the formula for its preparation: R Iodi, gr. xxiv; amyli, ʒj. Triturate the iodine with a little water, gradually adding the starch and continuing the trituration till the compound assumes a uniform blue colour, so deep as to approach black. The iodide should be dried with a heat so gentle as to run no risk of driving off the iodine, and it ought to be kept in a well-stoppered bottle. On no account should spirit be used in its preparation instead of water. The dose is a heaped-up teaspoonful in a draught of water or water-gruel thrice daily; but it may be safely increased even up to an ounce in some cases.

sizes, of a dusky red or livid colour, on the face, the ears (and often the extremities)—the skin being thickened, wrinkled, rough, unctuous, divested of hair, and the perspiration highly offensive—in which there is loss of feeling, or other disorder of innervation, and a tendency to ulceration or death of the affected parts. The eyes in extreme cases are fierce and staring, and the voice is hoarse and nasal.

Pathology.—(a.) *The History* of leprosy is as ancient as the records of man in the countries where it seems to have been indigenous. More than 3360 years ago (B. C. 1490), Moses described leprosy, (Leviticus, xiii.)* More than 1800 years ago it was described by Aretæus in clear and unmistakable language; and, from the earliest records to the present day, the distinctive characters of the disease have continued, and have been transmitted unchanged from generation to generation. In the Middle Ages it prevailed almost epidemically upon the continent of Europe; and various minute descriptions of leprosy are left us in the writings of different European physicians and surgeons of those days, who had opportunities of studying the disease during its actual prevalence. The details given by these observers (as shown by the late Sir James Simpson, in a series of admirable papers in the fifty-seventh volume of the *Edin. Med. and Surg. Journal*) are similar in all essential parts; and the symptoms and cause of the disease described by them are the same as those described by Aretæus. The leprosy of Europe, during the Middle Ages, was undoubtedly the elephantiasis of the Greeks. It is one of those diseases which history has shown to change its geographical stations to such an extent as to have made inroads upon whole districts and regions of the world where it was formerly unknown, leaving now untouched the localities which, in olden times, suffered most severely from the disease. It is now almost entirely unknown as a native endemic disease on any part of the continent of Europe, except in some provinces of a few countries; and yet, from the tenth to the sixteenth century, it prevailed in nearly every district of this great continent. In the Scandinavian peninsula it still lingers. In a portion of Norway it appears to be as rife as it has ever been, whereas the conterminous country of Sweden is comparatively

* Directions to Moses and Aaron for the diagnosis of leprosy:—"When a man shall have in the skin of his flesh a rising (swelling), a scab, or bright spot, and it be in the skin of his flesh like the plague (lesion) of leprosy"—he is to be the subject of observation; "and when the hair in the plague (lesion) is turned white, and the lesion be deeper than the skin of his flesh, it is a lesion of leprosy. If the bright spot be white in the skin of his flesh, and in sight be not deeper than the skin, and the hair thereof be not turned white," he is to be the subject of observation for seven days. If at the end of that time, there be no change, he is to be shut up for seven more days. If the lesion be then dark and not spread in the skin, it is but a scab. "But if the scab spread much abroad in the skin, it is a leprosy. If the rising (swelling) be white in the skin, and it have turned the hair white, and there be quick raw flesh in the rising, it is an old leprosy in the skin of his flesh. And when raw flesh appeareth, it is a leprosy. Also in the skin of the flesh, when a boil has healed, and in its place there be a white rising, or a bright spot white and somewhat reddish, and it be in sight lower than the skin, and the hair thereof be turned white, it is a lesion of leprosy broken out of the boil" (verses 1-20, and so on, describing also the cleansing or the garments of lepers). The hereditary character of leprosy is also referred to in the case of Gehazi, when he went out from the presence of his master Elisha, who said to him that "the leprosy of Naaman shall cleave unto thee and unto thy seed for ever. And he went out from his presence a leper as white as snow" (2 Kings v. 27).

exempt. In 1858 the number of known lepers in Norway was 2,087 (*Report of International Congress of 1862*). Several districts in the south of Europe—in Spain, Portugal, and Italy—are still affected with leprosy to a considerable extent. These exceptions to its almost complete departure from Europe, have some importance in pointing to the probable causes that favour the continued development and persistence of the disease.

Happily, in Great Britain the disease is now unknown; but it prevailed to a great extent during the Middle Ages, as shown by the records of those institutions, well known as *Lazar-houses* or Leper Hospitals, which were instituted for the reception and seclusion of the infected, as well as to restrain the ravages of a disease believed to be “most powerful in dragging men to death, disgusting to the sight, and in all respects terrible, like the beast of the same name” (ARETÆUS).

The most clear and distinct accounts of the disease in modern times have been given by Dr. Bateman, of London, and Dr. Schedel, in Paris. Sir James Simpson has adduced the clearest proof that the leprosy of England and Scotland was of the same specific nature as the leprosy of the Greeks; and in one instance, that of the Leper Hospital of Shirbon, one of the largest in England—founded in 1181 by Hugh Pudsay, “the jollie Bishop of Durham”—the inmates are directly designated “*Elephantuosi*,” instead of being termed “*Leprosi*” (SIMPSON). Long after the disease had left the more southern parts of the British islands, it continued to prevail in the Shetlands, down to 1742, when the infected were kept in the Island of Papa (contracted for Papastown), as shown by a document drawn up for Sir John Pringle, and bequeathed by him in MS. to the College of Physicians of Edinburgh. Afterwards, as late as 1778, and lastly, as late as 1798, patients with leprosy were sent from the Shetlands to the Edinburgh Infirmary for the cure of their disease. These cases all presented the same unequivocal signs of ancient leprosy; and in the last, if not the very last, Scotch leper, in a member of a Shetland family, the disease was a family inheritance, and decidedly marked by the true and genuine characteristics of the elephantiasis of the Greeks. No change has taken place in the characteristic features of leprosy. It is still seen among the hideous victims of the disease “on the plains of Egypt and Palestine, in the summer climates of Madeira and the Crimea, on the cold hills and valleys of Iceland and Norway, among the cities of British Hindostan, and the villages of Central Africa. Its existence may still be traced over those vast regions of Europe and Asia inhabited by the Mongolian tribes, and the wandering life of the northern nomadic Tartars does not escape its ravages. It is common also among the Malays of Java and the Dyaks of Sumatra and the Indian Archipelago. It is frequent in China; and the traveller in Burmah may see, in the neighbourhood of Ava, one of those unclean villages of lepers which the Burman turns from his path to avoid, lest its very air should blow upon him” (PARKES).

The geographical distribution of true leprosy is now as follows:—

1. *British North America*, in a small part of the province of New Brunswick, and of comparatively recent date—since 1815.

2. *In all British West India Colonies*, to a greater or less extent. In Jamaica it has been long known; and also in Barbadoes, where it is very common.

3. *In South America*, especially in British Guiana, it is very common, and has excited much attention for several years. It is met with throughout the whole extent of Guiana—Dutch, French, and British—while in Dutch Guiana it is said to have increased.

4. *Africa*.—Amongst the Hottentots and half castes, it has been long prevalent at the Cape of Good Hope. It is met with at Sierra Leone.

5. *In Palestine and Syria*, it mostly prevails at Jerusalem, where it is confined entirely to the Mohammedans. It is known in the consular district of *Damascus*, chiefly among the mountain classes of the poorer peasantry—both Moslems and Christians. The districts most subject to it are the highlands and the tablelands, and very rarely on the sea-coast. It is occasionally also met with in the villages adjacent to *Aleppo*, but not in the city itself.

6. *Islands in the Archipelago*.—In *Rhodes* the disease is chiefly confined to the Greek population. It has been rare in *Smyrna* for twenty years previous to 1868; but has been known in *Scio* from time immemorial. It is endemic in *Mytilene*, and prevails extensively in *Samos*. In *Crete* it has existed for centuries; and in 1866 or '67 there were no fewer than 1,000 lepers out of an island population of 250,000. Leprosy has long existed in the *Ionian Islands*; and it appears principally in the villages of the mountainous parts of *Corfu*, rarely in the towns and plains.

7. *Turkey in Europe*.—On the sea-coast of the district of *Salonica* and the adjacent provinces of *Thessaly* and *Macedonia* leprosy is endemic.

8. *Persia*.—It exists certainly in the north-west provinces, and probably in other districts.

9. *China*.—Leprosy prevails extensively throughout the Chinese empire, especially in the southern provinces. It is very common in and around *Canton* and *Shanghai*; and a large number of lepers flock from the mainland to the island of *Macao*, to be under the kind treatment of the Portuguese authorities.

10. *Japan*.—The disease is believed to exist in this country, but nothing certain is known.

11. *Australia*.—In and round *Ballarat*, *Castlemaine*, and *Beechworth*, cases of tubercular leprosy have been met with among the Chinese emigrants to the gold diggings.

12. *New Zealand*.—The disease described by the late Dr. Thomson, of the 58th Regiment, styled by the natives *Ngerengere*, is a form of leprosy, dying out as civilisation advances.

13. *Mauritius*.—In this island the disease is very common, and occurs among all classes. It is also remarkably prevalent in the chain of islands lying between the *Cape of Good Hope* and *Ceylon*, including *Madagascar* and the adjacent islands of *St. Marie*, the French colony of *Bourbon* or *Reunion*, and the group of *Seychelles*, where there is a leper establishment.

14. *India* has been for all ages, and continues to be, the greatest seat of leprosy in the world. No province of the empire, from *Point de Galle* to *Peshawer*, or from the *Indus* to the *Straits of Malacca*, seems exempt. (a.) *Ceylon*.—Among the lower orders of the natives it is not uncommon, and it has occasionally been seen among Europeans and the better classes. It is said to have increased during the fifteen years previous to 1868, chiefly from the influx of Malabars. (b.) *In the Bombay Presidency, including Aden*, leprosy is well known, particularly in the *concan* south and east of Bombay. In some villages lepers are in the proportion of 1 to 80 or 100 of the inhabitants. (c.) *In the Madras Presidency* leprosy is of frequent occurrence, more especially in the larger towns on the eastern and western coasts, particularly the latter. (d.) *In the Bengal Presidency*, and generally throughout India, especially in the lower provinces and districts bordering on the sea, leprosy prevails, and is held in great dread by Europeans and natives.

A most trustworthy account of this disease has been given by Dr. Parkes, in Dr. Thomson's treatise on *Diseases of the Skin*, on the authority of Schilling, Robinson, Kinnis, Gibert, Simpson, Bielt, Cazenave, Pruner, Danielssen, and Boeck (the most recent authorities up to 1848 on this disease). Since then (1867), an exhaustive report has been published by the College of Physicians of London, at the request of the Secretary of State for the Colonies, and the account of leprosy given in this work is almost a transcript of the record of the College, supplemented from the records of Dr. Parkes. The great extent to which leprosy prevails in many distant dependencies of the British empire, as well as a report that the disease was increasing, and the inevitable destitution and mendicancy that attend its existence among a population, rendered a thorough investigation a matter of special duty on the part of the Government of this country. In many regions of India the lepers may be counted by thousands; and in several of the West India Colonies their number is very large. The disease is known to be present also in the colonial dependencies of many European countries, more especially those of France, Spain, Portugal, and Holland. A few rare cases of indigenous origin have been met with in the British Islands during the present century. Reference is made to the case of a youth from one of the Hebrides, admitted to the Edinburgh Infirmary (*Edin. Med. Journ.*, July, 1855); to a case in Guy's Hospital, described by Dr. Gull, in the Hospital Reports for 1859, Vol. V., p. 147; and to a case by Mr. Norise, of Brighton, in *Medical Times and Gazette*, Sept. 2, 1865; and in 1866 a case of the mixed form of the disease was received into Guy's Hospital, under the care of Dr. Owen Rees. But the majority of cases recently recognised in this country have occurred in persons who have either been born in one of our tropical possessions, or who had been long resident there. Such cases are more frequent than is generally believed, and some anomalous forms of intractable skin-disease are pronounced to be vestiges or obscure expressions of a partially leprous diathesis.

(b.) *Causation*.—The position which this disease now holds in the nomenclature of the College of Physicians implies that it is of constitutional development—a theory which is now more generally accepted

than the belief in the propagation of the disease by contagion. To a very great extent the disease is hereditary, and springs usually from some specific taint transmitted from parents to children. Out of 213 cases recorded by Danielssen and Boeck, 185 were derived from families in which one or more persons had suffered from the disease. In 28 cases only, no hereditary history could be made out. In Iceland in 1837, Hjaltefin, among 125 diseased persons, hardly found one who did not belong to a diseased family. As with other diseases of hereditary development, it sometimes passes over one or two generations, to appear in the third and fourth; and it has been found to manifest itself more in the second and fourth generations, and with much greater intensity, than in the first or third generation. The hereditary influence is most marked on the maternal side, and more in a collateral than in a direct line. Leprosy is believed to be decidedly more frequent in males than in females; but females afflicted with leprosy usually live much more secluded than men, and are less willing to expose themselves when afflicted; so that the real truth may be that the disease is quite as frequent in women as in men. The proclivities of particular races to the disease are not known with any certainty; but the great majority of cases of leprosy in all countries occur among the lowest and poorest of the people. In some countries, however, it is believed that the better-conditioned classes are quite as frequently affected as the poor. It appears to be most frequently met with in low and malarial districts, especially on or near the sea-shore; but it is also common in inland and hilly districts, as among the Hottentots, the mountain inhabitants of Lebanon and of the highlands in the north of Persia and Hindostan. The dwellings of the leprosy poor are generally as miserable and unwholesome as they can be; and personal uncleanness of the sufferers is on a par with the filthiness of their abodes. Ablution of the body is seldom or ever thought of, so that the skin is often encrusted with the impurities of years. Their clothing is not less foul, being seldom taken off by night or by day, but kept on the person as long as it will hold together. The food of the leprosy poor is almost always described as deficient in nourishment, generally unwholesome, and often insufficient in quantity. The frequent or constant use of fish—much salted, and often tainted or semi-putrid—is more frequently referred to as a cause of the disease than any other article of food, especially in the West Indies, the Cape of Good Hope, Egypt, Crete, Corfu, Calcutta, and Ceylon. Deficiency of fresh meat and vegetables in the diet is also frequently noticed. Rancid oil is also believed to be an exciting cause of the disease when consumed in large quantities. In India the eating of unsound pulses is believed to favour the development of leprosy. In Norway most of the cases occur among the very poorest of the people living round the shores of the deep bays or fiords on the west coast. Their food consists almost entirely of fish—fresh, or very much salted—meal, potatoes, and badly made cheese. Fresh meat is rare. The leprosy themselves generally ascribe their disease to constant exposure in the cold, damp, and wet weather of the climate, frequent at all seasons, and especially in the long severe winters. They often get thoroughly wet to the skin

and chilled, without the means or opportunity of drying their clothes, or obtaining any warm nutriment. The development of leprosy is not restricted to any period of life. It occurs most frequently about puberty, and onwards to maturity; but it has been observed from infancy or early childhood up to fifty years of age or upwards. Occasionally signs of the tuberculated form of leprosy have been seen in the offspring of lepers at or soon after birth. According to the observations of Danielssen and Boeck, the tubercular form of leprosy begins to manifest itself generally at some period between ten and forty years of age, most frequently between the twentieth and the thirtieth year of life; the *non-tuberculated* form between the tenth and the thirtieth year. Scarcely any period of life is exempt. Young children have been seen afflicted with the tubercles of leprosy, and their parents stated that these children had at birth bluish spots on the skin, which subsequently became tuberculated. At eight years of age the anæsthetic form has been met with in Norway; and in those cases there had been bullæ on the extremities at a very early period of life.

(c.) *Morbid Anatomy*.—The principal morbid changes have been carefully investigated by Drs. Danielssen and Boeck; and they have been especially confirmed by Dr. Carter, of Bombay (*Journal of Med. and Phys. Soc.*, Vol. VIII., N.S.) The anatomical signs of the disease are derived from the infiltrating of a peculiar exudation into the skin, the mucous membranes, the glands, the serous membranes, and the parenchyma of some organs. In the developed stage of the *tuberculated* form, the *chorion* or *cutis vera* of the affected parts is tumefied and thickened. On squeezing it between the fingers, a yellowish-white, viscid, or gruelly fluid exudes, and a gelatinous or lardaceous effusion infiltrates the areolar tissue underneath the skin, to which it firmly adheres. The subcutaneous veins and nerves are also thickened and enlarged from this effused material on their surface. In the more advanced stages of the disease, the deep-seated parts, as well as the superficial nerves especially lying near to ulcerations, are very much thickened and enlarged, in consequence of the results of inflammation of their sheaths. The *mucous membrane of the nares, fauces, and larynx* is swollen, occupied with tubercles or nodules, soft, and of a yellowish colour, and often ulcerated. The opening of the larynx is frequently the seat of morbid deposit, so as nearly to close up the *rima glottidis*. Similar nodules are occasionally found in the mucous lining of the trachea and larger bronchi. The *cervical glands* are occasionally much enlarged. The *substance of the lungs* is seldom altered; but the pleuræ are often much thickened, in consequence of the exudation into its tissue, and the formation of an infinite crowd of tubercles which sometimes run together. The *subperitoneal connective tissue* may also be similarly infiltrated. The *mesenteric glands* are generally more or less enlarged. Isolated rounded ulcers are occasionally found on the inner surface of the intestines. The *liver* is sometimes the seat of the deposit of nodules. In the advanced stages of the disease the *kidneys* are usually affected with a form of albuminous nephritis.

In the *non-tuberculated* form, when anæsthesia is the characteristic feature, and where paralysis of muscles as well as of the skin exists, the

skin is often very much attenuated, and the muscles atrophied. All fat has disappeared. The connective tissue in the parts surrounding the seat of ulceration or necrosis is infiltrated with a serous or lardaceous deposit; and the nerves are excessively swollen, their sheaths being filled with a fine albuminous matter, in which the ultimate nerve filaments are imbedded. The axillary and inguinal glands are often much enlarged. The central organs of the nervous system are usually the seat of notable morbid changes, chiefly congestion of the posterior or dorsal veins of the spinal marrow, effusion of an albuminous serum within the *arachnoid membrane*, and between it and the *dura mater*, adhesion of the *arachnoid membrane* to the *dura mater*, and consolidation or hardening of the substance of the spinal cord at the part affected. The cord is generally somewhat contracted in size, and sometimes so atrophied as not to be much larger than a quill in its dimensions. The cineritious substance has generally acquired a dirty yellow colour, so as to resemble the medullary substance. The roots of the nerves within the vertebral canal are invested with albuminous exudation. The axillary and ischiatic plexuses, and the principal nerves issuing from them, may be visibly atrophied. The *cervical and lumbar regions* of the cord are always most conspicuously affected. Within the *cranial cavity* the lesions are sometimes similar to those in the vertebral canal, but less decided and advanced. In well-marked anæsthesia of the face, the Gasserian ganglion is always the seat of lesion; usually it is imbedded in a sero-albuminous exudation, so considerable that the part bulges out, and the nerve filaments are glued together by the exudation. The *blood* contains albumen and fibrine in excess, even before spots appear on the skin. Local congestions and hyperæmia then ensue. Danielssen and Boeck have made several analyses of the blood after the method employed by Simon; and their results have been condensed by Dr. Parkes in the following tables:—

A. ANALYSES OF VENOUS BLOOD IN NORWEGIAN TUBERCULOUS
ELEPHANTIASIS, BY DANIELSSEN AND BOECK.

No.	Sex.	Age.	Period of Disease.	Sp. gr. of whole blood.	In 1000 parts.						
					Fib.	Fat.	Album.	Globu- line.	Hæm- atine.	Salts and Extract	Water.
1	M.	24½	Early precursory symptoms	1·046	3·201	2·531	100·609	65·831	3·273	11·244	813·311
2	M.	26	Advanced	1·049	4·539	3·421	73·139	96·186	5·465	10·930	807·521
3	M.	38	Do. (12 years)	1·051	4·265	4·240	116·971	39·672	6·135	17·382	811·335
4	M.	36	Do. (8 years)	1·042	4·722	2·806	93·092	46·719	4·153	6·921	851·687
5	M.	29	Do. (6 years)	1·048	4·878	5·309	93·913	74·504	2·713	15·861	802·822
6	M.	22	Do. (10 years)	1·053	3·592	4·623	128·786	65·336	2·830	15·332	779·522
7	F.	34	Do. (between 3 and 4 years)	1·048	3·111	2·336	106·926	66·774	3·547	13·532	803·771
8	F.	43	Do. 6 years, complicated with Anæsthetic Elephantiasis.	1·052	4·	6·1	113·6	68·	4·1	1·41	802·8
Healthy blood analysed by the same method.											
1	F.	20	Healthy.	1·051	2·205	2·129	79·353	94·437	3·299	11·339	807·228

B. ANALYSES OF VENOUS BLOOD IN ANÆSTHETIC ELEPHANTIASIS,
BY DANIELSSEN.

No.	Sex.	Age.	Period of Disease.	Sp. gr. of whole blood.	In 1000 parts.						
					Fib.	Fat.	Album.	Globu- line.	Hæm- atine.	Salts and Extract	Water.
1	F.	48	Advanced (17 years)	1·052	2·578	2·457	100·500	84·420	4·020	10·497	205·524
2	M.	29	Do. (13 years)	1·046	2·409	4·854	135·975	80·850	4·900	17·150	247·184
3	M.	41	Do. (9 years)	1·045	6·027	3·440	104·649	62·189	7·077	14·582	199·010
Same. Second analysis, Albu- minuria (10½ years).				1·042	4·361	3·567	66·733	72·209	4·449	6·844	159·205
4	M.	29	Do. (12 years)	1·058	3·092	2·777	52·221	139·404	5·089	8·851	212·492
5	F.	40	Do. (24 years)	1·052	2·967	4·662	60·150	121·652	5·757	9·011	199·251

The *minute anatomy* shows that the material of infiltration into the skin composing the tubercles, and into the sheaths of nerves and spinal cord, has the same microscopic and chemical composition. When newly formed it consists of a delicate fibrous network or stroma, in the meshes of which lie a great number of adherent whitish granules, which cannot easily be separated by washing. Acetic acid renders the fibrillæ transparent, but increases the opacity of the granules. At a later period the fibrous network and granules disappear, and a great number of cells can be seen, rather larger than the so-called exudation corpuscles, oblong in shape, and inclosing a large nucleus, which leaves only a small space between itself and the cell-wall. This space resembles a shining ring. The nucleus is of a grey colour, and less transparent than the ring. It incloses from seven to eight well-marked brownish granules; which probably accounts for the general brown colour which the old nodules of leprosy acquire. The outer cell-wall is rendered transparent by acetic acid; but the nucleus is not much changed. The texture of the cutaneous vessels and nerves is all destroyed; the sudoriferous glands have disappeared, and only a homogeneous mass is left. The hair follicles are in part destroyed; but the sebaceous follicles are enlarged. In the Museum of Oxford, in 1865, I saw some very good specimens preserved and prepared by Vanderkolk. The material exuded in the spinal sheath was yellow, diaphanous, and interspersed with numerous bright points, perhaps fatty. Chemical analysis of the exudation in the anæsthetic form, by Danielssen, gave the following results:—*Water*, 80·45; *Albumen*, 17·38; *Fibrine*, traces only; *Salts*, 2·10. Before softening in the tuberculated form, no effect was produced on test paper; after softening the reaction was alkaline. The firm mass contained fibrine, albumen in large quantity, fat, and salts. After softening there was less fibrine.

Symptoms.—Two forms of the disease, the “*tuberculous*” and the “*anæsthetic*,” have been described; but as the term *tubercular* or *tuberculous* is suggestive of some alliance with *tuberculosis*—which leprosy does not possess—the College of Physicians propose to designate the form in question by the term “*tuberculated leprosy*,” and inasmuch as the loss of sensibility is not confined to the “*anæsthetic*” form of the disease, the term “*non-tuberculated*” might designate the other form. But as those two forms, which have often been described as distinct, are not unfre-

quently known to co-exist, or to succeed one another, in the same patient, they are now regarded as modifications of one morbid condition. There are also cases included under the varieties of *non-tuberculated* leprosy characterised by white spots or blotches on the skin, and which are also more or less anæsthetic. They are sometimes named *leucopathic*. Non-tuberculated leprosy also comprehends those cases in which the cutaneous eruption consists of circular or annular spots, not unlike *lepra vulgaris*, but in which the centre of the spot is anæsthetic, and having other distinctive characters of leprosy. These two varieties seem to be of frequent occurrence in the East Indies. Heberden witnessed the acute and rapid course of the disease in Madeira, and Danielssen and Boeck in Norway. It is there accompanied by fever, and commences suddenly by violent delirium, sleeplessness, a dry, reddish skin, a quick full pulse (120 to 130), dry and red tongue, constipation, scanty and pale urine. In from twelve to fifteen days there suddenly appears patches all over the body, which become elevated, and pass into large tubercles; and the general constitutional symptoms subside as the tubercles rise up. Thus, in a few weeks the disease has arrived at a stage which it usually takes years to attain, and its course afterwards is usually chronic. The disease is one *sui generis*, characterised by certain kinds of cutaneous eruption and discoloration, associated with a tendency to ulceration or to the death of the affected parts, and with disorders of innervation, such as *hyperæsthesia*, but more particularly by impairment or loss of sensibility (*anæsthesia*).

Before any visible or external mark of leprosy is seen, there are usually premonitory symptoms expressed, which continue for a longer or shorter period. A feeling of general *malaise* is obscurely marked and ill-defined, having no uniform or regular course. It is usually indicated by recurrent ague-like chills, or by formication and itching in the limbs, by numbness in a hand or foot, or in one or more of the fingers or toes, and by general weakness and depression alike of mind and body. In certain cases of the non-tuberculated form there is sometimes in the early stage of the disease an intense burning sensation, and a painful tingling along the course of one or more of the nerves of a limb, increased by pinching or tapping the skin over the affected part. This sensation is sometimes accompanied by a dry fissured state of the skin, falling off of the hair, and shrivelling of the nails. Before the eruption of the elevated, discoloured, and shining spots characteristic of the tuberculated form of leprosy, an erythematous redness of the parts about to be affected takes place—generally the face—attended with a feeling of heat or burning, a puffiness of the features, and increased sensibility of the skin. The duration of these premonitory symptoms varies much in different cases before the appearance of the characteristic eruptions of the cutaneous *tubera* or nodules of leprosy. Hyperæsthesia, or increased sensibility, is invariably replaced, in course of time, by anæsthesia of the affected parts. Drs. Danielssen and Boeck have noticed that an excessive sensibility in some spots is sometimes accompanied by periodic rigors. This hyperæsthesia is sometimes limited to patches of the skin; at other times it affects extensive surfaces, as entire limbs, and a great part of the face. It may gradually increase to such a degree that, on the slightest touch, the patient experiences a sensation almost like an electrical shock. Every movement causes violent pains, as

if the patient were pricked with a thousand pin points. Such extreme sensitiveness may continue for several years; but eventually it diminishes till it ceases altogether. It is gradually succeeded by anæsthesia of the affected parts, which becomes more and more complete. The same observers record that in the tuberculated form several successive outbreaks and disappearances of the discoloured spots of the skin occur, after intervals of several weeks or months, or even of a few years, before they remain and become persistent. Such external symptoms were generally preceded by constitutional *malaise*, accompanied sometimes with a slight febrile disturbance of longer or shorter duration. In a few rare instances the disease has set in with sharp paroxysms of fever, enduring for a week or two, and followed by the eruption of bluish spots on the surface. The case then either lapsed into the ordinary chronic form of the disease, or the patient was carried off by an attack of *pleurisy*, *pneumonia*, or *meningitis*. The formation of bullæ in the anæsthetic varieties is usually among the earliest symptoms, and they supervene upon a state of general weakness, lassitude, and depression. The seat of these bullæ is very often the palm of the hand or sole of the foot. They burst, and form superficial ulcers, which heal after a time. Although these bullæ may go on recurring at short intervals for a length of time without the general health being much impaired, yet their occurrence is an almost certain premonition of the anæsthesia which will follow. The appearance of white spots or blotches on the skin is also a frequent but not a constant precursor of anæsthesia.

The following summary may express in a short form the symptoms of the two forms of leprosy:—(1.) *Tubercular Leprosy* presents the following characters:—(a.) Tumefaction or tubercular thickening of the skin, principally of the face and extremities, less marked over the trunk. (b.) The affected skin is discoloured, dark-brown, bronzed or shining, with the sensibility much diminished or entirely lost. (c.) The mucous membrane of the mouth and fauces ultimately becomes affected, and the voice altered. (d.) Contraction of the fingers and toes is a frequent symptom, and the phalanges may drop off from ulcerated fissures forming over the articulations, or from *sphacelus* supervening on ulceration. The entire hand or foot may thus be lost. (e.) The constitutional disturbance is much greater in this than in the other form. (2.) *Non-tuberculous Leprosy*.—The anæsthetic form is the most frequent in India. It presents the following characters:—(a.) Anæsthesia of the skin of the face, ears, and extremities, followed in the latter case by atrophy, interstitial absorption, and occasionally ulceration of the benumbed parts, notably of the fingers and toes, with little or no constitutional disturbance. (b.) Large circular superficial ulcers may form on the lower extremities. (c.) The affected fingers and toes become contracted, the joints enlarged, the ends of the fingers broad, flat, or clubbed (*Edin. Med. Journ.*, Dec., 1868, p. 546).

Diagnosis is between syphilis and venereal tuberculated affections; and more especially it is difficult, if the two morbid states co-exist. *Yaws* is another disease with which it may be confounded, as it is a disease met with in many countries where leprosy is common. *Yaws* was formerly very common in several of the West India islands. The disease may also be confounded with *Scrofula*, with which some consider it is at least closely allied. The elephantoid enlargements of the scrotum and lower

extremities have also been considered in many places as allied to leprosy; and the circumstance of the two diseases bearing the same generic name (*Elephantiasis Arabum*, the "Barbadoes" or "Cochin leg"), has doubtless contributed to the belief. They have no real affinity with each other, although both are sometimes endemic in the same countries, and may co-exist in the same patient.

Prognosis.—The more the disease is developed, the more unfavourable must be the prognosis. Nevertheless, Drs. Danielssen and Boeck will not say that it is incurable, even in its advanced stage. Nature seems to have brought about a cure in several instances where the patients were grievously affected. No definite duration can be assigned to leprosy. According to Danielssen and Boeck, the average duration of the *tuberculated form* among patients in the Bergen Hospital, from 1840 to 1847, was between nine and ten years; and of the *anæsthetic form*, from eighteen to nineteen years. The shortest period was three years, and the longest twenty-two years of the tuberculated cases. The shortest period of the anæsthetic cases was five years, and the longest thirty-one years. Much seems to depend—(1.) On the age at which the disease appears; (2.) upon the constitution of the patient; and, (3.) the circumstances in which he is placed. The tuberculated form is the most rapidly fatal; but in all forms the disease may remain stationary for many years. Death is generally the result of some intercurrent affection, such as *diarrhœa*, *dysentery*, or *inflammation of the lungs and air passages*; and if lepers are attacked with the *malarious fevers* of the country they usually die. *Disease of the kidney*, with *albuminuria*, is not unfrequent; and death in some cases is from *marasmus* and *atrophy*. Destitution and neglect greatly aggravate the liability to such intercurrent diseases. Dr. Fiddes relates a case of spontaneous cure of leprosy, after an attack of erysipelas (*Edin. Med. Journal*, June, 1857).

Treatment.—The only hope of exterminating leprosy lies in the adoption of hygienic measures tending to improve the general conditions, physical and moral, of the leprous poor. Medicinal treatment is of no avail, unless combined with the regular use of a nutritive unstimulating diet, suitable clothing, protection against the vicissitudes of the weather, personal cleanliness, and exercise in the open air. The medicines which have been found of most service are *tonics* and *alteratives*, especially preparations of *iron* and *iodine*. *Mercury* is extremely injurious. *Arsenic* has for centuries been held in high esteem in India as a remedy in true leprosy. It is given in the following combination:—105 grains of arsenious acid are triturated with five or six times the quantity of black pepper. Thus made into a mass, it constitutes the material of the well-known "Tanjore pill," of which one the size of a "*tare*" is to be taken night and morning (Waring's *Therapeutics*, p. 110). The systematic use of baths, simple, saline, or sulphurated, are decidedly beneficial. Cupping-glasses or moxas, along the line of the spinal column, has been of marked advantage in relieving the lesions of innervation, whether of increased or diminished sensibility. At a very early period of the inquiry carried out by the College of Physicians, the nature of the replies received enabled the College to assure the Secretary of State for the Colonies that "there was no evidence which, in their opinion, justified any recourse for

the compulsory segregation of lepers." After receiving this decided opinion, the Duke of Newcastle forthwith issued a circular to the Governors of the Colonies, expressing his opinion, "that any laws affecting the personal liberty of lepers ought to be repealed; and that, in the meantime, if they shall not be repealed, any action of the executive government in enforcement of them, which is merely authorised and not enjoined by the law, ought to cease." There appears no more need (or just about the same) for restricting the liberty of lepers as for restricting the liberty of those afflicted with gout.

SCROFULA.

LATIN EQ., *Struma*; FRENCH EQ., *Scrofule*; GERMAN EQ., *Scrophulose*; ITALIAN EQ., *Scrofola*.

Definition.—*A constitutional disease, resulting in the deposit of a soft unorganised matter of a white or pale-yellow colour, firm, resembling curd or soft new cheese, but less tough, sometimes granular and friable, and consisting of a large proportion of albuminous matter. It is often found mixed with purulent-like fluid, or occurring in rounded masses of different degrees of firmness, and varying in bulk from that of a millet-seed to a hen's egg or larger; sometimes contained within natural canals and cavities of the body, and sometimes inclosed in cysts, or occasionally diffused as if by infiltration through the texture of a part. To the rounded masses the name of tubercle has been given; and the substance itself has been named scrofulous or tuberculous matter. Scrofula also expresses itself in specific forms of inflammation or ulceration. It manifests a remarkable tendency to certain specific forms of nutritive disorder, which are strikingly wasting in their effects upon the body, namely:—(a.) Scrofula with tubercle; (b.) scrofula without tubercle; and (c.) certain local scrofulous affections, such as eruptions of a peculiar kind, many cutaneous ulcers, certain enlargements of the joints, tubercular meningitis, hydrocephalus, scrofulous ophthalmia, tubercular pericarditis, scrofulous diseases of glands, phthisis pulmonalis or pulmonary consumption, acute miliary tuberculosis, tabes mesenterica, and tubercular peritonitis.*

Pathology.—(a.) *History.*—Previous to 1829, when the late Sir James Clark published his classic work *On Climate*, the tendency of pathological research into the nature of the diseases just mentioned was to keep up the idea that they were merely local, especially *pulmonary consumption*, and referable to a local cause. The terms *phthisis*, *consumption*, or *wasting*, were originally used vaguely to designate a variety of chronic diseases, described entirely by local symptoms and physical signs, and therefore having few characters in common, except the marked emaciation which attends them. When researches in morbid anatomy became more frequent and efficient, the local lesion most commonly connected with the wasting of the body was discovered to be a peculiar morbid condition of the lungs, and to this the name of "*consumption*" was applied, and afterwards more definitely, "*pulmonary consumption*." From the frequency of this local lesion the pulmonary state of necessity attracted far greater attention than any other form of *scrofula* in which *wasting* was a marked

feature; and when lesions in other organs were found associated with "*pulmonary consumption*," they were regarded as complications rather than as local manifestations of one and the same general constitutional state, and were looked upon as the primary cause of the emaciation. In 1819 Laennec first showed, by his accurate *post-mortem* observations on the state of the lungs, that growths to which the name of "tubercles" were applied formed almost the sole cause of *consumption*, and consequently he restricted the term *phthisis* to "*the disease produced by tubercles in the lungs*." Louis and Andral confirmed the observations of Laennec; and thus the view of this morbid state, based as it was on morbid anatomy *alone*, led to the enunciation of limited and erroneous doctrines regarding the real nature of the disease which the existence of *tubercles* indicates, either in the lungs or in any other part of the body. The relations subsisting between *pulmonary phthisis*, *consumption*, or *wasting*, and the occurrence of "tubercle," must now be even still more modified under the combined researches of morbid anatomy and progressive knowledge in pathology. *Consumption*, or *wasting*, in the common acceptance of the term, is not now found due always to tubercle (ADDISON, GAIRDNER, REINHARDT, VIRCHOW, JENNER, and MACLACHLAN), so that the terms *phthisis* and *tubercle* cannot now be considered always as synonymous. Ulceration of the lungs and partial destruction of pulmonary tissue are found to arise from other causes than the existence of *tubercles*, which become equally efficient causes of phthisical mischief (BAYLE, ARMSTRONG, GRAVES, STOKES, and MACLACHLAN). Nay, we know also that many states of the body to which the name *scrofula* or *struma* is applied are not necessarily attended with "tubercles" at all; yet, when the local lesions of a tubercular nature are observed to be connected with marked constitutional states under various circumstances, the connection between *scrofula with and without tubercle* is seen to be of the closest description, and their pathological history is now generally believed to be identical—the view which the College of Physicians would seem to favour, judging from the terms of the definition which I have incorporated in the one I have placed at the head of this account of *scrofula*.

The extensive observations of morbid anatomists since the time of Laennec, elucidated by the most learned pathologists of the age, now tend to establish, on a very broad foundation, the doctrine first so strenuously and ably advocated by Sir James Clark, that the morbid conditions now mentioned are due to what he termed a "tuberculous cachexia;" and accordingly it is proposed to consider the *constitutional* disease, of which the occurrence and growth of "tubercles" is but a local expression, under the general title of SCROFULA, of which the varieties are—(a.) *Scrofula with tubercle*; and, (b.) *Scrofula without tubercle*; while the local scrofulous affections are those named at the commencement of this topic. Many questions bearing upon the relation of *scrofula*, *tuberculosis*, and *tubercle* to each other remain still a subject of discussion; some believing in the identity of *scrofulous* and *tubercular* affections, while others consider them as distinct; so that in the history of *scrofula* there is still much doubt and uncertainty. The question has been mostly studied in relation to *pulmonary consumption*, to enlargements of the lymphatic glands, and to the relation of true miliary tubercle to some antecedent condition—a

relationship which has been the question of the day for 200 years back, especially as regards the relation of true miliary tubercle to pulmonary consumption—a relationship which will be considered in detail under the head of “pulmonary phthisis.” The history of “*tuberculosis*” goes farther back than that of *miliary tubercle*. Hippocrates describes the disease as developed in the lungs. Stark in 1785, and Baillie in 1794, first described *miliary tubercle*. From the time of Gordonius in the twelfth century, writers on scrofula have particularly dwelt upon the fact of there being a temperament or *diathesis* proper to it; and the *diathesis* which expresses the latent existence of scrofula has been variously described by the terms *tuberculous*, *scrofulous*, or *strumous* diathesis. Schönlein, in the third decade of the present century, first used the term “*tuberculosis*.”

When the late Sir James Clark published his treatise *On Pulmonary Consumption*, in 1835, he expressed some doubts as to tuberculous diseases being comparatively more prevalent at the time he wrote than they were some fifty or a hundred years before. He was of opinion that while many circumstances favoured the probability of a diminution of tuberculous diseases, there were circumstances which might materially counteract such an influence; and while he was convinced that tuberculous diseases had increased in the middle and upper ranks of life at least, he believed that, as a rule, the constitutions of the three past generations had deteriorated progressively from father to son. The annual returns of our Registrar-General up to 1846 show a progressive increase in the mortality from consumption. In 1854, however, we find it recorded “that phthisis is twice as fatal as any other disease in England, but that within the last eight years it appears to have declined to some extent.” The inquiries of Dr. H. Greenhow show that this diminution is in a great degree due to those hygienic measures which have contributed to diminish the causes of *miasmatic* diseases in general; and which have especially lessened the prevalence of those *febrile exanthematous diseases* which, by weakening the constitution, tend to bring about those conditions under which that bad habit of body is established which leads to the growth of tubercles and the development of scrofulous affections. To no kind of sanitary measure are we more indebted for this result than to the influence of vaccination in diminishing small-pox—a disease which, of all others, seems to have tended to the development of the scrofulous cachexia as a sequel to its existence. Accordingly we find it recorded by Dr. Greenhow that “during the middle of last century, before vaccination was known, the scrofulous death-rate was more than five times as great as our present one; and the pulmonary death-rate of the present time is seven per cent. lower than the pulmonary death-rate of 1746–55.” While, therefore, such statements and careful observations, extended through long periods of time, show how much may be done by general sanitary measures in preventing the extension of scrofulous diseases, there is still great necessity for a careful study of the nature of these diseases; for we find them in reality *decimating* the civilised part of the world, cutting off in some instances as many as 35 per cent. of our metropolitan populations, and a much larger percentage of the army.

(b.) *Causation*.—Clinical and pathological research tend more and more to confirm the belief in the constitutional origin of scrofula. There is

undoubtedly a predisposition to scrofulous growths, either hereditary or acquired; and that there is a bad habit of body—a cachexia—which precedes the development of tubercle, is now almost a general belief. The development of such masses of matter, as stated in the definition, to which the name of tubercle has been given wherever found, is undoubtedly a local lesion or expression which indicates a *constitutional disease*. It has been hitherto, however, the custom to name the local lesion as the *disease*, rather than to describe the constitutional state. Thus, when the lesions have been most marked in the bones and glands, the name of *scrofula* has been given to the condition; when the lungs are the site of the deposits, *phthisis* or *consumption* is the name by which the condition is familiar; and when in the glands of the mesentery, it has been called “*tubercles mesenterica*,” and in the meninges of the brain it has been named “*hydrocephalus*,” or “water in the head.” The constitutional disease under which all these affections are now comprehended is named “*scrofula*,” and has been shortly defined by the College of Physicians as “*a constitutional disease resulting either in the deposit of tubercle or in specific forms of inflammation and ulceration.*” The presence of this scrofulous or tuberculous matter is satisfactory evidence of the existence of scrofula.

The changes in the general system by which the scrofulous cachexia is brought about are apparent in the abnormal physical condition of the patient, and in the vitiated exercise of some of the vital functions especially connected with nutrition. To the late Dr. Tweedy Todd, Sir James Clark, and Professor Bennett, the profession is principally indebted for the clear and earnest elucidation of *scrofula* or *tuberculosis* considered as a constitutional affection; and on our recognition of it as such rests our only hope of success in the prevention and treatment of this most formidable scourge of civilised society. Many observations have been made and statements recorded with the view to connect scrofula with morbid states of the blood, but hitherto no constant morbid condition of the circulating fluid can be said to be peculiar to the disease. We must look to something beyond—we must look to the pabulum which ministers to the nutrition of the body itself through the blood; for with an impoverished state of that fluid there is doubtless an impoverished state of the tissues (Dr. J. H. BENNETT).

But there are some curious and detached observations which, when connected together in certain pathological relations, appear to throw more light on the nature of the constitutional state which leads to the development of scrofula than any single observation of individual authors. These are—(1.) There is to be noticed the albuminous character of the fluid material which infiltrates the tissues of an organ previous to the process by which this infiltrate is in part changed by coagulation into *tubercles*, each of which may block up from three to twenty air-vesicles in a lung. (2.) There is in some cases, as shown by Drs. Alison, Williams, Bennett, and Rokitsansky, an obvious affinity between the lymph of the blood and tubercle. (3.) There is an albuminosity or venous state of the blood which is considered by some as peculiar to the scrofulous state of the constitution. (4.) There is the peculiar state of the blood, amounting to an appearance of leucæmia, which immediately succeeds digestion in healthy persons, as observed and described by Dr. Andrew Buchanan, of

Glasgow, in *The Transactions of the Philosophical Society* (Vol. II.) of that city, resembling the molecular and corpuscular elements of chyle or lymph, and consisting of fat emulsionised with albumen. To this substance he gave the name of *pabulin*, and which is still further elaborated in the blood, in the glands, and in the lungs, before it takes part in the general morphological changes connected with nutrition. (5.) The observations of Dr. Acherson, of Berlin, and of Dr. Bennett, relative to how nutrition may be impeded by diminishing the *molecular* state of the nutritive elements, and improved by increasing them. (6.) It was first observed by Dr. Wilson Philip "that there were some forms of indigestion which ended in phthisis;" and it was subsequently distinctly stated by Lepelletier that the causes of scrofulous disease were referable—(a.) To those agents which impair the assimilative action; (b.) To the elements of nutrition being insufficient, by the influence of bad diet, impure air, deficient exercise of the general functions, and obstruction of the functions of excretion. (7.) The observations of Panum and Parkes relative to the precipitation of albumen by acids and neutral salts, in which Dr. Parkes especially shows that the albumen as it exists in the serum of the blood is usually in that condition in which it is most easily precipitated by acids and chloride of sodium (*Med. Times and Gazette*, July, 1850 and 1852). (8.) The excess of intestinal acidity in the alimentary canal of phthisical patients, as shown by Dr. Bennett, by which, under some conditions, the albuminous constituents of the food are rendered easily soluble, whilst the alkaline secretions of the saliva and the pancreatic juice are more than neutralised, and so rendered incapable of transforming the carbonaceous constituents of food into oil, or of so preparing fatty matters introduced into the system as will render them easily assimilable. Hence an increased amount of albumen enters the blood compared with fatty elements. According to the observations of Mr. Jonathan Hutchinson, acid eructations were present in 62 per cent. of the cases of dyspepsia which preceded the deposition of tubercle, and were a prominent symptom in 46 per cent. of cases reported on by him in an admirable paper "On the Forms of Dyspepsia preceding and attending Phthisis" (*Med. Times and Gazette*, Vol. X., 1855).

Any one of these statements considered by itself does not seem of much importance; but considered as a whole, in their relation to nutrition and their influence upon morphological changes between the solids and the fluids of the body, they leave very little room for doubt that the bad habit of body in scrofulous affections associated with the growth of *tubercle-matter* must be established in the first instance through the digestive processes, as first described by the late Dr. Tweedy Todd under the name of *strumous dyspepsia*, and which has been since so fully described by the late Sir James Clark, Bennett, Hutchinson, and others. The more closely these links of circumstantial evidence can be bound together, the more intimate a pathological relationship will be found to exist between the albuminous constituents of the food, the blood, and the saline constituents of the circulating fluid, the mal-assimilation of food, and the waste of the tissues, as connected with the development of the bad habit of body associated with scrofula and tubercle. Although it must be confessed that we do not fully understand the living processes by which all the molecular

changes take place to which I have just adverted, yet there can be no doubt that in the seven statements enumerated we obtain some glimpses of a rational pathology which may yet tend to explain the very complex *constitutional morbid state* which precedes and is associated with the growth of tubercle in scrofulous affections, or with the specific inflammations and ulcerations of scrofula. The tendency to the formation of scrofulous matter is not equally great at all periods of life, nor in all parts of the body. Age exercises a paramount influence on the generation of scrofula. It is peculiarly a disorder of childhood and youth (GUERSENT, LOMBARD, PAPA VOINE, ALISON, CLARK, BENNETT). Scrofulous masses in the bronchial glands, the lungs, the cervical glands, the mesenteric glands, the spleen, the pleura, the liver, the small intestines, and the brain, are most common in infancy, childhood, and early adolescence. But the occurrence of tubercle, which forms so large a portion of the lesion in all scrofulous affections, although it has been found in the fetus, and at every period of life up to eighty, yet, after the age of fifteen it is more frequently met with in the intestines, the mesenteric, cervical, and lumbar glands. Scrofulous disease is frequently developed during infancy; and "I have met with several cases of infants," writes Sir James Clark, "dying of consumption within the first year of life, in whom the lungs were not only extensively tuberculous, but contained large caverns, with all the characters of those found in the lungs of adults." After the second year of life, pathologists agree as to the universal prevalence of scrofulous disease: and generally it exists in its greatest extent between the period of the first and second dentition.

The broadest fact established regarding the exciting cause of scrofula is, that the domesticated animal is more liable to scrofulous disease than the same animal in a wild state. The stabled cow, the penned sheep, the tame rabbit, the monkey, the caged lion, tiger, or elephant, are almost invariably cut off by scrofulous affections—no doubt due to deficient ventilation and the abeyance of normal exercise of the pulmonary function.

"There appears to be little doubt that persons who breathe continuously air vitiated by respiration furnish a large percentage of phthisical cases. This cause is potently aided by the coincident conditions of deficient exercise, poor feeding, and, often, excessive work. Baudelocque years ago asserted that impure air was the chief cause of phthisis. Carmichael, in his work on *Scrofula* (1810), gives a number of examples of the influence of foul air and want of exercise in the production of the disorder. Lepelletier, Neill Arnott, Toynbee, Guy, Greenhow, Baly, and others, have collected a good deal of evidence in support of this cause of tuberculosis. Of 104 compositors who worked in rooms of less than 500 cubic feet for each person, 12·50 per cent. had had hæmoptysis; of 115 in rooms having from 500 to 600 cubic feet, 4·35 per cent. had had hæmoptysis; and in 101, in rooms of more than 600 cubic feet, 1·98 had had hæmoptysis (Guy). In the prison of Leopoldstadt, at Vienna, very badly ventilated, in the years 1834–1847, 51·4 per 1,000 died from phthisis, there being 42 cases of acute tuberculosis; while in the well-ventilated House of Correction, in the same city, the deaths from phthisis were only 7·9 per 1,000. The great prevalence of phthisis in the Indian jails is ascribed to bad air and bad food (PARKES, *loc. cit.*, p. 96, 2nd Ed.) Dr. Henry MacCormac is, perhaps, the most enthusiastic advocate of the

doctrine of the effects of re-breathed air in the etiology of tuberculosis; so far does he carry his doctrine that he makes it almost an exclusive cause of phthisis. 'Wherever the air, habitually respired, has been respired in whole or in part before, there tubercular deposits are found.* The greater relative frequency of phthisis in females Dr. Lawson attributes to their indoor occupations and sedentary habits.† The chief cause of the extraordinary frequency of phthisis in the European armies, to be presently spoken of, Dr. Parkes says, 'can scarcely be accounted for in any other way than by supposing the vitiated atmosphere of the barrack-room to be in fault. This is the conclusion which the Sanitary Commissioners for the British Army came to in their Report, after assigning all probable influence to exposure on duty, intemperance, syphilis, and faulty diet. So also the extraordinary amount of consumption which prevails among the men of the Royal and Merchant Navies, and which, in some men-of-war, has amounted to a veritable epidemic, is in all probability attributable to the faulty ventilation' (PARKES). The statistics of the Hospital for Consumption and Diseases of the Chest, London, show that among 3,214 men, who composed all the cases of decided phthisis which were registered as in-patients in ten years, more than one-half—1,812—had followed indoor occupations; and of the 2,413 females nearly all had. Dr. Pollock, one of the physicians, states that the occupation of out-patients would give the same result. It must then be admitted, from overwhelming testimony, 'that deficient ventilation and overcrowded apartments are eminently productive of tubercular disease.' Those trades which require a stooping posture—as tailors, bootmakers, weavers—which by mechanical hinderance to the free entry of air into the lungs, restrict the expansion of the chest-walls, and result in imperfect performance of the respiratory function, and consequent interference with the proper nutrition of the pulmonary tissues, and general lessened vitality of the system, furnish, no doubt, a large proportion of cases of tuberculosis. The influence of lessened breath-motion as an exciting cause of phthisis has been particularly insisted on by Dr. Edward Smith, in his excellent work on *Consumption, its Early and Remediable Stages*" (CLYMER).

Hirsch also has collected a large number of facts, proving that in foundling hospitals, orphan asylums, boarding schools, factories, and similar institutions, the continual abode in a badly ventilated atmosphere, saturated with steam, and poisoned by animal effluvia, and the products of putrefaction, is in the highest degree favourable to the development of scrofula; and that, according to the experience of those institutions where there is no lack of cleanliness, good food, and warm clothing, the above pernicious agents alone suffice to induce the disease. Bowditch, in America, ascribes the most efficient development of scrofula to moisture and damp. The disease springs most frequently, however, from the combined effects of all these different anti-hygienic influences, the chief of which are bad nourishment, want of fresh air, and the constant presence of moisture and damp.

Inheritance of Scrofula.—Parents whose digestive organs are habitually disordered, who suffer from gout, the injurious influence of metallic and other poisons, such as mercury, malaria, syphilis, chlorosis, or paludal anaemia,

* *Consumption, as engendered by Re-breathed Air, &c.*, 2nd Ed. London, 1865.

† *A Practical Treatise on Phthisis Pulmonalis*. Cincinnati, 1861.

the debility of advanced age, so that a cachectic state is induced, entail upon offspring begotten during such morbid states of the parent, to an extent still undetermined, but obvious and appreciable, a tendency to the scrofulous constitution, not necessarily to be expressed by the growth of tubercle in the lungs, but certainly and surely by various forms of dyspepsia and a precarious state of health. There are many circumstances in the state of the parents' health presumed, with justice, to influence the health of the children born under them. Among these may be mentioned a disordered state of the health of the mother, interfering with the state of the fœtus *in utero*, depressing passions, and generally an unhealthy mode of life. "In the present state of our knowledge, it is not possible to determine the various circumstances in the health of the parent which may give rise to scrofulous growth in the child, much less to explain the development of scrofula. I rather allude to them as subjects deserving the investigation of the general pathologist and practical physician. There may be differences of opinion as to the particular condition of the parent which induces the tuberculous constitution in the offspring, and also as to the degree in which this constitution may exist in the child at birth" (Sir JAMES CLARK). In the experience of Dr. Pollock, the disease was hereditary in 362, or 30·16 per cent. of all the cases. Of the remote causes of tuberculosis he writes:—

"Of all these, doubtless, the most important is hereditary predisposition; for, in its absence, how many struggle through influences and errors which destroy others in early life, and attain a considerable longevity? Not all the predisposing causes united could, in a given instance, induce consumption with certainty, without some subtle agent to precipitate, concentrate, and shape those elements of disease into tubercle. . . . In the absence of more accurate knowledge than science yet possesses, we are inclined to refer the actual precipitation of the disease which we call tubercle to the influence of hereditary predisposition acting in conjunction with, or occasionally even in the absence of, all or any of the other remote agents which give rise to phthisis. . . . If to any or all of these conditions that of inherited tendency to phthisis be superadded, very few indeed escape the disease. . . . Finally, the forms of disease, those modifications on which depend its rapidity, development, and duration, are most distinctly reflected in families. How often do we witness child after child carried off at the same age by the same variety of tubercular disease. Tuberculosis, though not present in the parents, may have an ancestral origin generations back."

The concurrent opinions of the entire profession now fully establish the fact that the tuberculous predisposition is capable of transmission from parent to offspring; and the scrofulous constitution has been observed to manifest itself in the child under the following circumstances:—(1.) At birth tubercles have been found in one or more organs (CHAUSOIER, OEHLER, HUSSON, BILLIARD). (2.) The scrofulous cachexia may rapidly show itself. (3.) By the rapid occurrence of tubercles commencing very soon after birth, subsequent to the gradual appearance of symptoms of the scrofulous cachexia. (4.) By a disposition to the various forms of dyspepsia, whose characters have been already noticed (page 1004, *ante*).

Race has also an influence in the production of scrofula. In this country

the tendency of the Creole and Negro to phthisis is notorious. But it is notorious also that the Creole and the Negro, when removed beyond certain limits of the land of their nativity, become tuberculous in other lands besides Great Britain. In the West Indies some of the black races are by no means exempt from this disease, and the Creoles die of phthisis in large numbers in Martinique (RUFZ, NOTT, MORTON). This is the more unlooked for, because as children they live almost in the open air, bathe daily, or still more frequently, and are singularly cleanly in their persons.

Depressing Passions.—Among the predisposing causes of phthisis, there are none more certain than the *depressing passions*, especially when they are profound and long indulged; and this perhaps is the cause of the greater prevalence of this disease in large towns, where bad habits and bad conduct are more common, and often the cause of those bitter regrets which neither time nor consolation can assuage (LAENNEC). There existed in Paris for ten years a nunnery of a new foundation, which had not been able to obtain from the ecclesiastical authorities anything but a temporary tolerance, on account of the severity of its rules. The alimentary regimen of the nuns, although extremely severe, was still not beyond the bounds of nature; but the spirit of the rules of the nunnery, directing the mind to the most terrible rather than to the consoling truths of religion, as well as compelling the inmates to resign themselves in everything to the will of the abbess, produced effects as sad as unexpected. These effects were the same in all. Laennec relates that at the end of two months' sojourn in this house the *menses* became suppressed, and in a month or two afterwards symptoms of phthisis appeared. As the nuns had not been allowed to take the usual vows, he entreated that they would leave the house; and all who followed this advice recovered. But during the ten years that Laennec was physician to this establishment the members were renewed twice or thrice, with the exception of the superior, the *tourière*, the sisters who had the care of the garden, of the kitchen, of the infirmary, or of such as had more frequent intercourse with the city, and consequently greater distraction. The rest died of phthisis."

These circumstances now noticed—namely, *hereditary predisposition*, or the influence of parental cachexia generally, of various sources, especially *syphilis*, the influence of *race*, and of *depressing passions*—all co-operate, where they exist, in establishing the scrofulous cachexia; but the efficient cause seems to be "the impoverished nutrition resulting from *impure air*, and an *improper quantity, quality, or assimilation of food*. So long as misery and poverty exist on the one hand, or dissipation and enervating luxuries on the other, so long will the causes be in operation which induce this terrible disease" (BENNETT).

(c.) *Morbid Anatomy.*—The peculiar growth (called a deposit) which sometimes attends the disease now under consideration is named *tubercle* from its external form, occurring as it does in various sized nodules, isolated or grouped together, or as large irregular masses dispersed through the textures of an organ. One essential character of this scrofulous matter is its incapacity to development beyond the state in which it first becomes visible, and in which state it may remain latent. It generally, however, exhibits a tendency to degenerate in various ways, involving in

such degeneration the destruction of the tissue with which it is surrounded. It contains no trace of fibrous development (ROKITANSKY). The term *tubercle* is always understood to refer to adventitious masses of this nature, the type of which is found in the lungs as the essential anatomical constituent of some forms of pulmonary consumption, especially scrofulous or catarrhal inflammation. But the same material which composes the masses in the lungs is also found in many different forms in other organs; and wherever it occurs it is described as *tubercle*, or *tuberculous* or *scrofulous matter*; and *tuberculous disease*, *scrofulosis* or *tuberculosis*, is the usual designation of the specific malady of which the essential feature is the production of this peculiar matter. It occurs also in the tissue of the alimentary canal; in the peritoneum, arachnoid, or pleura; in the lungs, liver, spleen, or kidney; in the tissue of the lymphatic glands, especially the cervical, inguinal, and mesenteric glands; and sometimes in the pancreas and the tonsils.

I. *General Morbid Anatomy.*—The most eminent pathologists of the day are not at one as to the nature of this scrofulous product. It is a soft unorganised matter, of a white or pale yellow colour, firm, resembling curd or soft new cheese, but less tough, sometimes granular, friable, or even brittle, and consisting of a large proportion of albuminous matter. “The opinions entertained regarding the nature of *tubercle*,” writes Sir William Jenner, “may be divided broadly into two classes” (*Med.-Chir. Rev.*, Jan., 1853). One class of pathologists holds that tubercle is an exudation essentially morbid in character (ROKITANSKY, BENNETT, ANCELL, LEBERT). Another class holds that tubercle is merely a retrograde metamorphosis of pre-existing structures, tissue-elements, or morbid products (WILLIAMS, REINHARDT, HENLE, GULLIVER, ADDISON). Virchow may be said to hold a doctrine combining both views; for while he holds that tubercles are essentially composed of dead tissue-elements, whether these are physiological or morbid products, he also holds that a local process in all cases leads to an exudation of a material (which is poured out during what he terms “a tuberculous inflammation”), which becomes organised to a certain extent, and then dies, breaks up, shrivels, and so leaves a knot, to which the name of tubercle is given. This process Virchow calls *tuberculosis*; and *scrofulosis* is the general constitutional state in which this *tuberculisating* process occurs, which commonly leads to *tuberculosis*; or, in the words of Sir James Paget, “the relation between the two (terms) is, that the *scrofulous* constitution implies a peculiar liability to the *tuberculous* diseases.” According to Virchow, *tuberculosis* is the local process in scrofulous affections in which there occurs an exudation of a material, nutritive or pathological, which develops into cells, and that these cells *tuberculise*, or undergo the tuberculous metamorphosis. *Tuberculisating* is therefore the local process by which the metamorphosis of the elements of a part into tubercle is effected by endogenous development, atrophy, shrivelling, and desiccation of its textural element. A form of *tubercle* thus results out of the detritus of the metamorphosed and atrophied cells, with the remains of the vessels and other structures of the part in which they were seated (JENNER). Whatever, therefore, may be the view entertained regarding the exact nature of *tubercle*, this morbid product appears to us under two conditions, in forms more or less spherical, the contour of the masses

being influenced—(1.) By the nature and movements of the surrounding tissue; and, (2.) by the form of the part in which it first accumulates. The more recent and accurate microscopic observations which have been made into the nature and seat of the tuberculous deposit serve but to establish and confirm the more crude but scientific generalisation made by the late Sir Robert Carswell, when he wrote that “the *free surfaces* of mucous membranes form the chief seat of tuberculous deposit.” It is necessary, however, to extend the significance of the term *free surface*, and make it now apply to the ultimate and microscopic *cul-de-sac* terminations of all mucous tubes. *Grey* and *yellow* tubercle-masses are the names by which such scrofulous lesions are described, and they are first visible in the form of roundish granulations about the size of millet-seeds, and isolated or in groups of nodular masses of more or less irregular form. The *grey tubercle* is tough, soft, and compressible, of a pearly-grey colour, and semi-transparent. Microscopically, it is seen to be composed of irregular-shaped bodies, approaching a round, oval, or triangular form, and varying in size from $\frac{1}{4000}$ to $\frac{1}{2000}$ of an inch. These sometimes appear to be imbedded in a hyaline adhesive basis-substance, infiltrated with granules and molecules, varying from a point scarcely measurable in size to the $\frac{1}{6000}$ part of an inch in diameter. The most characteristic semi-transparent grey granulations appear to contain more of the hyaline basis or connecting substance than of formed elements, the whole field of view being more transparent, and the elements less well expressed or defined. Acetic acid (weak, one part to four or six of water) dissolves many of the granules, and renders all the corpuscles more transparent, while a similarly weak solution of potash completely dissolves them. Various opinions are entertained as to whether or not *tubercle masses* ever consist of *tubercle-cells* with *nuclei*. Rokitsky holds that there are cells present containing one or more nuclei, which indicate an endogenous development and growth of the elements just noticed. Gulliver and Vogel believe in the existence of such nucleated cells; Bennett, on the other hand, has never been able to discover nuclei in the corpuscles of tubercle. By many these cell-like elements are described and believed to be nuclei (BENNETT, SCHROEDER VAN DER KOLK, ROKITSKY, PAGET, SIEVEKING); and the view now related as entertained by Virchow (how existing tissue-elements may tuberculise) renders it highly probable that they may be the nuclei of epithelial cells, as suggested by Van der Kolk, or of other cell-elements, normal or pathological, as believed by Virchow; or they may be cell-particles, like nuclei, of slow formation and without any tendency to reproduction, but tending to disintegrate or break down into molecules, as described by Bennett. Sir James Paget has enumerated the elements of tubercle as follows:—

“1. Molecules, granules, and oil-particles, usually of small size and extremely predominant in *yellow tubercles*. 2. Nuclei of cytoblasts, of various shapes and structures, but all degenerate or defective: some glittering, hard-edged, wrinkled, and withered; others granular; and few or none with distinct nucleoli. 3. Nucleated cells, similarly misshapen, withered, or granular. 4. Certain compound cells, as described by Van der Kolk, and consisting of epithelium charged with the nuclei which become the common tubercle-corpuscles.”

Yellow tubercle is of various shades of colour, and occurs more often in the lungs in masses of variable size, generally larger than the *grey* deposits. These masses are opaque, friable, and of a cheesy lardaceous consistence (*tyroma*). The (tyromatous) masses of yellow tubercle are commonly grouped so close together that the movements of the lungs cause them to become fused in masses half an inch or more in diameter. Microscopically, *yellow tubercle* contains a much greater abundance of fine molecules than the grey tubercle, and there are also present in it elements similar to those in grey tubercles, which are shrivelled, indented, and wrinkled. There are thus no positive or characteristic morphological elements in the tubercle of scrofula, and the pathologist is obliged to make its microscopic diagnosis by a process of exclusion or elimination. For example, if he finds that the substance he is examining consists of the elements just enumerated, and that there are *no blood-vessels nor blood-spaces* in its interior, and that there is nowhere in it any fibrous matrix inclosing cells, he is surely dealing with a scrofulous (tyromatous) formation. But, on the other hand, if he finds, besides these elements, a fine vascular provision for its nutrition and growth, with cells of a fully developed kind, and others degenerating, with blood-spaces, and having an areolar matrix-tissue, then he is most probably dealing with a malignant infiltrating growth, or a pneumonia in the lung, between the gradual invasion of which and the tubercle of scrofula there are points of analogy as well as of difference. The walls of the pulmonary artery are thickened in both; also the lining membrane of the bronchial tubes.

The fatty degeneration of the tubercle of scrofula seems to be a subsequent process, associated with a softer state of the mass in which the organic cell-elements still remain. To this oily or fatty degeneration is due the *yellow colour* of the tubercle mass; and hence, also, this yellow form is regarded as a secondary form to the grey granulation. It is in reality a degeneration or retrograde change, cheese-like or tyromatous.

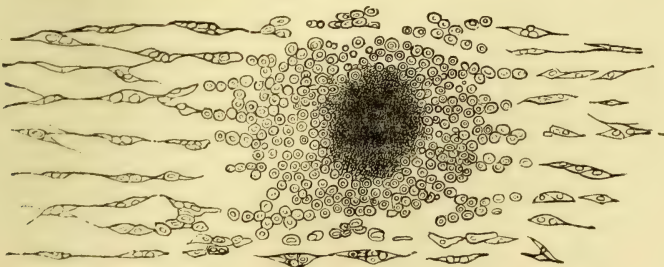


Fig 106.*

And this oily degeneration is not so favourable a local change as that by which the tubercle already formed wastes and *dries up*, while its further development ceases, and the mass assumes a cartilaginous-like consistence.

* Development of tubercle from connective tissue in the pleura. The whole succession of transitions is seen from the simple connective-tissue corpuscles, the division of the nuclei and cells, up to the production of the tubercle-granule, the cells of which in the middle are disintegrating into fatty granular *debris*. 300 diameters (After VIRCHOW.)

At the same time calcareous salts are deposited in the mass, which then hardens or cretifies.

Vogel, Wedl, Virchow, and several other first-rate observers, are agreed that "an organised new formation constitutes the basis of the pathological structure known as tubercle"—the crude tubercle of scrofula. When examined in its early state, tubercle takes the form of a granule or a knot, as Virchow has demonstrated (Fig. 106) from examination of tubercle commencing in serous membranes like the pleura and arachnoid. The knot constitutes a new formation of cellular development, having its first beginnings in the connective tissue, and appearing, *ab initio*, in the shape of a granule. The special anatomical characteristic of the new growth is its extreme richness in nuclei. By isolating the constituents of the young tubercle-knot, either very small cells with one nucleus are obtained—so small that the membrane closely invests the nucleus—or larger cells with multiple division of the nuclei, so that 12, 24, or 30 are contained in one cell. The nuclei are always small, and have a homogeneous, glistening appearance. The elements of scrofulous tubercle are relatively the smallest of any histological formation. It is only at its commencement it is pervaded by blood-vessels. When it enlarges, the little cells are so closely pressed together, that the blood-vessels are gradually obliterated, and only such large ones remain as traverse the tubercle altogether. Fatty degeneration very soon sets in at the centre of the growth, where the oldest cells exist; and when all trace of fluid disappears, the cells of the new growth begin to shrivel, the centre of the tubercle becomes yellow and opaque, and a yellow spot is seen in the midst of the grey translucent granule, knob, or tubercle. This is the commencement of the so-called *cheesy metamorphosis*. This change advances from without, outwardly progressing from cell to cell, till the whole tubercle-granule is gradually involved in the change. The elements of tubercle at last lie so closely together that the growth destroys itself; inducing, by pressure, the disappearance of the blood-vessels which supply its nutrition. The tubercle then begins to die away, and nothing remains but *débris* in the form of the shrunken cells, and cheesy matter—a condition common alike to *pus*, *cancer*, and *sarcoma*. The nature of tubercle cannot be learned after it has reached this stage. It is only at the early period, when engaged in germination, that its anatomical nature and structure can be determined. The histological elements of tubercle resemble those of the lymphatic glands more closely than they do any other normal element of the body (*Cellular Pathology*, Lecture XX). Few objects of morbid anatomy have been submitted to more minute research than "tubercle—crude or old, and fresh or recent;" and there is scarcely any organ where the growth may not occur. Considerable differences of opinion have been entertained respecting the relation of these two varieties. Laennec taught, and it has been the general belief since his time, that the *grey tubercle* is the earliest stage of the deposit, which subsequently becomes converted into *yellow*. Dr. Walshe has also investigated this point, and has taught a similar doctrine. Rokitsansky, in the first edition of his great work *On Morbid Anatomy*, regarded the *yellow* and the *grey* as essentially distinct forms, and considered it an error to believe that the one is converted into the other. He more recently affirmed that *grey tubercle* is

sooner or later converted into *yellow tubercle*, as Laennec first taught, and which is now a common belief. When the grey tubercle does not pass into the yellow, it withers away. It loses its lustre, and becomes dry, dense, and hard, and shrivels into an indistinct, shapeless, fibrous-like mass. Sometimes such a change is associated with a calcareous degeneration, the surrounding tissue in which it is imbedded becoming dark with the deposit of pigment. It is now believed and taught by Rokitansky, Virchow, and others, that certain abnormal products, not apparently at first tubercular, afterwards *tuberculise*, and assume the appearance of yellow tubercles. To this change Virchow proposes to restrict the term "*cheesy metamorphosis*." The metamorphosis of *grey* to *yellow* tubercle generally commences in the centre of the growth, and subsequently the yellow tubercle undergoes most important secondary changes. These changes consist—(1.) in softening, (2.) in cretification or calcification; but it is not to be understood that the first of these two changes always passes into the second. When the scrofulous mass softens its substance breaks down into a tolerably uniform creamy pus-like fluid, in which are to be seen an immense number of fine granular points or molecules. The basis-substance appears to soften first; but in some kinds of softening much of the corpuscular forms are retained in a thin, whey-like, flocculent fluid, approaching in character to what is known as scrofulous pus, both microscopically and to the naked eye. The softening appears, on the whole, due to the breaking up of the corpuscular elements, as well as of the basis-substance. It appears difficult in every instance to ascertain at what part softening commences, so that it is incorrect to give a general description of this change as always commencing in one place. In one case it may begin in the centre and proceed to the circumference of the growth; in another it may begin at the circumference and go round the whole mass, detaching it from the surrounding textures; in a third case it may begin at once in the centre and at the margins; and in other instances it has been observed to commence at the same time in several parts of the substance of the tubercular mass, and this is especially the case where the masses are large (CRAIGIE, BENNETT). The portion or spot about to soften loses its firmness and becomes friable, the cells swell and break up, adding thus by their solution to the diffuency of the mass. Inspected with a common hand lens, the mass seems as if perforated here and there with holes, and this softening extends by degrees throughout the whole growth. The air-vesicles necessarily are destroyed by this process, and the terminal extremities of the bronchi are amongst the first structures to suffer. The mucous membrane which covers them is red and villous in the vicinity of the softened part; and as tuberculosis proceeds, the whole of the pulmonary mucous membrane presents the anatomical characters of chronic bronchitis. The softening of the growth and the inflammation of the textures seem to act and react upon each other; liquefaction seems to extend more rapidly when local inflammation is set up, and the softenings, on the other hand, appear to augment the local reaction.

The "*yellowness*" of what is known as "*crude tubercle*" is in reality a fatty degeneration common to the drying up of many products of common inflammation, such as pus and fibrine. Hence *yellowness* and *cheese-like*

matter (sometimes called *caseous* or *tyromatous*) is no evidence of miliary tubercle, but is evidence of inflammatory products having become inspissated. We are thus justified in regarding *caseation* as anatomical evidence of the *chronicity* of the scrofulous inflammation (RINDFLEISCH). The completion of this cheese-like metamorphosis is favourable rather than otherwise. It establishes a state which tends but little in itself to further changes; and so long as it remains firm and cheese-like *without softening* or establishing surrounding inflammation, it remains a latent lesion, which is a mark, stamp, or evidence of the relative chronicity of the processes which have led up to its existence.

These yellow masses in scrofulous disease have also recently acquired a great pathological interest in connection with the discoveries of Villemain, Cohnheim, and Buhl, that such matters injected into the veins of dogs and other animals produce general tubercle; while Buhl holds that their absorption from the old scar into the blood may create phthisis long after the healing of the scrofula. After such inoculation miliary tubercles were found in various organs, in which the substance introduced appeared in an unencapsuled focus surrounded with mucous or pus, the taking up of which by the lymphatics and blood-vessels appeared to be the condition for the secondary tubercle formation, in from 20 to 25 days after inoculation. Miliary tubercles were thus found chiefly in the following organs—Most often in the lymphatic glands nearest the point of inoculation and the corresponding serous membrane; in the lungs and liver, more rarely in the spleen, kidneys, and intestines. The tubercles so induced by inoculation are similar *microscopically* to the miliary tubercle. The *microscopical* identity is a subject of dispute. Thus the inoculation experiments of Villemain, Burdon-Sanderson, Wilson Fox, and others, have shown—(1.) That the character of scrofulous inflammations ending in tyromata is infective under certain circumstances, and so capable of starting or originating tubercle alike in the lungs and in other parts of the body, however distant from the *foci* of infection. (2.) That the softening and absorption of the cheese-like matter wherever situated, furnish such infective matter for absorption. So also does the putrefaction of pus of diseased bones, hence the frequency of lung destruction after caries and necrosis of bones. The *milk* of tuberculous animals is also believed to generate tuberculosis in those nourished by it. It is difficult, however, in all cases to reconcile the older pathology with the views now gaining ground, especially as to the real significance of the cheese-like, tyromatous matter, which is the real lesion in scrofula, which has long been known as crude tubercle, and which is the starting-point of the infective process whence true miliary tubercle is believed to spread. These cheese-like masses of crude tubercle ought now to be distinguished from the young "miliary tubercle" of adenoid or lymphoid tissue. At certain stages in the process of softening of these yellow masses, infective properties seem to be acquired by it. The infection is transmitted mainly by the lymphatics, at the extremities of which true miliary tubercles are developed from the lymphatics themselves.

Thus true miliary tuberculosis (scrofula with tubercle) is a specific reabsorption and infective disease process, observing the following sequence of phenomena:—(1.) The basis for the development of the miliary tubercle is a *cheesy* (scrofulous) *focus*, owing its origin to previous scrofulous

inflammations; the elements of the breaking up of which, being taken into the blood and lymphatic vessels, give rise to the multiple development of true tubercle (*self-infection*). One or many of these *scrofulous cheesy foci* may be found in the body. (2.) The infectious material of the *scrofulous cheesy foci*, upon which tuberculosis (*i.e.*, general development of miliary tubercle) follows, contains something specific. (3.) The infective process probably takes place through the lymphatic vessels or plasmic canals. (4.) Tubercles (true miliary) always appear in the largest accumulations in the immediate vicinity of the infecting focus, and spread thence by intervals in eccentric areas—*e.g.*, old intestinal ulcers, cheesy carious vertebrae, cheesy bronchial glands, and in cheesy masses in the lungs. (5.) The occurrence of similarly large and fresh miliary tubercle in many or almost in all organs and tissues at the same time (*general infection*). This general infection is most frequent under 24 years of age—*i.e.*, during the growth of the body, when lymph and blood development are most active.

The naked eye is but of little use in recognising what is true miliary tubercle and what is not. Tubercle can only be distinguished from scrofulous masses when it appears in its miliary form. The true tubercle structure is essentially a microscopic object. It is a lymphadenoma, best seen in such lymphatic glands as contain one, two, or three roundish spots composed of exquisitely typical cytogenous tissue. In the centre of these nodules there is constantly a many-nucleated giant cell, with numerous processes, and separated from surrounding parts by a concentric ring of connective tissue. This cytogenous tissue is distinguished from that of the lymph gland substance, in that the nuclei and cells within the reticulum are larger, that they are easily isolated, and that in the neighbourhood a giant cell is always present. The reticulum itself has also a more cell-like structure (SCHÜPPEL, WAGNER). "Tubercle itself, so far as it has any structure, has a coarse resemblance to the simple structure of the lymphatic glands" (MOXON and WILKS). In the lungs, pleura, liver, kidney, spleen, exactly the same structure of true miliary tubercle prevails. It occurs mostly in organs altered by inflammatory action, especially where there is a local scrofulous or caseous source of infection, or necrosis of parts. Buhl and Klein's descriptions coincide, and now it seems certain that there is a definite structure invariably met with in true miliary tubercle—not the presence of any one particular cell-form, but a definite arrangement of various elements into a system of cells and reticulation of fibres; and several of these being in close contiguity may form a tubercle nodule, visible (and only just visible) to the naked eye, of a grey gelatinous appearance. In all these growths there is a tendency to the formation of a concentric band of fibrous tissue at the periphery of each cell-system, hence the rounded form of the nodule. Although such a nodule, being a combination of cell-systems, is visible to the unaided eye, there are numbers of tubercles of the most typical construction which are quite invisible, and which are overlooked if a microscope is not used to look for them. Hence we can never say with certainty whether any nodule is made up of miliary tubercle, nor whether any organ of the body contains tubercle, until a systematic and minute examination has been made of it microscopically. This I have found to be the case, especially with regard to the liver, and such solid

organs in which miliary tubercle in this minute form is often to be found, when the organ is examined microscopically. It must therefore be remembered that *miliary tubercle* is a microscopic object; it is not capable of being recognised as *such* by the unaided eye. The most recent observers are now agreed that several giant cells form an integral and most important part in the structure of a tubercle. They are not identical with the myeloid cells of myeloid tumors, nor those of the marrow of bones; but they are surrounded by processes which run outwards, to divide and subdivide so as to form the branching reticulum which eventually joins with the surrounding concentric fibrous band. The processes of the cells in this way construct the reticulum. These processes are always to be seen where the tubercle has not undergone degeneration. It is too late to look for them in broken up and degenerate accumulations in the lungs which have become caseous. The nature of these giant cells is, however, the subject at present of much dispute. Hence next comes the question, "How do these giant cell-systems first arise?" Virchow says, from the cells of connective tissue. Rokitsansky described them as mother cells; and both these observers describe them in the peritoneum, where they are best examined on the serous covering of the bowel opposite those ulcerations we are accustomed to describe as tuberculous. Others describe the beginnings of these cells by subdivision of one, or running together of several, where cell structures are abundant (LANGHAMS). Their nuclei are particularly clear and vesicular. Others, again, represent them as formed from capillary lymphatic vessels (KLEBS). Others, that they form within minute blood-vessels by the running together of several colourless blood corpuscles; and as soon as nuclei appear in them the wall of the blood-vessel disappears (ZEIGLER). Several recent observers adopt this view (SCHÜPPEL, THAON, WAGNER, RUSTITSKY). Henry writes that the giant cell is simply a lymphatic cut across. Those who do not adopt this view, conclude that at any rate the giant cell-formation is in close relationship with the changes going on in the blood-vessels of the part; and if the cells do not take their origin from the walls of blood-vessels whose structure is complete, they do so from the germs of newly developing vessels; and are sometimes connected to vessels by long protoplasmic processes, similar in character to the mode of formation of young blood-vessels. Hence the belief that they are really developments by germination of the nuclei in the vascular walls of the minute capillaries, or from the nuclei of lymphatics. Such giant cells enter largely into the formation of young blood-vessels, and also into the granulation tissue of granulating ulcers and wounds; more especially are they large and obvious in those of a scrofulous nature (BUHL). They are not found in the pyogenic layer of the sore, but in the deeper part—the protoplasmic area—where they are abundant in healthy granulating wounds, not less than in those which are scrofulous. Klein and others consider that it is the offspring of the alveolar epithelium, and this seems most probable from the position of the cell and the possibility of its isolation. But wherever found, these giant cells *alone* never constitute true tubercle. The additional branching processes from the cells forming a reticulum, inclosing lymphoid and epithelioid cells in its meshes; and that again anastomosing at the periphery in such a way as to form a surrounding fibrous ring, constitute the

essential anatomical construction of a true miliary tubercle. The conclusions which have been arrived at regarding tubercle have been summed up by Dr. Burdon-Sanderson as follows:—

(1.) The characteristic product of tuberculosis is not a mere aggregation of shrivelled particles of irregular form, but a distinct tissue composed of lymph corpuscles, held together by a network of hyaline connective tissue, to which the name of *adenoid* has been given. (2.) There is a close structural analogy between this tissue and that of certain follicular organs belonging to the lymphatic system, such as the follicles of Peyer and the ampullæ of the lymphatic glands. (3.) All the favourite sites of tubercle are naturally characterised by the presence of this tissue, which from the analogy just stated may properly be called *adenoid*.* (4.) It is important also to remember that the natural distribution of this *adenoid* tissue in the body is in intimate relation with the lymphatic system. In all the great serous membranes it forms sheaths round the blood-vessels, or masses of microscopical dimensions and of irregular contour underneath the epithelium. In the solid viscera it is distributed here and there in the course of the lymphatic channels. (5.) In the peritoneum, tuberculosis primarily consists in the enlargement or overgrowth of these sheaths or microscopical masses of *adenoid tissue*; and consequently the tuberculous nodules which form there have the same intimate structure, standing in the same anatomical relation to the vessels and the epithelium. In the solid viscera, the essential lesion also consists, not in any new growth, but in the overgrowth of pre-existing masses of *adenoid tissue*. (6.) When tuberculosis is set up artificially, whether the cause be simply a wound or specific inoculation, the primary local lesion consists in the development at the seat of injury of granulations or nodules which have similar structural characters with those of *adenoid tissue* elsewhere, but cannot as yet be shown to be in relation with the absorbent vessels. (7.) The first step in the dissemination of tubercle consists in its being absorbed primarily by the lymphatics (which convey it to the lymphatic glands, of which they are tributaries), and secondarily by the veins. Thus it enters the systemic circulation, and is universally distributed by the arteries. The serous membranes seem, however, by preference to appropriate it, and from them it extends by contiguity to the superficial parts of the organs which they cover. (8.) The final stage of the process of tuberculosis consists in the tertiary infection of the glands of each diseased organ. These glands undergo enlargement and induration, and eventually become partly caseous. This enlargement is due to the multiplication of cells in all the tissues of the organ, but more particularly in the alveoli. The hardening is due to a process of fibrous degeneration, while the caseation is due to the slow necrosis of the previously hardened and anæmic parts. From the first commencement of this enlargement the gland is incapable of performing its functions; but it is not till induration commences that the absorbents of the organ to which it belongs are completely obstructed. (9.) As regards the question of a

* It must at the same time be remembered that this word "*adenoid*" is now used in two senses—(a.) In the anatomical sense in which I have now used it: and which is described in the recent editions of Quain's *Anatomy* in the special part written by Dr. Sharpey; (b.) In pathological nomenclature it has been introduced in an entirely different sense. It has been applied to certain tumors which consist essentially in overgrowths of elements belonging not to *adenoid* but to *acinous glands*—e.g., of the epithelium of the liver and mammary glands respectively.

specific contagium of tubercle, it is important to bear in mind that the facts of traumatic tuberculosis do not as yet disprove the existence of such a contagium. It still remains open to inquiry whether or not injuries which are of such a nature that air is completely excluded from contact with the injured part, are capable of originating a tuberculous process.

A considerable portion of the scrofulous matter may transude in a fluid state, in the first instance, through the capillaries, and collect in those places outside the vessels that offer least resistance (BENNETT). While, therefore, *infiltration* more or less extensive, as described by Baillie, is the first condition in which the scrofulous matter can be observed to exist, crude yellow *tubercle* may be observed to accumulate on the free surfaces of mucous and serous membranes, or on the outer surface of minute blood-vessels. While, therefore, scrofulous material does not differ in its seat from the simple or cancerous exudations, when these form minute growths in the lungs or other parts, yet there is an important difference in regard to their relation with the blood-vessels, which requires to be specially noticed. It was first pointed out by Dr. William Stark that when attempts are made to inject a tuberculated lung, the finest injection will not reach, far less penetrate, the crude yellow *tubercle-masses*. Blood-vessels which are of a considerable size at a little distance from such masses speedily become contracted; so that a large vessel, which at its origin measured nearly half an inch in circumference, could not be cut open farther than one inch; and when cut open, such vessels presented a very small canal, filled by a coagulated substance. Schroeder Van der Kolk made similar experiments. He sometimes found that large vessels remained pervious which crossed a tuberculous cavity in a lung, but that all the small or capillary branches which adhered and were given off from the larger trunks were obstructed and impervious. These observations lead to the conclusion that obliteration commences in the smaller vessels and proceeds to the larger trunks, and are of some importance with reference to hæmoptysis and to the process of softening, and other changes which are observed to take place in this *non-vascular* growth. Cancerous growths, on the other hand, are highly organised, containing large, voluminous, and generally multiform elements (see p. 962, *ante*).

Thus much is known regarding the histology of scrofulous or tuberculous growths, and of their results in the development of *true miliary tubercle*. These results are here stated minutely, because we have the testimony of most experienced morbid anatomists, such as Bennett, Paget, and others, that the microscope alone can decide as to the nature of those growths which closely resemble tubercle when examined by the naked eye. Indeed, such are the vague definitions given of *tubercle*, that, in the words of Bennett, "every morbid anatomist must frequently have experienced much difficulty in endeavouring to determine by the naked sight whether a certain morbid product be or be not tubercle."

The term "tubercular deposit" can scarcely be regarded as correct, although in common use. The expression ought rather to be "*scrofulous or tuberculous matter*." There is this constant difference between scrofulous crude or yellow tubercle and true miliary tubercle, that the uniform cheesy change of scrofula, is never secondary to any similar change in other

parts, while true miliary tubercles are secondary. *True miliary tubercle is the secondary form of the disease of which scrofula is the primary form* (WILKS, MOXON).

II. *Local Scrofulous Lesions*.—(1.) In the COMMON INTEGUMENTS there are three species of *porrigo*, namely:—*favosa*, *larvalis*, and *furfurans*, together with *acne indurata*, *eczema impetiginodes*, and *rubrum*, in their chronic forms, which are usually considered strumous diseases. They are eruptions which are found in combination with the usual symptoms of scrofula; and when they affect the scalp, ears, and face, they are apt to occasion swelling of the glands at the back part of the head, on the sides of the neck, and under the jaw. The swelling of the glands arises first from simple irritation, or the absorption of morbid fluids; or the swelling becomes permanent from the deposition of scrofulous matter; and where there is the hereditary predisposition to scrofula, the irritation of dentition or exposure to cold will induce such swelling of the glands as will end in the deposition of the same curdy substance. *Lupus* commencing by the slow development of a tubercular growth in the tissue of the true skin or mucous membrane, characterised by infective cell-growths, appears to be a truly scrofulous disease (MCALL ANDERSON, JONATHAN HUTCHINSON, STOWERS). It affects the lips, nose, or genitals. (2.) Swellings in the SUBCUTANEOUS AREOLAR TISSUE (adventitious glands of Wiseman) are soft and puffy, and manifest but little or no disposition to suppurate. They arise suddenly, without pain or discoloration, and are due to fluid effusion. They greatly resemble glandular swellings; but they are really cysts having a firm inner lining of coagulated lymph of a fibrinous appearance, filled with scrofulous substance in a crude state or as curdy pus. If not so encysted, the fluid forms a species of abscess—cold abscess—*abcès froids* of the French—generally found in the interstitial muscular spaces or under the fasciæ. Their contents are at first serous; then seropurulent, intermingled with curdy flakes; more rarely they consist of a thin pus. Sloughing of the areolar tissue is not uncommon, spongy masses of it, like wet tow, coming away along with the discharge or plugging up the abscess opening. This is usually the nature of the abscesses surrounding scrofulous joints (Drs. CUMIN and SHAPTER). Another scrofulous lesion of the skin is that where a portion of it assumes a livid colour, becoming hard and stiff, and so continuing for many months; and underneath a portion of areolar tissue dies. Pus forms around this dead portion, and the slough, as on a boil, is only set free by an opening being made through the hard skin. (3.) The MUCOUS MEMBRANES are a very frequent seat of the scrofulous lesions,—(a.) Especially at the junction of the skin with the mucous membranes the scrofulous lesions appear, as at the nose, where the mucous membrane becomes inflamed, and discharges a thin acrid sanguineous matter, which excoriates the external surface. The *alæ*, the *septum*, and the upper lip become swollen, and a chap often appears in the middle of it. The patient is obliged to breathe in a great measure through the mouth, so that the constantly half open mouth becomes almost characteristic of scrofula. The odour exhaled is often so offensive as to be disagreeable to all save the sufferer himself, who is prevented from being sensible of it from the destruction of the mucous membrane within the nose. The mucous membrane of the eye and

lachrymal passages are also frequently the seat of scrofulous ophthalmia. In the *mucous membrane* of the alimentary canal the seat of scrofulous inflammation is principally in the lower part of the small intestines. The scrofulous mass grows originally in the submucous tissue, and elevates the mucous membrane stretched over the granulations. They project in the form of nodules assembled together in several groups. New granulations spring up between the older ones; and as these growths degenerate and soften rapidly in the intestines, a fusion of the whole very soon takes place. The circulation in this part of the intestine is impeded, and its vitality is gradually destroyed; its mucous membrane is lost, and ulcerations form, whose shapes are decided by the form of the original growth. Thus,—A single tubercle-nodule gives a crater-like ulcer. A number produces extensive ulceration, with irregularly excavated borders, the growth of the tubercle-masses being continued at the margins of the ulcers; and hence the thick border, with particles of yellow crude tubercle imbedded in it, so characteristic of these ulcers. Similar material may also be seen on the floor of the ulcers, the softening of which leads to perforation of the peritoneum. (4.) The SEROUS MEMBRANES.—In the PERITONEUM the seat of scrofulous inflammation is immediately beneath the serous coat, where sanguineous congestions and minute extravasations first take place during the rapid formation of the tubercular granulations. So also the pleura, the arachnoid, and the pericardium may each be the seat of scrofulous lesions. (5.) The LYMPHATIC GLANDS have long been regarded as the special seat of scrofulous deposit. Their enlargement and induration are still considered as more unequivocal evidence of scrofula than any other sign, except the actual discharge of scrofulous matter. When first affected they are soft, fleshy, with an elastic feeling, and increased in size; afterwards they become firmer and the colour paler than in health. The glands continue to enlarge by irritative hypertrophy to three, four, or even six times their natural size. There is an increase of the lymphoid cellular structure, causing at first a slightly translucent swelling, but with a dryness which characterises scrofula. This dryness signifies the dense aggregation of the new cell-growth in the gland and its want of freedom, so that it compactly compresses its own vessels, thus leading to gradual loss of vitality and caseation. The caseous change shows itself by all the elements of the texture gradually losing their sharpness of outline, and wasting into fatty remains. These fatty relics may be entirely absorbed, which is a rare but most favourable end. More often the caseous tissue breaks down into a curdy matter, which may dry into a pasty remainder, and so become calcareous, especially in the mesenteric and bronchial glands, where the stony material in long intervals afterwards may undergo enlargement by accretion, until a mass of stone as large as a walnut or larger may be found (ABERCROMBIE, CRAIGIE, WILKS, and MOXON). The glands may retain their shape, but they may become irregular on the surface, lobulated, and generally projected in the free or unattached direction. On section they are of a cream-yellow, grey, or whitish-grey colour; and like soft cheese as to consistence, but pretty firm and resisting (*tyroma*). It is believed to be effused in a fluid or semifluid form at different parts of the gland, which gradually become more consistent. In its most fluid form it is like thick

cream or like chalk and water; or it may be like soft putty; or it may be made up of consistent granules diffused in a whey-like or milk-like fluid. It is most common in the bronchial lymphatic glands, in the mesenteric lymphatic glands, and in the lymphatic glands of the neck, especially the sub-maxillary and sub-lingual (CRAIGIE). Tumefaction of the tonsils is seldom absent. They become very liable to inflammation, to aphthous sores and ulcerations. (6.) In other glands, such as the KIDNEYS, *scrofulous inflammation* constitutes a primary and fatal disease, sometimes called *strumous pyelitis*. The leading symptom is a large amount of pus in the urine. It is of slow progress; and after some years the organ becomes quite destroyed. Large hollow spaces exist in one or both kidneys, with only a trace of the secreting tissue between them. The mucous membrane is thickened, covered with scrofulous matter, and beneath it there is the same matter running into the tissue. (7.) In the BRAIN it occurs in two forms—(a.) As one, two, or more homogeneous individual masses of considerable size; and (b.) several, sometimes many, minute spheroidal bodies separate from each other. In general they are surrounded by a vascular cyst of variable thickness. It is generally so found in early life—between nineteen months and twelve years—a chronic disease, causing but few symptoms (CRAIGIE).

For description of scrofulous ulcers, scrofulous lesions of bones and joints, the reader must refer to works on surgery.

Symptoms of Scrofula, and of the Cachexia which precedes and accompanies the Growth of Tubercle.—It is *the constitutional state* previous to the actual occurrence of local scrofulous affections like tubercle which it is of the utmost importance, if possible, to recognise; and the question has been proposed, “Whether or not a predisposition to scrofula can be anatomically or otherwise demonstrated?” There can be no doubt that an assemblage of progressive phenomena, more or less well expressed and obvious, are characteristic of the scrofulous constitution. They are generally also more pronounced when the disease is of hereditary origin than when it is acquired in after life. These phenomena are mainly due to impaired nutrition of the fundamental parts of the frame, associated with imperfect growth, especially of the skeleton, the nervous system, and the intestinal tract—imperfections capable of hereditary transmission to a remarkable extent. Of the structural characteristics of scrofulous persons, none are so significant as the weakness of cell-growth apparent in imperfections of the areolar tissue, as seen in the smooth, soft, delicate, blanched, and transparent skin. It becomes sallow in the dark complexioned; while in the fair it assumes an appearance not unlike blanched wax. The nails are generally defective, elongated, and remarkably incurved. The tips of the fingers are square and flat, usually described as being clubbed. The mucous membranes are thin, pale, delicate, attenuated, and easily expanded. The vascular system is thin, the vessels are transparent, more distensible and less contractile than in the robust and healthy; and the visible tenuity of the vascular coats is sometimes rendered apparent in the tendency to hæmorrhages from slight causes. The veins are conspicuous, and appear to be more numerous under the skin. The bones are imperfectly grown; and there is often a greater want of symmetry than usual in the skeleton. The weight of the body is light compared with size and age. On these

points in health there ought to be a due concurrence. So important is the information capable of being derived from accurate observations in this direction, that the following tables are given for the purposes of comparison. Growth expressed by stature and weight, is thus most marked between the ages of fourteen and sixteen years. Its rate is as much as three inches in height during that time; and about ten inches from the age of eleven to eighteen; and it goes on to be marked between eighteen and twenty-five years of age, to the extent of about two inches. There are also ample physio-

MEAN CORRELATION OF PHYSICAL QUALITIES INDICATING THE "TYPICAL MAN" OF THE LABOURING CLASSES FROM 14 TO 30 YEARS OF AGE
(Based on Roberts' Tables).

Age.	Height.	Annual mean growth.	Weight.	Annual mean growth.	Girth.	Annual Increase.
Years.	Inches.	Inches.	Lbs.	Lbs.	Inches.	Inches.
14	56·5	2	80	8·57	28	1·5
15	58·5	2	91	11	29·5	1·5
16	61	2·5	107	16	31·	1·5
17	63	2	118	11	32·5	1·5
18	64	1	127	9	34·	1·5
19	65	1	131	4	34·5	·5
20	65·5	·5	133	2	35·	·5
21	65·5	...	135	2	35·5	·5
22	65·5	...	136	1	35·5	...
23	66	·5	137	1	35·5	.
24	66	...	137	...	35·5	...
25 to 30	66·5	·5	137	...	35·5	...

ANALYSIS OF MR. MACLAREN'S ANNUAL MEASUREMENTS OF 168 BOYS
AT THE GYMNASIA OF THE MAGDALEN COLLEGE SCHOOL AND THE
ST. PETER'S COLLEGE, RADLEY, OXFORD, IN 1862 AND 1863.

Number of Observations.	Year of age.	STATURE.		WEIGHT.		Increase of Stature during the Year.	Increase of Weight during the Year.
		Shortest.	Tallest.	Lightest.	Heaviest.	Average.	Average.
		Ft. In.	Ft. In.	St. Lbs.	St. Lbs.	Inches.	Lbs.
5	9-10	4 0	4 10 $\frac{1}{4}$	3 11	4 7	·5	4
7	10-11	4 3 $\frac{1}{2}$	4 7	4 6	5 11	·8	3·7
14	11-12	4 3 $\frac{1}{2}$	4 11 $\frac{3}{8}$	4 2	6 5	1·1	5
28	12-13	4 4 $\frac{3}{8}$	5 5 $\frac{5}{8}$	4 8	7 11	1·6	5·9
28	13-14	4 5 $\frac{3}{8}$	5 7 $\frac{3}{4}$	4 9	9 10	2·2	7·9
23	*14-15	4 5 $\frac{3}{8}$	5 7 $\frac{1}{2}$	4 5	9 6	2·5*	8·1
33	+15-16	5 0 $\frac{1}{2}$	6 0	5 8	10 4	2·2	10·6+
19	16-17	4 8 $\frac{1}{2}$	5 10 $\frac{1}{2}$	5 10	10 10	1·3	8·3
7	17-18	5 3 $\frac{1}{2}$	6 0 $\frac{1}{4}$	7 13	11 8	·6	5·5
4	18-19	5 5 $\frac{1}{2}$	5 6 $\frac{1}{2}$	8 8	9 10	·5	5

* Year of greatest increase in stature. + Year of greatest increase in weight.

HEIGHT AND CIRCUMFERENCE OF CHEST OF 1,270 YOUNG PERSONS,
RECORDED BY DR. HARRISON OF PRESTON (*Edin. Med. and Surg.*
Journ., 1835, p. 425).

Age.	Number Examined.	HEIGHT.			CIRCUMFERENCE.		
		Average.	Tallest.	Lowest.	Average.	Great.	Small.
		Ft. In.	Ft. In.	Ft. In.	Inches.	Inches.	Inches.
11-12	210	4 2 $\frac{3}{4}$	4 11	3 10 $\frac{1}{2}$	25 $\frac{1}{2}$	30	22
12-13	203	4 5	5 0	3 11	26	29	21
13-14	192	4 6 $\frac{1}{2}$	5 3	4 0	26	32	23
14-15	197	4 7	5 3	4 0	26 $\frac{1}{2}$	32	21
15-16	186	4 10	5 6	4 1	27 $\frac{1}{2}$	32	23
16-17	131	5 0 $\frac{3}{4}$	5 7	4 4	28	34	24
17-18	151	5 0 $\frac{3}{4}$	5 7	4 3	27 $\frac{1}{2}$	34	23

logical reasons of the most cogent kind which clearly show that great care is necessary in the physical training of young persons.* The main reasons are to be found in the correlations which obtain amongst the following elements, and which ought to be maintained at the healthiest standard, namely,—(1.) Age; (2.) Weight; (3.) Stature; (4.) Development and growth of the skeleton; (5.) The vital capacity of the chest; (6.) The growth of the muscles in relation to the bones; and the progressive increase of muscular force with advance of years.

There are individual peculiarities connected with the skeleton which at once ought to excite suspicion and suggest careful training. These are,—(1.) Narrowness of the thorax, especially at the expansion of the false ribs—a condition, when combined with functional incapacity or inefficiency in the acts of respiration, which is of most characteristic significance. The vital capacity of the lungs, as determined by (Hutchinson's) spirometer, compared with age, weight, and height, ought to be observed and recorded in all physical examinations. Hutchinson's observations show that men from five to six feet high have a vital capacity of lung ranging from 174 to 262 cubic inches, in an ascending scale, according to height and age; and whenever the quantity of air is 16 per cent. deficient, there is reason to suspect some local affection of the chest. Dr. Graham Balfour has subsequently followed up these investigations with the spirometer. He has especially examined how far a capacity

* I. Compare the average height of *full-grown* men at twenty-five years of age, as given by the following observers:—

Quetelet, . . .	5 feet 5·27 inches	= 65·27 inches.	} Average, 66·37.
Danson, . . .	5 „ 6·30	„ = 66·30 „	
Boyd, . . .	5 „ 7	„ = 67 „	
Liharzik, . . .	5 „ 8·89	„ = 68·8 „	
Roberts, . . .	5 „ 6·5	„ = 66·5 „	

II. The average height of the *growing lad* at eighteen years of age:—

Boyd, . . .	5 feet 0·5 inches	= 60·5 inches.	} Average, 63·35.
Liharzik, . . .	5 „ 4·17	„ = 64·17 „	
Danson, . . .	5 „ 4·34	„ = 64·34 „	
Roberts, . . .	5 „ 4	„ = 64 „	

under the average may be taken as an indication either of a tendency to pulmonary disease or of a feeble constitution, rendering such men liable to a higher rate of mortality than that to which men of or above the average are subject. He found that the loss to the British Army by consumption was much greater among the men having a "*vital capacity*" *under the average* than amongst men of *average capacity* or *above it*; and although the proportion of deaths did not differ materially amongst those three classes, yet the invaliding was *four times as high* among men *under the average* as among the others. A "*vital capacity*" *below the average* may therefore be considered as indicating a generally feeble organisation, less capable of resisting the deteriorating influences to which a soldier is exposed ("Contributions to the Study of Spirometry," *Med.-Chir. Transactions*, Vol. XLVIII.) Such functional incapacity is further indicated by the "breathing being shorter," with less "breath motion." The *Expiration* is quick and forcible; and there is a minimum quantity of air taken in by ordinary *Inspiration*. Such lessened respiration tends of itself to induce accumulation of mucous in the air-cells, and thereby to set up inflammation. Everything which tends to impede or to interrupt or obstruct the regular, complete, and continuous performance of the respiratory acts has a most prejudicial effect upon the lungs (especially of "growing lads"), favouring the accumulation of growing material in the air-cells, which may eventually degenerate and form a cheesy mass in all respects resembling tubercle. Life not only depends on breathing, but the energy and the vigour of life are in a great measure ruled by the capacity and the free play of the breathing organs (SIBSON, 1844).

Ever since scrofula has become more studied as a constitutional affection, it has been rendered more apparent that impairment of the digestive organs is the primary disorder of function which ushers in the cachexia, and that a certain form of dyspepsia is not only present in the hereditary strumous constitution, "but is capable of generating the bad habit of body, and of leading ultimately to the deposition of tubercle." It was reserved for the late Dr. Tweedy Todd, to show that in cases in which the strumous cachexia was present a particular form of dyspepsia prevailed, to which he gave the name of "strumous dyspepsia." Subsequently the views of Dr. T. Todd were more or less fully accepted and developed by Sir James Clark, Bennett, Ancell, and others in this country; but the most precise exponent of them has been Mr. Jonathan Hutchinson, in two valuable papers in *The Medical Times and Gazette*, Vol. X., 1855. He there shows from statistical evidence, that the tuberculous cachexia is preceded by a *peculiar form of dyspepsia*; in other words, that there is a peculiar form of dyspepsia which has a natural tendency to end in scrofulous phthisis; and that it is a form of dyspepsia not common to other conditions of the system—different, for instance, from that which precedes and attends gout, chlorosis, or cancer, although accurate observation is wanting on the exact symptoms of dyspepsia which are associated with these diseases. The conclusions which have been arrived at by Mr. Hutchinson are as follow:—(1.) In a very large majority of cases of established phthisis a condition of well-marked dyspepsia is present as a complication. Out of fifty-six cases it was absent in four, present

mildly in twenty-one, moderately in twenty-two, and severely in nine. (2.) Of the form of dyspepsia most common in established phthisis, the prominent symptoms relate to difficulty in the assimilation of fatty matters. The patient acquires a remarkable distaste for all fats, which occasionally extends itself to sugar and even to alcohol; he suffers much from "biliousness," heartburn, flatulence, and, above all, from acid eructations after taking food; everything he takes "rises acid," to use a common expression of such patients, but more particularly everything containing fat, oil, or sugar (*butyric* fermentation). The *dislike for fat* was present in 71 per cent. of the cases of confirmed phthisis, and had existed through life in 48 per cent. It invariably produced what is commonly called "biliousness," and "rose acid" from the stomach. The fat of fresh meat was generally the first to disagree, then salted meats, such as bacon, and lastly butter; but many could enjoy butter who could not touch any other kind of animal fat. Such an intense dislike to an important element of a mixed diet indicates a deficiency on the part of a patient to digest it. Coincident with the development of such symptoms *emaciation* usually becomes apparent, the adipose tissue of the body already existing being reabsorbed by the blood to supply the respiratory element deficient in that fluid. The fat disappears from the subcutaneous tissue, hollows of the cheeks, orbits, and mammary glands, and other parts where it generally abounds in quantity, just as it disappears from the body of an animal during hibernation. The patient now gradually loses weight and becomes thinner. Other tissues of the body, such as the muscles, skin, and areolar tissue, subsequently begin to be used up for a similar purpose, till the patient is wasted to a living skeleton. The *acid eructations* were present in 62 per cent. of the cases, and were a prominent symptom in 46 per cent., small quantities of an extremely acid fluid being repeatedly brought up into the pharynx at various periods after taking food, and in bad cases these eructations were persistent throughout most of the day, and almost always attended by heartburn. *Sick headaches* and *biliousness* were very commonly present. Dr. Pollock's experience shows that *acidity* of digestion has only a constant relation to the acute form of the disease. Of that form it is a distinctive character. Laennec, Louis, and Pollock all record diseased states of the stomach in *post-mortem* examination of phthisical patients. (3.) The majority of cases of *phthisis pulmonalis*, whether hereditary or otherwise, are *preceded* by a stage of dyspeptic symptoms. By this it is meant that symptoms referable to the digestive organs have preceded those connected with the lungs. Out of the fifty-two cases in which, as has been shown, dyspepsia was present, it had followed pectoral symptoms in nine, was developed nearly coincidently with them in ten, and preceded them in thirty-three. (4.) The symptoms of the dyspepsia premonitory of phthisis are the same in character with those which complicate it when developed. The very earliest are alterations in the tastes, and the most constant of all is a disrelish for fat. (5.) The subjects of phthisis have in a large number of cases had peculiarities of likes and dislikes for different articles of food even from very early life, and whilst seemingly in perfect health. Amongst those peculiarities the dislike of fat, often amounting to extreme aversion, ranks first.

Thus it might be predicated of a family in which one child distinguishes itself from its brothers and sisters by constant refusal to eat fat, that such a child will, *cæteris paribus*, be more prone to become the subject of tuberculous disease in after life than any of the others.

Besides the indications afforded by the functions of digestion, there is a peculiar modification of the whole organisation, as regards structure, form, and the exercise of functions generally, which impresses distinctive characters on the scrofulous cachexia, especially when it is of hereditary origin. That there is a general temperament or diathesis proper to it there can be no doubt, the general characters of which have been already noticed. The constitution is described as scrofulous; and in many respects it conforms to the *sanguineous*, as well as to the *melancholic* or *atrabilious*, and to the *lymphatic* or *phlegmatic* temperaments of the older writers. It is thus evident that no particular temperament nor complexion can strictly be called *scrofulous*. Nevertheless there are certain anatomical characteristics which undoubtedly indicate an inherent predisposition to the disease. These may be summed up as follow:—The complexion is fair, and frequently beautiful, as well as the features. The form, though delicate, is often graceful. The skin is thin, of fine texture; and subcutaneous blue veins are numerous, shining very distinctly through the otherwise pearly-white integument. The pupils are usually very open, and the eyeballs are not only large but prominent, the sclerotic showing a lustrous whiteness. The eyelashes are long and graceful—unless *ophthalmia tarsi* exists, as not unfrequently is the case; then the eyelashes are wanting, and their place is occupied by the swollen, red, unseemly margin of the lid. In the phlegmatic form the complexion is dark, the features disagreeable, the countenance and aspect altogether forbidding, the joints large and rounded, the general frame stunted in growth, or otherwise deformed from its fair proportions. The skin is thick and sallow; the eyes are dull, though usually both large and prominent; the general expression is heavy and listless; yet not unfrequently the intellectual powers are remarkably acute, as well as capable of much and sustained exertion. The upper lip is usually tumid, and often chapped in the centre. The *columna* and *alæ* of the nose, are also tumefied, and the general character of the face is flabby; the belly inclines to protuberance; and the extremities of the fingers are square or flatly clubbed, instead of the tapering form. A third characteristic type is marked by an unusually large head, coarse features, a thick chin, swollen abdomen, enlarged cervical glands, and flabby, spongy flesh. The skin is of remarkable whiteness, with a tendency to redden easily; and through it the rose-pink or bluish subcutaneous veins are visible; there is a deep redness of the cheeks and lips, blueness of the thin and transparent sclerotic, which imparts a swimming and languishing look to the eyes. The muscles of such persons are thin and soft, and their weight is light in proportion to their stature, indicating a slightness of their bones. The teeth are handsome and of a bluish lustre, or of a pearly-whiteness and tending to early decay, though long and narrow; and the hair is soft (CUMIN, SHAPTER, MILLER, CONSTATT).

The growth of the body is generally unsteady in its progress; very often it is slowly and imperfectly developed; in other cases it is unusually rapid, particularly towards puberty. The physical powers are generally

feebly developed, and incapable of sustained exercise. The muscles of the limbs, though full, are soft, flabby, and weak, and have neither the form nor the firmness of health. The general circulation is feeble, the weak pulse and cold extremities indicating the debility which is rarely absent in the scrofulous constitution, and which affords an explanation of many of the phenomena of the disease. Digestion is feebly and imperfectly performed, the bowels being irregular, and more frequently slow in their action than the reverse. The mucous membranes generally are very susceptible to disordered action. Discharges from the nose, ears, or eyes are not uncommon; the tonsils enlarge, and the air passages inflame from the slightest causes. The insensible perspiration is defective, and is said to be unduly acid, and loaded with sebaceous matter; while, on the other hand, copious partial perspirations are common, particularly over the feet, where the odour is often fetid. In children, the impaired functions of the digestive organs are indicated by increased redness of the tongue, especially toward the extremity and along the margins. The anterior part is thickly spotted with small red points of a still brighter colour, the central portion being more or less furred, of a white or of a brown colour, in the morning. Thirst prevails; the appetite is variable, more frequently craving than deficient, seldom natural; and the breath is fetid. The bowels are occasionally loose, and the evacuations are always unnatural, generally of a pale greyish colour, of the consistence and appearance of moist clay; and they are often mixed with mucus and partially digested food. While the urine is often turbid, and sometimes high coloured, it is also often abundant and pale. The skin is generally harsh and dry, or subject to cold perspirations, particularly the hands and feet, which are habitually cold; and copious partial night sweats are common. The skin is also subject to scaly or furfuraceous eruptions. The sleep is seldom sound; the child is restless, talks in his sleep, or grinds his teeth (T. TODD, Sir JAMES CLARK). Subsequently, when the disordered state has continued for some time, the internal fauces become full and red, and inflammatory sore throats are common, the tonsils often becoming permanently enlarged. The nostrils are generally dry, or thick mucus may be discharged in large quantity. Epistaxis occasionally occurs. The nervous sensibility is sometimes greatly increased, and the intellectual functions are often performed with a preternatural degree of activity. So frequently is this observed, that it has become a popular belief and saying regarding such a child, "that it may be too wise to live long." Inflammation in any tissue in a scrofulous subject generally assumes a slow chronic type, accompanied with little pain or heat; and suppurating parts heal very slowly. The skin is easily irritated, and wounds made in it have a tendency to fester, rather than to heal.

Frequent observations, extended over a considerable interval of time, are necessary to appreciate the wasting of delicate tissues which occurs in scrofula when it becomes active or acute, and progresses so insidiously, and creeps on almost imperceptibly, but with a result so sure "that day by day and grain by grain the mortal part wastes and dies away." It is this progressive atrophy which it is so important to detect. Time is necessary to do this, and some certain mode of detecting the gradual decay. For this reason it is absolutely necessary to have the means of

weighing men, and so to determine the ratio of the weight of the person to his age, to his height, and to his respiratory and other functions. These data are absolutely necessary to be known and recognised before any conclusions can be arrived at as to those healthy or normal physiological states with which weight has to do. It may, indeed, be stated generally that every organ and function has a physiological relation to weight and age; and, as a general rule, weight increases with the height and age, and there are physiological limits beyond which the range may not extend. Loss of body weight is therefore *exceedingly significant*, if PROGRESSIVE, as indicating a *persistent atrophy*—the *grain by grain decay* which is frequently the earliest appreciable sign of disease. The first manifestation of weight lost results from absorption of the fat and wasting of the muscles of the body. The appetite may be good and the secretions regular, yet emaciation below the lowest average normal to the individual steadily goes on—a condition more marked and more perilous in those who have been previously stout than in others. The loss of weight is regularly progressive, and necessary to be distinguished from transient loss of weight. In senile phthisis a long period of emaciation commonly precedes positive evidence from physical signs. Sudden and great loss of weight is always serious. It indicates, in the early stages, rapid pulmonary lesions; and generally the most rapid reduction of weight is in males (POLLOCK). The most rapid loss of weight observed by Dr. Pollock was a loss of 56 lbs. in three months; and in twenty-eight cases the following table shows the progressive reduction in weight:—

Period.	No. of Cases.	1 stone.	1½ stone.	2 stone.	2½ stone.	3 stone.	4 stone.
In 5 weeks	1 had lost	1
" 2 months	4 " (respectively)	...	1	2	...	1	...
" 3 "	7 " "	1	2	1	...	1	1
" 4 "	3 " "	2	...	1	...
" 6 "	3 " "	...	1	1	1
" 8 "	1 " "	1	...
" 9 "	1 " "	...	1
" 10 "	3 " "	...	1	1	1
" 12 "	5 " "	1	...	1	...	2	1
Total cases,	28						

The lowest weight in health of one of these persons was 10 st.; he lost in five weeks 1½ st. The rest in health averaged 12 st.; and one was 14 st. 10 lbs., and he lost 3 st. in eight months. Another, whose weight in health had been 13 st., was reduced to 10 st. in three months; 20 out of 28 observed, were between twenty-five and thirty-five years of age; 6 were in the first stage, 10 in the second, and 12 in the third of phthisis when examined, showing that the period of deposit was exceedingly short, and the softening and destructive processes accelerated.

During the incipient stage of scrofula, there is great liability to colds and slight feverishness. The stage of the disease between the second and twelfth years of life is characterised by indolent swellings of the glands,

subcutaneous areolar tissue, and of the joints. In the milder cases there occur, in the form of small spherical or oval tumors, movable under the skin, enlargements of the conglobate and lymphatic glands, especially of the neck.

The Diagnosis in cases of scrofula may often lie as between syphilitic lesions and malignant disease; and the reader is referred to the descriptions of these diseases and the diagnoses of them.

Prognosis.—When the scrofulous cachexia becomes fully developed into *phthisis pulmonalis*, the duration of the disease ranges from about nine months to two years; but in what are called “acute” cases of consumption, it may terminate fatally in three or even in two months, and occasionally in as short a period as three weeks, or even less. The most reliable results of the duration of phthisis are those given by Dr. Pollock. His experience shows that it lasts much longer than has been generally believed. Early invasion, subsequent subsidence of symptoms, and long tolerance of the deposit obtain in many cases; and show an undoubted tendency to extreme chronicity. Since greater accuracy in diagnosis has prevailed, cases of consumption have been known to last much longer,—the average duration out of 3,566 cases observed by Dr. Pollock being *two years, six months, and three-fifths nearly*, including cases of the most acute form. Alison demonstrated that the mortality from scrofulous diseases in the children of the lower orders in Edinburgh and other large towns was so great that they died in the enormous proportion of *forty-five or fifty to five* and even *three*, as compared with the agricultural and upper classes. Tubercles prevail most through the third, fourth, fifth, and sixth years, when the annual growth does not exceed one-tenth of the child’s weight, and the mortality declines to nearly one in a hundred. More than a fourth of those who die in the interval from birth to puberty, are affected with scrofulous disease; yet the disease itself causes death in about one-sixth only of the cases (Sir JAMES CLARK). The greatest number of deaths occur between the ages of twenty and thirty; the next in proportion between thirty and forty; the next between forty and fifty. The mortality is at its maximum about thirty; and, from that age, it declines. Mental disorders claim an alliance with scrofula, which has not been sufficiently appreciated. More than one-half of those who are subject to mental derangement are of a scrofulous constitution, the existence of which is obviously indicated in them. Scrofulous symptoms also often alternate with attacks of mania (CUMIN).

Healing of Local Lesions in Scrofula.—It is of the greatest importance to study how some of the most unmanageable and hopeless diseases will sometimes spontaneously become cured, and how the local lesions will heal. That scrofula sometimes tends to these results we have abundant testimony daily afforded in almost every *post-mortem* examination of those who die of *phthisis pulmonalis*. Laennec, Carswell, Clark, and Bennett have recorded their testimony on this interesting point; and the practical result of their observations shows that if the further growth of tubercle can be arrested, the masses already existing may silently retrograde, become absorbed, or diminish in size, and the part cicatrise; or, it will remain latent as a cretaceous mass in the lung.

Treatment.—(a.) *Preventive*.—There is perhaps no subject in the whole

range of medical science which the student ought to study more carefully than the cachexia, or special form of ill health, associated with the occurrence of *tubercle* and *scrofulous affections* as here described. As a practitioner he will find that he becomes often painfully concerned in the deepest interests of families and society, through the threatened or actual ravages of scrofulous diseases. The extensive prevalence of the scrofulous cachexia—the great and almost inevitable mortality of the scrofulous diseases themselves when completely developed, stamp the morbid state associated with them as a topic which, at the outset of the student's career, ought to engage a large share of his study. Most assuredly the physician will have to turn his knowledge of the pathology of scrofula to account in every phase of his professional life. When he fully appreciates what experience has adequately demonstrated, that the scrofulous cachexia springs from causes over which the public (rather than the medical profession) have control, he must be at once impressed with the belief and encouraged with the hope, that when he acquires the confidence of families in the practice of his profession he may exercise a powerful influence for good in teaching how much the public may control the ravages of *scrofula* and *consumption* by prudent marriages, by sanitary attention to offspring, and by the necessity of free ventilation and of fresh air in dwellings. There are several circumstances which show the great influence of public sanitary measures in controlling the development of scrofula, when these measures are scientifically directed to the preservation of general health, especially where men are associated together in great communities—an influence much greater than the best directed efforts of the medical profession can establish through their *materia medica*. It is by the mode of life as citizens of the world, in the social relations of husbands and wives, parents and children; in the public relation of masters and workmen, that the extent and ravages of *consumption* and *scrofula* are to be controlled. It is by a strict attention to the rearing of offspring, and in the subsequent regulation of food, clothing, cleanliness, and occupation; in the choice of a profession, and by many other circumstances which have an obvious influence (perhaps at first sight inappreciable) on the maintenance of the general health, that our hopes of success as practitioners of medicine must rest in the prevention of that bad habit of body which develops and propagates the scrofulous diseases in civilised society.

(b.) *General Treatment*.—From what has been already stated in illustration of the pathology of scrofula, it is to be observed that the general treatment now in most repute is founded on the doctrines—(1.) That scrofulous local lesions will heal of themselves if the nutrition of the system can be maintained and the continuous growth of tubercle arrested; (2.) that the periods of frequent temporary arrest of all the general and local symptoms of disease ought to be diligently taken advantage of to improve and preserve health by hygienic means; (3.) that the efforts of the practitioner should be directed to the digestive rather than to the pulmonary system; (4.) that the kind of morbid nutrition in the body generally, and altered morphological change in the tissues of the organ where the deposit takes place, appear to be chiefly due to excess of albuminous and deficiency of fatty elements in the chyle. The treatment, therefore, to be pursued must be essentially reparative of the waste of

tissue generally; corrective of what has been unfit in the individual diet and mode of life; and, lastly, supplementary of the elements of nutrition which have been deficient. During the past *thirty* years in Germany, and *twenty-three* years in this country, the treatment of scrofulous affections has gradually but steadily become more and more firmly based on those pathological doctrines which the late Dr. J. H. Bennett, of Edinburgh, was mainly instrumental in first elucidating, and in earnestly recommending to the notice of the profession generally in this country. The view here taken regarding the nature of scrofula leads to the belief that—(1.) The blood is impoverished through the preliminary dyspepsia which precedes the growth of crude tubercle; (2.) that in *pulmonary phthisis* the growth of crude tubercle results in a consolidation primarily in the air-vesicles; (3.) that the successive formation and softening of these crude tubercles lead to ulcerations of the pulmonary and other tissues—to the infective processes which set up the growth of true miliary tubercle—and, promotes wasting of the body generally.

It has been now fully shown, especially by the observations of Dr. Bennett, in the first instance, confirmed by the extensive experience of the physicians at the Brompton Hospital for Consumption, in London, that such treatment as is directed to remove the malassimilation of food frequently checks the tendency to the repeated occurrence of tubercles, while those which previously existed remain harmless; that general symptoms and physical signs may disappear completely; and that even extensive excavations in the pulmonary tissue may heal up and cicatrise. According to the testimony of Dr. Wood, of Pennsylvania, the fatal results have not only been postponed, but the death-rate from phthisis has diminished in the principal cities of the United States since such principles of treatment have been adopted. The indications of general treatment are therefore—(1.) *To improve the faulty nutrition, which is the cause of the scrofulous cachexia, and of the exudations assuming the characters of tubercle;* (2.) *To subdue the fever which attends the growth and changes going on in the tubercle-nodules, and to favour the absorption either of the entire exudation, or of such portions of it, that what remains may undergo such changes as are consistent with its future harmless existence in the organs or other parts where it may have grown;* (3.) *To prevent the recurrence of fresh exudation by careful attention to hygienic regulations, especially during the intervals of apparent return to health.*

To fulfil the first of these indications it is of all things important that fatty matter be assimilated in large quantities, and it appears that such is most readily absorbed and assimilated when in the fluid condition. The substance of all others found most beneficial has been cod-liver oil. At the Brompton Hospital more than 600 gallons of this oil are used annually. There, on a great scale, its merits have been tested and compared with the effect of treatment conducted on general principles, and irrespective of its use. The result has been to confirm, in the estimation of the medical profession, the great value of the remedy in the treatment of phthisis, when appropriately administered, and combined with the use of such other measures as any special circumstances in the individual patient may require (THOMPSON). The general opinion of the profession with regard to cod-liver oil may be summed up in the

statement originally made by Dr. Bennett,—namely, that “it rapidly restores the exhausted powers of the patient, improves the nutritive functions generally, and stops or diminishes the emaciation. The night perspirations subside, the cough is quieted, and the expectoration is diminished after the oil has been used for a few weeks. A very constant and well-marked favourable change under its use is to be noticed in the diminution of the pulse, which gradually but steadily has been observed to come down at the rate of five or six beats weekly; and during the same period the weight of the body has been known steadily to increase at the rate of half a pound to a pound. In females it is a significant and highly favourable symptom when the catamenia return after the oil has been used for some time. The usual dose adopted by the late Dr. Thompson, at the Brompton Hospital, is *one or two tea-spoonfuls twice a day* at first, and gradually increasing the quantity to *half an ounce three times a day*. Dr. Bennett recommends somewhat larger doses—namely, for an adult, *a tablespoonful three times a day*, which may often be increased to *four or even six* with advantage. When the stomach is irritable, however, *a tea or a dessert-spoonful is enough to commence with*. It appears from the observations of Dr. Thompson that no additional advantage is obtained by pushing the oil beyond the limits of the doses adopted by him at the Brompton Hospital, from the fact that where its use has most obviously increased the weight of the body, to the extent in one instance of a pound per week for twenty-one weeks, only three pints had been taken during that time. The kind of oil used, as far as coarseness or fineness is concerned, seems not to affect the beneficial result in any material degree. Some patients even prefer the coarse to the fine oil. The experience of some is, moreover, favourable to combining the oil with *liquor potassæ* as an emulsion; and as it appears that undue acidity prevails as well in the stomach as in the intestinal canal, the addition of the alkali ought, on theoretical grounds, to be advantageous. It seems also that, when cod-liver oil was first used as a medicine, more than fifty years ago, in the treatment of rheumatism, it was then ordinarily combined with an alkali. It may be taken, however, unmixed, or it may be floated on milk, or nitro-muriatic acid mixtures, or on lemonade, soda-water, lemon juice, or on a saline draught during effervescence, when such combinations are suited to the patient. Creosote has been recommended to be added, as it is said to render the stomach more tolerant of the remedy. The following formula, quoted from Dr. Thompson’s *Clinical Lectures on Pulmonary Consumption*, yields a palatable mixture, which ought to be combined as an emulsion:—

“An ounce and a half of cod-liver oil, four drops of creosote, two drachms of compound tragacanth powder, and four ounces and a half of aniseed water. Of this mixture an ounce may be taken thrice daily.”

Besides cod-liver oil, other animal fats and oils, where they can be taken and assimilated, are sure to be followed with benefit. Hence milk rich in fatty matter, such as asses’ milk, and milk drawn from cows at a short interval after the greater part of their milk has been withdrawn, and which is known in Scotland as the “afterings,” are found to be

followed by improvement where they are persevered in and are assimilated. So also has it been with cream and butter. Dr. Bennett instances the partial success occasionally of caviare, bacon, pork, mutton chops, and the marrow of the bones of oxen; while Dr. Thompson instances the good effects he has obtained from the use of oil obtained from the foot of the young heifer (neat's-foot oil). The administration of any of these remedies is quite consistent with doctrines now taught regarding the pathology of scrofula, and it is useful to know their individual value, in order that in particular cases one may fall back upon their use where a change may be desirable.

It has been considered that some of the good effects of cod-liver oil may be due to the biliary elements with which it has been incorporated. This view is not supported by the experiment of adding ox-gall to other animal oils not derived from livers, as no beneficial results have been observed to follow. But as the active principle of the gastric juice has been now successfully isolated by chemistry, and has been successfully used to aid the digestion of food in the stomach, might not some principle be obtained from the liver which might aid the assimilation of fatty substances when mixed with the intestinal juices?

In fulfilment of the second indication mentioned (p. 1031), the propriety of abstracting blood has been much discussed. It has been already seen that febrile symptoms of a very severe kind sometimes attend the exudation and consolidation of crude tubercle. It is also a more or less frequent clinical observation that pneumonia, bronchitis, and pleuritis, in acute or chronic forms, are intercurrent attendants on the deposit and future changes of tubercles in the lungs. "Hence," as Dr. Bennett justly observes, "there are all kinds of intermediate changes between the simple and tubercular exudations constantly going on in the progress of a case of pulmonary tuberculosis. The phenomena of phthisis, pneumonia, pleurisy, and bronchitis, in their acute or chronic forms, may appear together, and be inextricably mingled, or they may succeed each other at intervals." Thus, scrofulosis, both as a constitutional and as a local disease, is scarcely ever free from exacerbations, the various local and constitutional states acting and re-acting on each other. While, therefore, on the one hand, the system requires an increased and well-directed supply of nutritive materials, on the other hand, there are constitutional states of excitement, depending on local irritation, which require to be subdued, and which may even demand antiphlogistic treatment. Whatever theoretical view may be taken as to how the exudation may most readily be absorbed, all physicians are now at one as to the propriety of preserving the general strength, of effecting elimination of effete material, and of meeting antiphlogistic indications rather by diaphoretics, diuretics, emetics, and purgatives, than by abstracting any considerable amount of blood, either at once or at repeated intervals. In fact, it is now observed that the administration of appropriate diet, and abstinence from lowering remedies, with cod-liver oil, while they correct the general nutrition, may be so regulated as to subdue the constitutional irritation by a perseverance in their use for a period of not less than four or six weeks.

In fulfilling the third indication, the real power of the science of medicine may be demonstrated. It is by well-directed hygienic measures,

successfully and efficiently carried out, that the real strength of the physician may be put forth to prevent the recurrence of fresh exudation; and his hygienic exertions towards the patient are to be redoubled during the temporary intervals of apparent return to health. It is now almost an axiomatic truth, that of all things which deteriorate the constitution on the one hand, and influence pulmonary congestion on the other, none are so detrimental as impure and deficient air, together with frequent variations of temperature, and changes from sudden heat to chilling cold. These latter vicissitudes may be considered characteristic of the physical climate of Great Britain and Ireland. "The conditions of preventive treatment which have seemed most useful are nutritious food and proportionate great exercise in the free and open air. So important has this last condition proved to be, that it would appear that even considerable exposure to the weather is better than keeping phthisical patients in close rooms, provided there be no bronchitis or tendency to pneumonia or pleurisy" (PARKES, l. c., p. 445). Tonic treatment consists essentially in the adoption of those means which promote or stimulate the healthy nutrition of the body. Its elements exist in fresh air, abundant exercise, sufficient repose, and judicious diet. A hygienic code applicable to the tuberculous cachexia has been recently laid down by Dr. B. W. Richardson, in the second volume of the *Sanitary Review*, and very recently in a special work *On the Hygienic Treatment of Pulmonary Consumption*. It is derived from these elements of tonic treatment; and as it puts well-known truths, too little appreciated, in a formal and more important aspect than is wont, its precepts are here quoted.

I. *A supply of pure and fresh air for respiration is constantly required by the tuberculous patient.*—As it is known that if one per cent. of carbonic acid exists in a room, the air is unfit for a healthy person, it is therefore much more so for a consumptive one. The temperature of a room ought to be equally maintained at from 55° to 56° Fahr., ventilation and heating being effected by open fireplaces. A single room ought not to perform the two offices of a bed-room and a sitting-room. The sleeping-room ought not to afford less than 1,000 feet of space; and if larger, so much the more healthful will it be. In connection with these statements, while it is objected, for obvious reasons, with much justice, to the treatment of tuberculous patients in special hospitals, there is much on the other hand that might be improved in all our hospitals, with reference to the arrangement of the patients, to secure to them fresher air than they generally obtain. "Constant though imperceptible movement of the air is the point to be attended to"—i. e., thorough ventilation. All who are able to be out of bed ought to have their meals in a common room, which is not used for any other purpose, and is apart from the wards or dormitories.

II. *Active exercise in the open air is imperatively demanded by the tuberculous patient.*—In the words of Dr. Jackson, "He must be made to feel that the risk is in staying in the house, and not in going out of it." But the skin must be perfectly protected; and while a chill, or inclement weather, is to be avoided, the patient must go out in all seasons, without being too fastidious about the weather, walking exercise being persevered in as much as possible. "The best climates for phthisis are perhaps not

necessarily the equable ones, but those which permit the greatest number of hours to be passed out of the house" (PARKES). Next to diet, exercise in the open air is, of all things, the most important: it should be carried as far as the vigour of the patient will permit. It should not be done rashly, but boldly; and, if possible, the patient ought to have faith in it; for without this he is not likely to pursue it so far as he can, and then he will not derive from it all the benefit which it can afford (JACKSON). It is very important to remember, in regard to training animals and young persons, that they have much more extent and range of lung than are required in the quiet everyday pursuits of life. Less lung is used when the young person or other animal lies down or sleeps, or is depressed; and, on the contrary, walking, running, wrestling, the force of the passions, each or all of them bring the greatest amount of lung into action or use. In short, the amount of lung in use is an ever-varying quantity; and just as much lung *may come to be used as* A HABIT as the listlessness or vigour of the individual moment requires. There is every range, every variety, till the top of the wind, the top of the speed (the full vital capacity) is attained (SIBSON). Practically, therefore, the more fully the lungs are judiciously used, the more is their capacity nursed; and, conversely, the less they are used and expanded, the more useless they are likely to become, if not absolutely diseased. Under a judicious system of training, an undeveloped man, even although he may be feeble, narrow-chested, and sickly, may yet become active, full-chested, and healthy. We have numerous examples of this among the boys in our training ships for seamen. The over-fed, short-winded pugilist, rower, or cricketer, may in a few weeks be changed, by training alone, to the firm-fleshed, clear-skinned, long-winded winner of the boxing fight, the foot race, or the rowing match. It is this want of use that probably renders the apices of the lungs more liable to the growth of tubercle in them than in any other part of their substance. It is a portion of the lungs which has less play or expansion than any other portion, and is apt to be bound down by the surrounding parts. The apices are, therefore, the parts most likely to remain in a quiescent state of non-expansion, especially when acts of respiration are inadequately performed, either owing to the constrained position of the body in certain trades, or from habitual stooping of the body from listlessness of habit or want of vigour in the system. This quiescent state of the air-cells is favourable to the growth of tubercle; and thus the apices of the lungs are the most common seat of tuberculous growths (Reviewer in *Medical Mirror*, Vol. I., p. 638). The effects of want of exercise and of impure air are thus most potent agencies in causing phthisis. "The much greater prevalence of phthisis in most of the European armies (French, Prussian, Russian, Belgian, and English) can scarcely be accounted for in any other way than by supposing the vitiated atmosphere of the barrack-room to be in fault." In all the places where phthisis has prevailed, in the most varied stations of the army, and in the most beautiful climates, the only common condition was the vitiated atmosphere which our barrack system everywhere produced; "and, as if to clench the argument, there has been of late years a most decided decline in phthisical cases in these stations, while the only circumstance which has notably changed in the time has been the condition of

the air" (PARKES, l. c., p. 91). Indeed, the air is rendered so impure by respiration, that while an atmosphere so vitiated has a most injurious effect upon the health, contamination of the air has been so great, where lung diseases abound, as to give rise to the idea that phthisis appeared to be propagated by contagion (BRYSON, PARKES). It is therefore within the power of the authorities of the Army and Navy so to direct the physical training of young persons, that the apparently sickly and the short-winded may in time be developed into the wiry and active young man, long in wind, sound in body, and lithe of limb—a result which, however, can only be attained by judicious feeding, careful exercise throughout the development of the body, and by the gradual nursing of the breathing powers. The opposite of this is seen in the breaking down of recruits at a very early period of service in the army—within the third year—a result likely to be greatly obviated by the *gymnasia* introduced by Government for the physical training of recruits before and after they are instructed in drill, by better and more abundant food, and improved barracks. The Austrian runners also furnish instances of breaking down by over-exertion in running at ages unsuited for their strength. They seldom live above three or four years, and gradually die of consumption (*Remains of Mrs. Trench*, p. 72). In all physical training the condition of the heart must be considered not less than the lungs, seeing that its movements respond to the movements of the chest and the lungs. They are members of one great system; and in nursing one we nurse the other; for by giving tone and health to one we must give tone and strength to the other (SIBSON). If, on the contrary, the development and gradual training of the lungs are not successful, the lesions which become developed are of a peculiar character, and are apt to be thought very lightly of; because, in the first instance, they are so little capable of appreciation by a single observation; and, secondly, because the lesions are insidious, and take some time before they reach a stage to be apparent.

III. *It is important to secure for the patient a uniform, sheltered, temperate, and mild climate to live in, with a temperature about 60° and a range of not more than 10° or 15°; where also the soil is dry, and the drinking-water pure and not hard.*—The classic work of reference on this topic is that by the late Sir James Clark. If it is possible to give a practical abstract of his extensive and valuable experience, it may be done somewhat as follows:—(1.) After the functions of the digestive organs and skin have been re-established in improved action, the patient who labours under a tuberculous cachexia may derive benefit by a residence in a mild or temperate climate (such as has been defined), conforming to all the hygienic and medicinal treatment already mentioned. (2.) When symptoms, however slight, indicate that tuberculous deposit has located itself in the lung, removal to a mild climate, especially if effected by a sea voyage, under favourable circumstances, may still be useful as a means of improving general health, of lessening the chance of intercurrent inflammatory affections of the pulmonary organs, and even of arresting the further progress of the disease. The nausea, squeamishness, or even sickness, which with some are always more or less associated with a sea voyage, are beneficial to cases of incipient tuberculosis. The effects of such nausea tend to increase the natural

secretion and elimination from the pulmonary mucous membrane; so that minute portions of tubercular exudation, commencing to consolidate in the air-vesicles, are effectually, gradually, and gently passed out with the motion of the pulmonary mucus in the expectoration. The sensation of nausea tends to subdue any local vascular irritation; and the unceasing motion of a sailing vessel tends to keep up a constant exercise which is advantageous to the patient. (3.) When extensive tuberculous disease exists in the lungs, little benefit is to be expected from a change of climate; and a long journey will most certainly increase the sufferings of the patient, and hurry on a fatal termination. (4.) There are cases, however, of chronic consumption which may derive benefit from residence in a mild climate,—namely, cases in which the deposit is limited to a small portion of the lungs, and little systemic irritation prevails; or in cases in which the disease has ceased to extend to the lungs, but where a long time is required to complete repair.

IV. *The dress of the scrofulous patient ought to be of such a kind as to equalise and retain the temperature of the body.*—Under this topic waterproof coats, boots, and shoes are to be condemned. Flannel ought invariably to be worn next the skin in all seasons; and in winter a chamois leather vest may be required over the flannel.

V. *The hours of rest should extend from sunset to sunrise.*

VI. *Indoor or sedentary occupation must be suspended; but outdoor employment in the fresh air, even in the midst of snow, has been and may be advantageous.*

VII. *Cleanliness of body is a special point to be attended to in the hygienic treatment of tuberculosis.*

VIII. *Marriage of consumptive females, for the sake of arresting the disease by pregnancy, is morally wrong and physically mischievous.*

IX. *The medicinal treatment must be adapted to the site of the local scrofulous deposits and the general nature of the particular case. Iron and iodine in various forms are the most useful remedies; but medicine is utterly powerless and useless unless the hygienic means now insisted upon are carried out to the utmost.*

(c.) *Medicinal Treatment.*—When medicine is required to move the bowels, as it often is, nothing serves so well as powders of *rhubarb*, *soda*, and *calumba*, taken three times daily, or morning and evening only, or an hour before food. *Mercury* in any form is not borne by scrofulous patients. *Lime-water* to the extent of half an ounce taken with milk, three or four times a day, is of marked benefit in those cases of long standing where gland after gland becomes the seat of abscess and ulcer (SHAPTER). The *muriate of barytes*, next to *iodine*, exerts the most decided influence over scrofula. It increases the appetite and the secretions (ADAIR, CRAWFORD, PHILLIPS). The whole class of tonic remedies are of essential service; such as, small doses of *quinine* with *conium*; *iron* in all its most digestible forms (see under *anæmia*). The employment of *acids* is often also called for, especially if perspirations become excessive. A useful formula consists of—

Acid Hydrocyan. dil. ℥i., *glycerine* ℥ii.; *acid nitric. dil.* ℥iii., *Infus quassia*
ad ℥xiiiiss—a tablespoonful for a dose.

But of all remedies *iodine* and its compounds are the most valuable in scrofula. The following formula is that which Dr. Shapter has found most useful:—

R *Iodine* gr. x.; *potassæ hydriodatis* gr. xx., *aquæ* ℥ii. From 8 to 12 minims are to be dropped into a glass of water and taken three or four times a day by an adult.

It improves appetite and the complexion; subdues the general symptoms, and promotes absorption of the scrofulous glandular swelling. *Iodism* or *iodic saturation* must, however, be very carefully guarded against.

RICKETS.

LATIN EQ., *Rachitis*; FRENCH EQ., *Rachitisme*; GERMAN EQ., *Rhachitis*—Syn., *Englische Krankheit*; ITALIAN EQ., *Rachitide*.

Definition.—*A constitutional disease of early childhood, characterised by a cachexia, which precedes for several weeks or months a peculiar lesion of the osseous system, manifested by curvature of the shafts of long bones and enlargement of their cancellous extremities. The growth of the bones is characterised by irregularity, by non-solidification of their growing layers, and by the progressive formation of medullary cavities in the old bone, thus rendering the bony laminae thin and brittle (VIRCHOW). Some of the solid visceral organs also exhibit peculiar lesions, such as in the spleen and liver, where there is generally lardaceous disease.*

Pathology.—(a.) *History.*—Because the disease affects, in an especial manner, the spinal column, Glisson was induced to name the disease *rachitis*, from *ράχι*, the spine, and also because it resembles the word “ricketts,” the name by which the malady was commonly known in England more than 200 years ago (before 1645), when it was described by David Whistler. It was then believed to be peculiarly an English disease.

(b.) *Causation.*—From the great vascularity and infiltration of the affected bones, some have regarded the disease as expressing some form of inflammation, more especially as pain accompanies the lesions from the first. But the course and the constant results of the disease do not favour this view. Another theory (based on the fact that the urine of rachitic children is often extremely rich in *lactic acid*, and contains as much as from four to six times of *phosphate of lime* as normal urine contains) holds that the calcareous salts taken up by the food cannot be deposited in the terminal and peripheral layers of bone, being held in solution by the lactic acid in the blood and excreted by the kidneys. But the actual changes in the bones do not support this view. Active decomposition, rather than digestion, also goes on in the stomach and intestines of rachitic children. Virchow considers the retarded ossification due to a diminished supply of chalky salts. The most probable hypothesis regarding the cause of rickets is that which refers to inflammation of the epiphysal cartilages and periosteum, and local disturbance of circulation

hindering the deposit of calcareous salts (NIEMEYER). The number of cases happening in the first or second year of life very greatly exceeds that of other periods.

(c.) *Morbid Anatomy*.—The changes in the bones consist of—(1.) Proliferation of the cartilages and epiphyses, and of the periosteum—the sources of the normal growth in length and thickness of the bones. (2.) The cartilaginous and fibrous tissues ossify more densely, and at a later period than in the normal growth of bone. The tissues remain abnormally soft for a length of time, after which they become abnormally hard (NIEMEYER).

Symptoms.—The earliest recognition of the cachexia associated with rickets is rarely apparent before the fourth month of infant life, and usually between the fourth and twelfth months. It does not in general declare itself until the child first begins his attempts to walk, or until he shows suffering during the first dentition; and at first the progress of the disease is so very slow as almost to be imperceptible. There is a period of at least six months during which a marked series of deranged actions succeed each other, which eventually culminate in the condition known as *rickets*. Many of the phenomena of these deranged actions are common to other diseases; but some are characteristic, and when they occur in sequence are sufficient indications of the specific characters of rickets. The symptoms may be arranged into four classes:—(1.) Those which always denote the precursory or incubative stage of rickets, which are common to many diseases—symptoms which might arise from deranged digestion, improper food, or scrofula, and which are often referred to the “irritation of teething,” or to the so-called “infantile remittent fever.” (2.) Those which at once mark the nature of the disease, render its diagnosis easy, and which enable us to predict that the bone affection will show itself. (3.) The stage of characteristic deformity. (4.) Phenomena of favourable or of unfavourable import, inasmuch as they may characterise a period of restoration to health, or one of irremediable atrophy of the body, or of approaching dissolution. During the precursory or incubative period, the most ordinary symptoms of impaired general health are those which indicate gastro-intestinal irritation. The bowels are irregular in their action; sometimes confined, but more commonly there is diarrhoea (*intestinal catarrh*), with tumidity or enlargement of the abdomen. The stools may be of a dirty brown or leaden colour, and of a most offensive odour. There is great proneness to decomposition of the contents of the intestines. In some respects the odour therefrom is peculiar in its resemblance to rotten or half-decayed meat. Appetite is feeble, or entirely lost, and digestion is difficult. The child becomes dull and languid, sad or peevish; febrile irritation prevails; the skin is hot; and the temper irritable. Although drowsy, it sleeps but little. It is thirsty, and will drink large quantities of water. It lies about, and is unwilling to play, or to be amused, or to indulge in any kind of action. It prefers to sit or to lie; and it appears to be feeble or indolent, and is unable to use exertion of any kind. The transition from health to these phenomena is always gradual and slow; but there are at least three sets of phenomena which, being superadded to these, are characteristic of the approach of rickets (Sir W. JENNER). The following symptoms stand by themselves as pathognomonic of this affection, namely:—(a.) *Profuse*

perspiration of the head, or of the head and neck, and upper part of the chest arrests the mother's attention; and so she seeks medical aid. She will say that the perspiration stands in large drops on the child's forehead—that it runs in streams down the face; and that it is especially when the child sleeps that such copious perspirations of the head occur: but they are not unfrequent when the child is at the breast, or even when only resting its head on the mother's arms. A little increased exertion, or a little increased temperature, may induce such excessive perspirations, which are extremely weakening and colliquative during sleep; and when they occur the superficial veins of the scalp are generally large and full, the jugular veins are much dilated, and sometimes the carotid arteries may be felt strongly pulsating (COPLAND, JENNER). (b.) Another characteristic feature of the disease is seen in the desire and in the efforts of the little patient to be cool, particularly at night. The child kicks the bed-clothes off, or throws its naked legs on to the counterpane; and this even in cold weather. (c.) There is also general tenderness. The child cannot be moved without its uttering a cry; pressure on any part of its body is followed by evidence of suffering. It ceases to play and to move, but lies with outstretched limbs as quietly as possible, for all movement produces pain; and it will cry at the approach of any one who has been accustomed to move it in play. As the disease progresses the child becomes staid and steady in appearance. It assumes a pensive, aged, and languid aspect. Its face grows broad and square; and when placed on the mother's arms, it sits (as she says) "all of a heap." The spine bends, and the muscles are too weak to keep the spine erect. Its head thus seems to sink between its shoulders, and its face appears turned a little upwards. Before the general cachexia has lasted long, the bone deformity begins to attract attention, and usually the lesion of the bones is out of all proportion in severity to the enlargement of the ends of the long bones. The younger the child, the softer usually are the bones. The consequences of the bone disease thus become superadded to the general cachexia; and as the disease progresses, the muscles lose their power and begin to waste. The child cannot support itself; and if it has commenced to walk before it becomes the subject of extreme rickets, it loses its power of walking. Intellect is invariably deficient. The teeth are retarded in their development, and they fall from their sockets early. The back, the arms, and the sides of the face are often covered with downy hair. In short, "the general aspect of the rickety child is so peculiar, that when the crooked limbs, the large joints, and the deformed thorax are concealed, you may even detect its ailment at a glance. Its square face, its prominent forehead, its want of colour, its large staring and yet mild eyes, its placid expression, and its want of power to support itself, like other children of its age, in its mother's arm, all conspire to form a picture which has no like in the gallery of sick children," (Sir W. JENNER, *Med. Times*, l. c.) *Laryngismus stridulus* is a frequent result of rickets.

Treatment.—Ventilation of the room in which the child lives is of first importance. Milk diluted with *lime-water* (about a fourth part), and a teaspoonful of cream added, is the best of food, or the Swiss preserved milk. Sugar ought not to be added to the milk. *Liebig's Food for Children*, or *Valentin's* flesh juice, "*Maltine*," *Parrish's Chemical Food*, and *Trommer's*

Extract of Malt, are all valuable agents in the dietary. About once a week a dose of *rhubarb*, *soda*, and *calumba*, in equal parts, should be given, followed next day by a teaspoonful or more of *castor-oil*. *Prepared chalk* and *soda* may also be given twice or thrice a day, with or without *rhubarb* and *calumba* (see under treatment of *Scrofula*). When the febrile disturbance is subdued, the child should live as much as possible in the open air. *Vinum ferri* ought then to be given in the following formula:—R. *Vin. ferri* f ʒi-ʒii.; *quinice sulph.*, gr. i.; *acid sulph. dil.* ℥i-ii. This form is especially useful when the skin is flabby, covered with perspiration, and *anæmia* well marked. Or small doses of the *syrup of the phosphates of iron, quinine, and strychnia*, or *syrup of the phosphate of iron and lime*, may be given along with the food, or just before meals. *Cod-liver oil* is of essential service; but the stools ought to be examined daily; and if any of the oil passes by stool its dose ought to be diminished, or its administration suspended for a time. (Sir WILLIAM JENNER, *The Medical Times* of 1860, Vol. I.)

CRETINISM.

LATIN EQ., *Cretismus*; FRENCH EQ., *Crétinisme*; GERMAN EQ., *Cretinismus*; ITALIAN EQ., *Cretinismo*.

Definition.—*A condition of imperfect development and deformity of the whole body, especially of the head. It is endemic in the valleys of certain mountainous districts, and is attended by feebleness or absence of the mental faculties, and sometimes of the special senses. It is often associated with goitre.*

Pathology.—(a.) *History.*—The condition of idiocy named *cretinism* (associated with *goitre* in many districts) is of great interest; but the relations of the two are not yet clearly understood. The idiocy of *cretinism* is associated with deformity and imperfection of the bodily organs—the brain, in common with other parts, participating in the imperfection and deformity. The affection of the mind varies from mere obtuseness of thought and purpose to the most complete obliteration of all intelligence.

(b.) *Causation.*—The *cretin* is found chiefly in the valleys of the Pyrenees and the Alps, in the mountains of Syria, in the hilly parts of China, and in the Himalaya regions; but the disease is not confined to the lower valleys of Switzerland, or to those other mountain districts of the Old and New Worlds which resemble it in physical conformation. All over Europe the victims of *cretinism* may be seen; and Virchow found, in his official inquiry into this subject, that no less than 133 decided cases were living in the villages of Lower Franconia; and in Germany, Sweden, Norway, England, and even in London, isolated cases of *cretinism* are to be met with. With but few exceptions, *cretins* are *goitrous*; and it has been said that when both parents are *goitrous* for two generations in succession, the offspring, being the third generation, are sure to be *cretins* (WATSON, *Lecture xliii.*, Vol. I., p. 758). *Fodère* makes an observation to the same effect. The predisposition appears to be hereditary. *Cretinism* with *goitre* is very prevalent in different parts of

the district of Goruckpore. The soil of the district is of two sorts. One, to which the natives give the name of *Bhat*, characterises the lands bordering the river Gunduk and its branches. This soil is remarkable for the large proportion of calcareous matter which it contains. One specimen, on analysis, yielded upwards of twenty-five per cent. of carbonate of lime. Cretinism and goitre are very prevalent in the villages built upon this soil. In some of them, ten per cent. of the population are affected; and of the children in the villages where goitre prevails, ten per cent. are *cretins*. The dogs and cats of these villages are also often affected with the disease. On the other hand, the lands on the banks of the Gogra consist of a soil to which the natives give the name of *Bangar*. It is much less retentive of moisture than the *Bhat* land, and requires irrigation for the production of winter crops. This Bangar soil is very siliceous, and contains scarcely any lime. Goitre and cretinism are unknown in the villages built upon this soil (*Brit. and For. Med.-Chir. Review*, Jan. 1861). The natives of Oude ascribe their goitres to drinking certain waters; and they adduce cases to prove that by partaking of the water of certain wells they get the disease, and by deserting those wells they sometimes become cured of it (GREENHOW). Thus almost all writers who have written on the subject agree that, in some way or other, the condition of the water has to do with the production of goitre. Remarkable instances are known wherein the exchange of well for rain-water for drinking purposes, has been followed by the best effects, and even by the disappearance of goitrous tumors. Dr. Greenhow states that in Oude, where the water of wells believed to be injurious in consequence of their excessive impregnation with lime has been given up, and other water used instead for drinking, great benefit has been felt, and goitres have decreased in size, even though the subjects of them have continued living in the same village as before. He was assured also, by several of his patients in Oude, that certain wells were known by them to be deleterious, and that the natives of the villages avoided them accordingly, having learned to do so from experience. He tested the water of the wells most shunned by the natives, and found it to contain a great excess of lime; and he concludes, from his own investigations, in connection with others, that the use of drinking-water containing lime is the main cause of goitre. How it acts on the system is as yet unknown. It may generally, therefore, be concluded, from the cumulative nature of the evidence, that a poison exists in association with lime and magnesia in geological formation, whose action induces undue ossification and thickening of the base of the cranium, tending to diminish the size of the foramina for blood-vessels (KÖLLIKER, VIRCHOW); and it is fair to connect the unusual quantity of lime taken into the system with such premature and abnormal ossifications. Wherever chemical examination of the water used by the inhabitants of the different places where goitre and cretinism prevail has been made (as it has been especially in India), it has always been found to contain a large quantity of carbonate of lime; whereas the water derived from the clay-slate rock, which was drunk by the inhabitants who did not suffer from goitre, contained none. Such observations as those described, and especially those of McLelland and Greenhow, show that neither the atmosphere, the elevation above the sea-level, the physical

aspect of the country, nor locality, have anything to do with the production of goitre; but they seem to prove, almost to a demonstration, that the affection is due to some specific action of the drinking-water which flows from rocks of a particular geological formation, named magnesian limestone. The circumstances under which these affections were found by M'Lelland to exist in the low burning plains of Bengal, formed a striking corroboration to his observations in the hills of Kemaon.

(c.) *Morbid Anatomy*.—The stature of the *cretin* is diminutive; his head is of great size, but flattened at the top, and spread out laterally; while the countenance is vacant and void of intelligence. The nose is flat, the lips are thick, and the tongue is large. The skin is dark-coloured, coarse, and rough. The abdomen is sunken and pendulous; the legs are short and curved. Virchow's dissection of the heads of *cretins* led him to conclude that the primary abnormality of the brain commences with the growth of the bones of the *basis cranii*, and especially with the *sphenoid* and the adjoining parts. In the normal state, the basilar part of the occipital bone, the sphenoid and ethmoid, with their intervening cartilages, form a portion of an arch; while the same parts in a *cretin* are early ossified into the form of a rectangle, early union of the bones taking place with various lesions of the intervening cartilages. With such early union, arrest of growth occurs at that part of the skull; but various compensatory developments continue in other parts. Hence the *prognathous* face, and the sinking of the root of the nasal bone. Irregular and partial union of the sutures at an early age is a frequent morbid condition of the insane *cretin*, associated with an atrophic condition of the *gyri* below the site of union. The oblique downward direction of the orbit in *cretins* is brought about by the compensating growth of the skull generally, and more especially of the *malar*, the *frontal*, the *temporal* bones and *zygomatic* arches, in consequence of the deficient development of the *sphenoid* bone. The stunted development of the bones at the base of the skull gives a very short distance between the front and middle part of the cranium; while the diminished growth of the nasal septum and of the jawbones gives a *prognathous* form of face alike to the *cretin*, the negro, and the monkey.

Symptoms.—Three varieties are to be distinguished:—“(a.) *Complete Cretinism*—Synonym, *Incurable Cretinism*.—*Cretinism* characterised by *idiocy*, *deaf-dumbness*, *deficiency of general sensibility*, and *absence of the reproductive power*. (b.) *Semi-Cretinism*.—*A degree of cretinism in which the mental faculties are limited to the impressions of the senses and the bodily wants; the general sensibility is obtuse, the head is badly formed and drooping, the speech is rudimentary, and the reproductive powers are feeble or absent*. (c.) *Incomplete Cretinism*—Synonym, *Curable Cretinism*.—*A degree of cretinism in which the mental faculties, though limited, are capable of development; the head is moderately well formed and erect, the special senses, the faculty of speech, and the reproductive powers are present*.” Although it seems that some cases of cretinism are congenital, yet Dr. Tuke, from personal observation, and from the Report of the “Sardinian Commission,” believes (*op. cit.*, p. 104) that there is no pathognomonic sign by which cretinism can be recognised at birth, but that a certain combination of symptoms permits the prognosis in childhood of the future development of cretinism. After the fifth or

sixth month he describes the infant cretin as presenting the following symptoms:—"The development of the body proceeds very slowly; the child, though weak, is remarkably stout, and appears swollen; the colour of the skin is somewhat dusky, sometimes yellow, sometimes natural; the head is large, the *fontanelles* widely separated, and sometimes all the sutures disjointed. The expression is stupid; the appetite is voracious; and much time is passed in sleep. The belly is swollen; the extremities are generally attenuated; the neck is thick, but not always goitrous; teething is not completed for many years, is generally accompanied by an offensive salivation, and frequently by convulsions. Usually the child cannot stand before its sixth or seventh year; and it is then that it begins to articulate certain sounds, supposing it has not been deaf from birth. The voice is hoarse and shrill, and words are spoken with difficulty." His stature is diminutive. His head is of great size, but flattened at the top, and spread out laterally. Dissections of Virchow show that the parts at the base of the cranium are early ossified in the form of a rectangle. Thus the character of the face in the cretin, as Dr. Tuke observes (*op. cit.*, pp. 105-6), remains unchanged from puberty to old age. The eyes are generally affected with strabismus; the zygomatic arch is large, and the mouth of large size; the lips thick, and the lower one hanging down. The lower jaw is small, retreating, and its angle very obtuse.

Treatment.—The indications of treatment are those which suggest improvement in all hygienic measures for the prevention of the disease. Dr. Guggenbühl, of Zurich, was the first to recognise the fact that the mental state of *cretins* could be improved by improving the growth and condition of the body. In 1842 he succeeded in buying the mountain of Abendberg, which incloses the plain of Interlaken, and there he established an hospital for these unfortunate children. The infant cretins, removed from the low close valleys (in which the malady too often finds the circumstances most congenial for its development), are there fed and trained in "the free, dry, cool, and bracing air of the open but sheltered and sunny slopes of the Abendberg." Once established, the condition of the cretin can only be ameliorated by institutions similar to those founded by Dr. Guggenbühl. The condition is beyond medicinal remedies.

DIABETES—*Syn.*, DIABETES MELLITUS.

LATIN EQ., *Diabetes*—Idem valet, *Diabetes Mellitus*; FRENCH EQ., *Diabète*—*Syn.*, *Diabète Sucré*; GERMAN EQ., *Diabetes*—*Syn.*, *Zucker Harnruhr*; ITALIAN EQ., *Diabete*—*Syn.*, *Diabete Melito*.

Definition.—*A constitutional disease produced through errors in the processes of assimilation either in the stomach, in the solid organs, or in the blood, and characterised especially by excessive thirst, an excessive discharge of urine, more or less constantly saccharine, to an extent of over 10 grammes (154 grains) of sugar per litre (35·3 ounces) of urine (LASEQUE), and associated with progressive emaciation of the body, often ending suddenly by coma from acetonæmia or by convulsions.*

Pathology.—(a.) *History.*—From the time of Charles II. of England, when Dr. Thomas Willis first observed the sweet character of diabetic urine, and suggested that it was due to sugar, no disease has had its nature more inquisitively examined, and with more interesting and instructive results. Nevertheless it still remains one of the most inscrutable of diseases; for opinions are by no means settled as to the fundamental points in its pathology. The abnormal state of the urine naturally at first led the inquiry towards the kidneys. They have been industriously examined, both as to their structural and functional relations, but without elucidating the nature of the change in the urine. Dr. Matthew Dobson, of Liverpool, in 1779, first established, by fermentation experiments, the fact that the sweetness was due to the presence of sugar. The next step in the inquiry was the detection of sugar in the blood of the diabetic patient. Ambrosioni, of Milan, in 1835, and Dr. Charles Maitland, in 1836, obtained crystals of pure sugar from the serum of the blood, and a large portion of fermentable crystallisable syrup. The late Dr. Robert Macgregor, of Glasgow, in 1837 (himself a victim to this disease), confirmed these observations by fermentation experiments with yeast, followed by those of Dr. G. O. Rees, of London, and Dr. Christison, of Edinburgh. Thus inquiry regarding the essential character of the disease was removed from the urine and the kidney to the blood; and research took a new direction, so as to ascertain, if possible, the source of the sugar in the blood and in the urine. Although this disease has sometimes found a nosological place under diseases of the kidneys, the researches of Bernard, Parkes, Pavy, Ringer, and others, very clearly show that amongst them it is misplaced. If the disease is to be regarded as a local one, it should rather come under *cerebro-sympathetic* or *hepatic* rather than *nephritic diseases*. But the weight of evidence clearly shows that *diabetes mellitus* belongs to the *constitutional* class of diseases; and hence it is only through a knowledge of the physiological processes of life “that we can expect to be able successfully to approach pathology with reference to this subject” (PAVY). The unusual discharge of urine in this malady was originally ascribed by Mead to a morbid state of the liver and bile; but subsequently nutritive and assimilative functions connected with the digestive canal were considered by Cullen, Home, and Dobson to give rise to the morbid state. That the process of digestion and assimilation in the stomach was the source of the evil has been hitherto the prevailing theory regarding the nature of this disease. The belief received confirmation, especially by the ingenious experiments of the late Dr. Robert Macgregor, of Glasgow, who ascertained that sugar was found in the stomach of diabetic patients during the process of chymification, even when no saccharine matter had been swallowed. Sugar has now also been detected in the saliva, the sweat, and in the stools. To the late M. Claude Bernard, of Paris, the science of medicine is indebted for the opening up of a new field of inquiry from which it is not too much to say that an altogether new era in the history of diabetes has begun. He must be credited with a great and important discovery—a discovery which has laid the foundation of our present position regarding the pathology of diabetes, and without which it is scarcely possible that any advance could have been made (PAVY). He endeavoured to show that one of the natural functions of the liver is to

generate sugar; and thus the sagacious speculations of Mead, which referred the phenomena of diabetes to a morbid state of the liver and bile, are now not only curious but interesting and instructive. While our knowledge regarding the transformation of sugar by the liver into amyloid substance (or "Bernardin," as Dr. Pavy proposes to name it), and its physiological relations to the animal economy in health and in disease, have been especially illustrated by Bernard, in France; it is to be observed that Dr. George Harley, of University College, and Dr. Pavy, of Guy's Hospital, London, have confirmed and cleared up many points, and especially elucidated the interesting doctrines of Bernard. Much is still unsettled as to the significance of the phenomena.

The experiments of Bernard appear to demonstrate that the animal organism has the power of forming sugar altogether irrespective of the nature of the food; and that sugar exists in a certain part of the circulation—namely, from the hepatic veins to the pulmonary capillaries—both in carnivorous and herbivorous animals. The liver he found to be this sugar-producing organ, as it was the only organ of the body which, in the normal state, he found to be impregnated with sugar. In man he examined especially the liver of five executed criminals, also that of a man who was killed by a gun-shot wound, and that of a diabetic patient who died suddenly from pulmonary apoplexy. In four of these he determined the absolute and the relative amount of sugar in the liver. The total weight of the three healthy livers was 4,205 grammes, which yielded of sugar a total of 66·074 grammes; so that the average weight of each liver being 1401·2 grammes, the weight of sugar yielded was 22·037 grammes, to compare with the liver of the diabetic case, which weighed 2,500 grammes, and yielded 57·50 grammes of sugar. He showed that the relative quantity of sugar varies little when the system is in a normal condition, very seldom exceeding 4 per cent. He especially believed he had demonstrated that sugar was secreted in the liver, and entered the blood from that organ; for, by a comparative analysis of the blood of the portal vein as it enters, and the blood of the hepatic veins as they emerge from the liver, he found sugar in the latter, but not in the former. Sugar he not only found in the liver of adult animals, but in the livers of the human foetus, the foetal calf, and the unhatched chick—thus proving that it does not merely accumulate there as a product from the digestive canal. He further contended that the sugar thus reaching the general circulation was destroyed by oxidation in the lungs. This view, however, has been since abandoned. The destruction occurs in the peripheral capillaries, and chiefly in those of the muscles, where it probably undergoes combustion, resulting in the production of carbonic acid and water, to which combustion it has also been suggested part of the heat engendered by muscular contraction is attributable. This is the doctrine comprehended under the "glycogenic theory." But, as Dr. Pavy puts it, the practical issue to be dealt with "is an ingress of sugar, on the one hand, into the general circulation through the hepatic veins, from alimentation and hepatic formation; and its destruction, on the other hand, in the peripheral capillaries. Presuming these two operations to be carried out, and to stand in such relation to each other that the destruction is equal to the ingress, there will be no accumulation within the system. Presuming,

however, the relation to be altered, and the influx to be increased so as to exceed the power of destruction, or, conversely, the power of destruction to be reduced below the rate of influx, it follows, as a necessary consequence, that accumulation of sugar will occur. The primary phenomenon in diabetes is accumulation of sugar in the circulatory system; and the question has to be put,—Which of the two factors is instrumental in determining the result?" Bernard concluded from his experimental induction of diabetes that the accumulation of sugar is due to an exaggeration of the formation of sugar, and not to a lessening of its destruction. Dr. Pavy's experiments show that there is only a small amount of sugar naturally existing in healthy blood; that corresponding with this small amount of sugar in the blood, there is a certain amount also (something under 0·5 per 1000) present in the urine; and that if any difference exists in the amount of sugar in arterial and venous blood, it is only of an insignificant nature. Experimental and other evidence show, that corresponding with the amount of sugar existing in the circulation there is elimination with the urine; and, therefore, whatever accumulation occurs becomes revealed by the condition of the urine. That healthy urine contains a minute amount of sugar shows that even with the small quantity that exists in the blood an escape of some of it occurs with the urine; and all other evidence is to the effect that according to the condition of the blood in relation to sugar so is that of the urine. Hence, the state of the urine furnishes an indication of the state of the blood. In proportion to the ingress of sugar into the circulation, so is the escape into the urine. In one case, with 1·543 per 1000 of sugar in the blood, the urine (being 30 fluid ounces in 24 hours, with a specific gravity of 1034) contained 31·76 per 1000 of sugar, or 14·40 grains to the fluid ounce. In another extreme case, with 5·763 per 1000 of sugar in the blood, the urine (being 232 fluid ounces, with a specific gravity of 1040, in 24 hours) contained 109·91 per 1000 of sugar, or 50 grains of sugar in the fluid ounce. Dr. Pavy, finally, maintains that instead of the liver being essentially a sugar forming, it is a sugar assimilating organ—preventing the sugar reaching the circulation to any material extent; but which, nevertheless, under a variety of deviations from the healthy state, it does do, and, as a consequence, finds its way correspondingly into the urine with all the phenomena of diabetes. In the diabetic patient the sugar reaches the general circulation in part derived directly from the amylaceous and saccharine principles of the food, and in part from the action of the liver—a true glycogenic action—upon nitrogenous matter; for, if lean meat alone be consumed, sugar in greatly diminished quantities is still found to be voided.

(b.) *Causation*.—The fault in diabetes essentially lies with the passage of sugar into the general circulation in place of another destination. The sugar escaping in diabetes, is the representative of carbo-hydrate materials which ought to be utilised in the system, but which, finding its way into the general circulation as sugar, is thence eliminated as it is. The main channel for the passage of sugar from the alimentary canal is the blood-vessels. Absorbed into the portal system it is conveyed to the liver, whence it becomes almost entirely checked in health in its onward progress, and prevented from entering the general circulation. It leads to an increased formation and accumulation of amyloid substance in the

liver. When not thus stopped and converted, it reaches the general circulation, and as a result gives rise to saccharine impregnation of the urine. Nitrogenous matter is also a source of this amyloid substance. What, then, is the cause of sugar passing into the urine in diabetes? Dr. Pavy's experiments showed that by altering the character of the blood going to the liver, an altered action occurs in the organ, which leads to the escape of sugar and the production of glycosuria. The effect of arterial or oxygenated blood introduced into the portal system, is to cause sugar to escape from the liver, to accumulate in the blood, and to appear in the urine. No new agent is called into play. The passage of blood through the vessels of the chylo-poietic viscera, in such a manner as to reach the portal vein in an imperfectly de-arterialised state, is subversive of the proper action of the liver, is productive of glycosuria, and supplies all that is required to account for the presence of sugar in the urine to the extent seen in diabetes. The state of the blood-vessels is also concerned in determining this; and the condition of the nervous system (by its influence upon the blood-vessels) stands at the foundation of the entire process.

The causation of diabetes has been also expounded by Dr. Pawlinoff, of Moscow, who, writing on the subject in Virchow's *Archiv.*, Vol. LXIV., July 1875, says that sugar cannot be oxygenated in the blood, but the muscles can decompose it into substances which are more easily oxygenated than albumen. In the normal organism, the oxygenation of albumen takes place principally in the arterial blood. By the oxygenation of albumen in the arteries, there is formed urea, while in the veins there is originated carbonic acid, by the action of oxygen upon the products of decomposition of sugar. In diabetes, the muscles cease to change sugar into substances which are easily decomposed, in consequence of which the process of oxidation loses its energy, as the albumen is oxidised with greater difficulty. Therefore, the consumption of oxygen is decreased, as well as the exhalation of carbonic acid. The urea which is now formed in excess in the arteries, as well as in the veins, and the accumulated sugar, absorb the water from the tissues, by which is caused the thirst of the patient, while his hunger is the sequel of the decomposition of albumen. Diabetes may be called forth artificially by poisoning with curare, which paralyses the intramuscular terminations of the motor nerves. The muscles, of course, cease to act upon the sugar in the blood, and consequently there is sugar in the urine. If a substance be introduced into the blood which is more easily oxygenated than those substances originated in the muscles, as albumen, then the consumption of these will be decreased. This takes place in case of poisoning by phosphorus, where paralactic acid appears in the urine. The consumption of albumen will be decreased, and consequently less urea will be formed, if paralactic acid be introduced into the blood. When the muscles do not produce paralactic acid, as is the case in diabetes, the albumen in the venous blood is no longer protected against oxygenation, and the quantity of urea is increased. Dr. Pawlinoff concludes that the formation of sugar in diabetes, and of paralactic acid in poisoning by phosphorus, make it apparent that the muscles turn, in a normal state, the sugar into paralactic acid, which becomes then further oxygenised in the blood. The oxygenation of albumen is

limited by the presence of paralactic acid. If this be no longer formed from sugar by the muscle action, the albumen in the blood is exposed to the influence of oxygen, not only in the arteries, but also to a greater amount in the veins; hence there will be a surplus of urea. Besides, there follows a decrease of oxidation in the organism, and a decrease in the exhalation of carbonic acid. As a result of these alterations, we have diabetes (*Brit. Med. Journ.*, Jan. 29, 1876). The inhalation of carbonic oxide has also given rise to glycosuria.

It has now also long been known that diabetes may be induced experimentally by puncturing in the mesial line of the floor of the fourth ventricle, in accordance with the original experiment of Bernard:—

“When we prick the mesial line of the floor of the fourth ventricle, in the exact centre of the space between the origins of the auditory and pneumogastric nerves, we at the same time produce an exaggeration of the hepatic (saccharine) and of the renal secretions; if the puncture be effected a little higher, we very often only produce an augmentation in the quantity of the urine, which then frequently becomes charged with albuminous matters; while, if the puncture be below the indicated point, the discharge of sugar alone is observed, and the urine remains turbid and scanty. Hence it appears that we may distinguish two points of which the inferior corresponds to the secretion of the liver, and the superior to that of the kidneys. As, however, these two points are very near to one another, it often happens that, if the instrument enters obliquely, they are simultaneously wounded, and the animal's urine not only becomes superabundant, but at the same time saccharine” (*Med.-Chir. Review*, l. c., p. 42).

The question now is,—How does the puncture act? In elucidation of this Dr. Pavy discovered that injury to certain parts of the sympathetic system also produced glycosuria, and looking to the medulla oblongata as a centre which must exercise an influence upon the liver, he directed his experiments to determine the channel through which this influence was exercised. He found that dividing the sympathetic filaments ascending from the superior thoracic ganglion (accompanying the vertebral artery in its canal through the transverse processes of the cervical vertebræ) produced strongly marked glycosuria; and also that glycosuria followed removal of the superior cervical ganglion. On the other hand, division of all the nerves immediately belonging to the liver, as they passed to the organ in company with the hepatic artery, hepatic duct, and portal vein, in every instance failed to produce glycosuria. These results have been confirmed by Cyon and Aladoff, Schiff and Eckhard.

The results of these experiments are,—That Bernard's puncture induces a hyperæmic state of the chylopoetic viscera. A dilatation of the small vessels of the intestines and liver ensues, producing a kind of paralytic hyperæmia of those organs (SCHIFF). The same condition has been induced through injury to the sympathetic. Blood unduly charged with oxygen reaching the liver by the portal vein, is just the state into which the portal blood is thrown by vaso-motor paralysis affecting the vessels of the chylopoetic viscera. It is a condition similar to the hyperæmic condition of the ear after division of the sympathetic in the neck,

where the veins also contain much redder blood than natural. Hyperæmia of the liver accompanies the excited flow of blood through the other viscera of the abdomen. Any agents or conditions also which cause a suspension of the functions of animal life, while the truly nutritive or organic functions remain intact, may bring about glycosuria. Thus the Indian woorari poison acts as it paralyses the intra-muscular termination of the nerves; and so does apoplexy produced by a blow on the skull. Local irritation of the liver itself, as Dr. Harley has shown, may also induce the condition. By the injection of alcohol and ether into the *vena portæ*, Harley was able to induce diabetes. The internal use of arsenic and quinine has also been said to have induced saccharine urine; and thus it is not improbable that irritant substances absorbed from the bowels by the mesenteric veins may sometimes bring about the morbid state. There are also various considerations connected with the disease which point to the brain as being the most likely part from which the morbid influence starts. By operating on the medulla oblongata and the sympathetic system, diabetes may be artificially induced; but in them only a part of the vaso-motor system resides. Eulenberg and Brown-Sequard have shown that the state of the arteries is affected by lesions of certain parts of the grey matter of the brain; and it is suggested that the vaso-motor nerves distributed in the sympathetic, besides being connected with the spinal cord and medulla oblongata, pass up to spots at the surface of the brain which stand in the position of cerebral vaso-motor centres. Dr. Pavy inclines to the opinion that some kind of textural change in the brain is at the foundation of diabetes; and that there are two ways in which the disease may be produced:—(1.) The vaso-motor system exerts an influence upon the arteries which gives them their *tonus*, or keeps them in a certain state of contraction. The effect of destruction or disease of the centres or tracts is to lead to arterial dilatation, by causing direct paralysis of the muscular coat, whilst that of irritation is the converse. (2.) By an action of the cerebro-spinal system controlling or inhibiting the activity of the vaso-motor system. To effect this a stimulus is transmitted from the cerebro-spinal system, which produces an inhibitory action upon vaso-motor centres or nerves that relax the muscular coat of the arteries, and so permits dilatation to occur. Diabetes may thus arise either from a lesion affecting and involving a loss of power in vaso-motor centres; or a lesion in some part or other of the cerebro-spinal system, leading to an inhibitory influence being exerted upon them. In spite of all these theories, the immediate cause of diabetes still remains obscure, and its beginnings are a mystery. According to Griesinger, diabetes occurs much more frequently in males than in females—nearly in the proportion of three to one. It occurs most frequently between the ages of thirty and forty in males, and in females between those of ten and thirty. The disease often shows an hereditary origin and transmission, several of one family consecutively falling victims to the disease. The usual recognised exciting causes are exposure to cold and wet, concussions of the body, injuries to the head, mental excitement, immoderate use of sugar, new wine, and fruit, great and continued mental exertion, mental depression, alcoholic intoxication, cerebral disease.

(c.) *Morbid Anatomy*.—No constant lesion is found. There is no remark-

able lesion in any part of the nervous system; but certain vascular and perivascular changes have been recognised by Dr. Dickinson in the brains of those who have died of diabetes. Ebstein found the seat of change to be either in the *medulla oblongata*, or the floor of the fourth ventricle; or portions of brain substance were found to be in a condition of inflammation, or of alterations from new growths, the latter exercising some pressure upon the medulla. Sometimes the floor of the fourth ventricle is diseased and sometimes healthy—nothing is constant. The pancreas is very often hypertrophied. The walls of the stomach are thickened, chiefly through muscular hypertrophy, with the mucous membrane thickened and softened. The lungs are almost always tuberculous, or contain caseous deposits, and not unfrequently there is pneumonia or gangrene.

In secondary lesions, the lungs are most frequently affected. In thirty-one out of sixty-four fatal cases, or in nearly a half, tubercle was found (GRIESINGER). Pavy and Wilks, however, do not believe this lesion to be that of tubercle, but the result of a secondary chronic inflammation, engrafted on the diabetes, by which the lung-tissue is broken down and cavities form. In about half the cases the kidneys are enlarged, and sometimes are in a state of chronic parenchymatous inflammation, as in some forms of Bright's disease. The liver usually appears normal, but is sometimes congested with blood. The bile resembles a mixture of rhubarb, and deposits a copious sediment of columnar epithelium and yellow amorphous granular-looking matter.

Symptoms.—The early symptoms of *diabetes mellitus* are obscure. The commencing period of true diabetes almost always escapes observation, the disease only exciting attention when it has become really established. Dr. Prout believed that there is a stage which precedes the formation of sugar, and which is marked by a superabundant and highly dense urine, loaded with an excess of urea. But much uncertainty prevails on this point, and nothing is assured except that the constitution is not greatly affected till the saccharine matter accumulates in excess in the blood, and is eliminated by the urine. In some very few instances the quantity of urine passed is hardly greater than in health; but more commonly it is in great excess, amounting to eight, ten, sixteen, thirty, and even more pints during the twenty-four hours; so that the patient is incessantly disturbed in the night, and loses his sleep, while the urethra and its orifice become inflamed and sore.

While therefore the onset of the disease is generally insidious and unobserved, there may be a sense of general discomfort, some emaciation may declare itself, while constant thirst and frequent micturition become well-marked symptoms, and among the first to attract attention. Practically, also, it is found that there are several classes of circumstances under which the presence of sugar in the urine presents itself, which differ in their accompanying phenomena, in their results and indications for prognosis, and which require different methods of management. These are:—(1.) The temporary malassimilation of saccharine and amylaceous articles of diet, producing an evacuation of diabetic sugar, sometimes intermittent or alternating with healthy urine, sometimes ending in complete health, and sometimes in the manifestation of an uric acid diathesis or gout. Any of these results may happen without special treatment. (2.) The

continuous malassimilation of an increasing proportion of saccharine and feculent material, increasing more and more the amount of sugar excreted under equal circumstances. This state of things may be identified by the sugar entirely disappearing from the urine when no food containing starch or sugar is taken, and reappearing when it is resumed. (3.) When not only the carbonaceous aliments, but also the nitrogenous, fail to be assimilated, and such of their elements as are capable of being converted into sugar are passed away in that form, the whole of the food is waylaid and robbed in its way to the tissues, and the patient is tortured by a more and more ravening appetite, and, at the same time, emaciated by starvation. (4.) Where the essential tissues of the body (notably the nervous) are degenerated in consequence of the deficient supply of normal nutriment (*Brit. Med. Journ.*, Oct. 2, 1875).

These several forms in which the disease presents itself are by some presumed to be stages eventually leading into each other. Others, again, consider that they do not necessarily do so: for, both the first and the second forms often cease spontaneously, or merge into other morbid conditions. The cases, too, of so-called "intermittent diabetes," in which the urine of digestion is saccharine, while no sugar can be detected at other periods, as in old persons, observed by Bence Jones, are cases in which life is in no wise shortened by the disease (*Med.-Ch. Trans.*, Vol. XXXVI., p. 401).

In middle-aged men also with an hereditary tendency to gout, we often find the first beginning of invalidism characterised by the appearance of sugar in the urine. This ceases, and its place is taken by uric acid and lithates; and then, perhaps, a swelling of the joints appears, and podagra fully declares itself—a disagreeable disease, indeed, but a subject of congratulation when it relieves one from the fear of confirmed diabetes (*Brit. Med. Journ.*, l. c.) The prognosis of the third form, in which we encounter true diabetes, is necessarily bad. The duration of life depends on the more or less amount of nitrogenous food which can be assimilated; usually, but not always, bearing a proportion to the notable symptoms. The general health begins to give way. Thirst is intense, and the patient often drinks many quarts, or even gallons, in the course of the day. The quantity of water drunk has been believed to be less than the quantity of urine passed: but from the careful experiments of Dr. Parkes and others to determine this point, it appears that the quantity of urine is actually *less* than the quantity of water taken, in liquid and solid food; and this is more especially seen to be the case if long periods (ten or twelve days) are taken for examination. Water is often retained for some time in the body of diabetics; and if an observation be confined to a single day, it may happen that some of the urine retained from the previous day is then poured out. It is such retention of water which may account for the apparent excess of urine over drink (PARKES *On the Urine*, p. 339). Hunger is also a characteristic symptom. The appetite is capricious, generally excessive, and often voracious; but some are also great eaters without excessive appetites, while a very sharp appetite may be very soon satisfied. But all diabetics are not voracious, and in a considerable number the voracity has preceded the appearance of the sugar. A great appetite is better than inappetence; and it cannot be too strongly stated that when after having eaten immoderately a diabetic patient

refuses food, the most grave prognosis must be pronounced. During the long period of exaggerated appetite it has also been observed that diabetic patients digest well, and with the same facility that is generally met with in great eaters. But they are liable to sudden and transitory diarrhoeas, with profuse evacuations, recurring at short intervals; and gastralgia has this peculiarity in diabetes, that it occurs prior to and not after meals.

The mouth is dry, and the tongue is large and often black at its middle part. It may also present in the worst cases an exceedingly injected appearance. Its bright red colour points to the existence of a hyperæmic state—the state that would be occasioned by a vaso-motor paralysis. The blood appears to be flowing through the organ without being deprived of its arterial character. But the state of the mouth is far from being pathognomonic in diabetes; for if in very well marked cases its dryness may be characteristic, in a great number of medium cases it remains quite normal. Some patients are liable to a pharyngeal uneasiness, somewhat analogous to that produced by skin eruptions. There is, in fact, a “diabetic pharyngitis” not dependent alone upon the altered condition of the saliva. On examination, the throat resembles that of an excessive smoker, the pillars of the pharynx being red and thinned, while the posterior wall is glistening. All prolonged vocal effort becomes intolerable; and, in some cases, patients have been compelled to renounce public speaking long before thirst or dryness of the mouth has come on. The teeth are often in a bad state, carious or breaking off. The origin of the diseased conditions of dentition has been so little studied that it is involved in obscurity. No one has adduced modifications of it as occurring in diabetes in young persons; and in older subjects there are so many causes that may produce these appearances. Again, some diabetic patients have their teeth quite uninfluenced. In advanced cases the drain upon the constitution is so great, that the alveolar processes are absorbed; and the teeth, loosened in their sockets, are apt to fall out. The saliva in diabetes is acid, and it becomes alkaline under the influence of food and stimulating sialogogues. In abundance it varies remarkably, being sometimes insufficient, and sometimes at intervals excessive—but in nowise affording a measure of the degree of the disease. Just as belladonna produces a most painful state of dryness easily distinguished from thirst, so the subjects of diabetes suffer from an ill-defined sensation quite independent of thirst, or even at the same time with it, which they compare to the feeling produced by dry dust when this pervades the atmosphere (BOUCHARDT, LASÈGUE). The skin is harsh and dry, especially at the commencement of the disease, and the patient becomes greatly emaciated, and loses sexual desire and sexual power. Almost all the water drunk passes off by the kidneys, and the insensible perspiration is diminished both by skin and lungs. The intestinal excretion of water is also greatly lessened; hence the bowels are costive and the fæces dry and hard. The water is not passed off by the kidneys so soon as in health. If a diabetic person drink water in the morning, the urine may not be increased till mid-day; but if grape-sugar be added to breakfast, the urine is passed as rapidly as in health. These symptoms are much relieved by medicine, and life prolonged; but often, when the case appears most favourable, destructive lung disease becomes apparent, and the patient sinks under this

complication. The emaciation of diabetes is progressive, and the muscles become atrophied, as well as the adipose tissue. Even the heart suffers. The skin is persistently dry, but on the appearance of fever, with increase of pulse and temperature, perspirations set in. As the disease progresses the digestive organs become weak and feeble, the inordinate appetite diminishes, and food is loathed. Nausea is frequent; a sinking at the pit of the stomach is complained of; the bowels become constipated, and the fæces pale. A short dry cough becomes frequent, the index of commencing disease of the lungs; and the patient generally suddenly sinks from exhaustion, or from coma or convulsions. The sudden occurrence of diabetic coma and death in a few hours are too often the startling results of a case of diabetes—after a prolonged, and distressing, yet comparatively uneventful course. Prout has noticed instances of this sudden ending. Some have regarded the phenomena as uræmic (B. FOSTER); others as due to thickening of the blood to a degree sufficient to render it unfit to circulate, from excessive loss of water. The nervous and respiratory systems have been invoked as the centres on which these grave symptoms depend. And, lastly, chemical transformations of the diabetic sugar, leading to the formation of poisonous compounds within the body, have been supposed by some German and Italian authors to be associated with such sudden death in diabetes. There are cases to which each of these hypotheses may be justly applied; but more recent experience warrants the last-mentioned view as the most satisfactory explanation of the hitherto obscure and alarming condition which has been aptly termed "*diabetic coma*." Dr. B. Foster, of Birmingham, has recorded some very interesting cases of this kind (*Brit. Med. Journ.*, Jan. 19, 1878).

"One morning when teaching in the wards, I was called away to see a boy who had been admitted two days before for diabetes. We found the boy in bed, tossing about restlessly, with a very anxious expression of face and slight duskiness of the skin. He had very remarkable dyspnœa. Each inspiration was deep, ample, and chest-filling. Large volumes of air passed in and out of the chest thirty-two times a minute, but seemed to have no power to satisfy the patient's craving for air. On examination, the chest was everywhere resonant, and loud and harsh breathing was heard all over. No odour beyond the ordinary diabetic smell was observed in the boy's breath. The pulse was small and weak, but regular, 136; the tongue was moist, with whitish fur; the temperature in the axilla was 97°. The boy was somewhat dull mentally, but when roused answered correctly. The symptoms had begun the evening before with *sickness*, which had occurred again early in the morning, and been associated with sharp pain in the epigastric region and rapid breathing. The abdomen was distended, especially in the epigastric region. When I noted these symptoms, I recognised the beginning of diabetic coma, and expressed to my class the opinion that the case was likely to end fatally in twenty-four hours. An injection to relieve the bowels, which had been confined for two days, an effervescing draught, with half-drachm doses of compound spirits of ammonia every two hours, and brandy in small doses, were ordered. The boy gradually grew worse; the surface grew colder, the face duskier; the rapid breathing lasted for twelve hours, and then grew short and catching; the pulse

became feebler and more rapid; the restlessness gave place to coma; the surface became cyanotic; and he died early the next day, less than twenty-four hours after my visit. The urine was passed freely in the bed during the last twenty hours of life.

"This boy had been diabetic for more than twelve months, and was admitted to the hospital, not on account of any urgent symptoms, but for the purpose of observing the effect of systematic treatment. On the day of admission, he had come by train ten miles to the hospital, as he was accustomed to do as an out-patient; and, when it was decided that he was to remain as an in-patient, he fretted a good deal, and continued to do so up to the time of the grave symptoms coming on. He was placed on partly restricted diet, and had no medicine, as I wished to estimate the sugar excretion before beginning specific treatment. He had been passing some 100 to 120 ounces of water daily before admission, with a high specific gravity (1035 to 1038), and containing 2,700 to 3,000 grains of sugar. On the day after admission, the urine fell in quantity and specific gravity (84 ounces, specific gravity 1026), and never rose above 1028 till his death. The sugar was not estimated. In this boy we had a typical example of the form of sudden death in diabetes, for which we have hitherto lacked any adequate explanation. The *post-mortem* examination was made by Dr. Saundby twenty-six hours after death. The brain and membranes were pale and anæmic, and there was no discoverable course or microscopic change, except hypertrophy of the muscular coat of the arterioles of the pia mater. The lungs and heart were quite healthy; the muscular structure of the heart was pale, but not fatty. The abdomen showed no sign of any inflammatory mischief. The spleen was small and soft. The liver was pale in colour, but normal in structure under the microscope, and weighed 3lbs. 3oz. A decoction showed the presence of sugar in small quantity. The kidneys were healthy. The stomach showed patchy congestion and catarrh. The blood in the brain sinuses, and throughout the body, was pale and cream-like. One observer compared it to grumous pus. It was fairly fluid, and did not clot, but, on exposure to the air, became pinker and brighter in colour, assuming a magenta-like tinge. The microscope showed the creamy condition to be due to the presence of a large quantity of molecular matter, which looked like fat, but did not dissolve in ether. There was a small quantity of sugar in the blood. These appearances were very striking, and pointed to the peculiar blood-change as the one abnormal condition to account for the patient's symptoms. Its nature still remained to be explained. While pondering over this problem, I came on a reference to the ideas of Petters and Kaulich, on the development of acetone in the system of diabetics. Acting on this, I at once determined to test the effects of acetone on healthy blood. Dr. Saundby willingly and ably assisted me. We found that, when acetone was added to the blood, it became paler and creamy-looking, presenting the same appearances as the blood of the boy. On exposure to air, the acetonised blood assumed after a time the same pinkish red coloration that had been seen in the *post-mortem* theatre. Under the microscope, the blood-corpuscles were seen to break down into granular *débris*, reproducing exactly the state observed in the blood of the patient. Roughly examined, it looked like blood containing a good deal of fat; but neither the artificially acetonised blood nor that of the boy owed its appearance to fatty matter. The addition to fresh blood of chloroform, alcohol, or ether, failed to produce similar effects.

In these results, then, we had a clue to the explanation of the case. Assuming for a moment that acetone was developed in the boy's system, its destructive effects on the blood would be amply sufficient to account for the great dyspnoea and the cyanosis, steadily increasing, in spite of the full and frequent inflation of his lungs. The blood-cells were so destroyed that they no longer were able to absorb and fix the oxygen drawn into the air-cells by the vigorous respiratory action of the patient."

Dr. B. Foster then sketches very briefly some of the observations made on this subject by others. Petters (*Prager Vierteljahrschr.*, 1857) was the first to show that acetone is developed in the system of diabetics, and Kaulich (*Ibid.*, 1860), in confirming this discovery, endeavoured to sketch clinically the state of acetonæmia. This state, he attempted to show, was not peculiar to diabetes, but was also met with in chronic affections of the stomach and digestive organs, and accounted for the depression of the whole nervous system observed in such cases. Betz (*Memorabil. für pract. Aertze*, 1861) followed, referring the presence of acetone in the breath to catarrh of the throat, &c.; and, some years later, Cantani (*Il Morgagni*, 1864; *Syd. Soc. Biennial Retrospect*, 1865-66) endeavoured to show that there were several forms of acetonæmia, occurring mainly in conditions of gastric catarrh. Alcoholic and acetic fermentations of grape-sugar, and possibly of other organic matters in the stomach, was assumed by these authors to be the sources of acetone. The following formula represents acetone: $\text{H}_3\text{C}_6\text{O}$. Since then, Bechamp (*Comptes Rendus*, 1872) has shown that alcoholic and acetic fermentations occur in the stomach under favourable conditions, such as catarrh. Rupstein (*Centralblatt für die Medicinischen Wissenschaften*, 1874) believes that the acetone may also be formed in the blood. It has been found there after death, and has also been obtained from the solid organs of a diabetic dying of coma by Berti (*London Medical Record*, September, 1874). Acetone is frequently found in the urine of confirmed diabetes, and is often developed in the urine after it is passed—an important fact to bear in mind in estimating the sugar. Dr. Foster has detected it in urine immediately after it was passed. Lastly, he refers to the important paper of Kussmaul (*Deutsches Archiv. für Klin. Med.*, Band xiv., 1874), who has observed cases of this kind, and experimented on animals with acetone; and to an excellent account of acetonæmia, with reference to most of the papers on the subject, in Lecorché's *Traité du Diabète*, Paris, 1877. The results of these experiments show that acetone is an anæsthetic less potent than ether and chloroform, and acting more like alcohol. It produces great muscular feebleness, quickens the pulse, and causes deep slow breathing, and in large quantities brings on stupefaction. The breath of the animals smelt strongly of acetone. Altogether the results obtained by Kussmaul's experiments closely resemble the symptoms of diabetic coma. Acetone produces partial anæsthesia, and in large quantities coma. It causes great muscular feebleness generally, but deep and energetic respiratory action. In man, before the coma comes on, there is partial unconsciousness, broken by a happy delirium, in which the patient laughs and jokes when roused, reminding one very much of the effects of alcohol. Summed up, the results so far go to show—

(1.) That acetone has been found in the breath, urine, blood, &c., of patients who have died of diabetic coma. (2.) That grape-sugar may be converted in the stomach by alcoholic and acetic fermentations into acetone. (3.) That the changes in the blood observed after death from diabetic coma can be artificially produced by the addition of acetone. (4.) That the administration of acetone in large quantities to animals produces similar symptoms to those observed in diabetic coma.

In such cases of *acetonæmia* the breath has a peculiar odour, strongly impregnated with acetone—a vinegar-like smell, likened very aptly to that of stale beer. The smell is unmistakable; every expiration, and every eructation of gas from the stomach, pours forth volumes of acetone, freely volatilised, on account of the high temperature of the patient.

The above case, in addition to others which Dr. Foster has seen, has led him to conclude that we have, in the acetonæmia hypothesis, the best explanation of diabetic coma. Patients who are seriously diabetic have, in all probability, a small quantity of acetone constantly formed in their economy. It is this which gives the odour to the breath and to the urine. It would appear that, under conditions of nervous depression and exhaustion, and more especially conditions favouring stomach-catarrh, the quantity of acetone formed may rapidly undergo great increase, and give rise to the acute symptoms which lead to sudden death; a chronic poisoning, as Kussmaul remarks, suddenly taking on an acute form, like delirium tremens in habitual drinkers. The similarity in the symptoms of some of these cases to the effects of alcohol make it highly probable that alcohol is also formed in the system. Senator, in his valuable treatise on Diabetes in *Ziemssen's Cyclopædia of Practical Medicine*, Vol. XVI., also holds this opinion. That in some cases there is no marked odour is to be accounted for by the fact that acetone requires a high temperature for free volatilisation. In some experiments recently made, Dr. Foster finds that from urine, for example, very little acetone is given off till the temperature reaches 100° Fahr.; but, above this point, it is given off quickly. The cases in which he has observed the smell of acetone most distinctly have been cases with high temperatures. The temperature of diabetics is not, as a rule, high enough to send it off in any quantity.

Where the disease affects the external genitals great distress is experienced by the diabetic patient, and especially by females. Professor Winckel has invariably found *pruritus vulva* in one of the following forms:—(1.) The slightest form, which is simply a *mycosis labiorum*; (2.) *furunculosis labiorum*; (3.) the most frequent and most obstinate form—a phlegmonous affection of the skin over the whole vulva, *mons veneris*, inguinal fold, sacrum, and nates. In all such cases of *pruritus* the urine ought to be examined for sugar. In this severe form the parts are of a dark red colour and considerably swollen, not always uniformly so, but in a knot-like manner. The redness and swelling do not disappear on pressure. The secretion is often considerable, which distinguishes this form of disease from *scrofulous vulvitis*; but, from scratching and rubbing, the parts become moist. The irritation creeps inwards, and the vagina becomes hyperæmic. The appearance of a fungus first supervenes as a complication *after* the vulvitis, which is

not due either to the fungus, or to the constant wetting of the parts with urine, or to the circumstance that the latter contains sugar. Professor

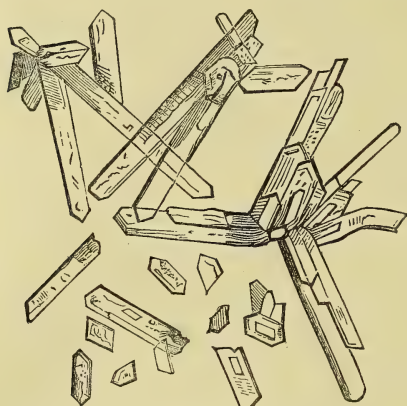


Fig. 107.*

Winckel regards the condition as eventually a phlegmon associated with the diabetes, and consequent on an impoverished condition of the blood (*The Practitioner*, Sept. 1876, p. 200). The cramps so common in this disease, affecting especially the lower limbs, the frequent occurrence of boils, and even of carbuncle, must be attributed to a like morbid condition of the blood.

Condition of the Urine.—The special sign of saccharine renal secretion separates diabetes from all other diseases in the nomenclature; and when *diuresis* is con-

siderable, the urine should invariably be examined, and its constituents determined. The excretion of sugar to the extent of over ten *grammes per litre*—that is, of 154 grains in 35·3 ounces—is the most characteristic symptom. A faint sweetish odour may then be perceptible in diabetic urine, comparable to fresh hay or milk. The chamber-vessel should be examined for crystals of sugar, which may have been formed (Fig. 107). The best evidence, however, is that derived from chemical tests.†

* Crystals of diabetic sugar from diabetic urine (after BEALE, p. 150).

† TESTS :—A portion of urine, which is usually of a light-straw colour, should be taken, and its specific gravity determined; and if greater than 1·020, it should be evaporated, and if sugar be present we shall have a dark brown residue, something like treacle. This extract, like the natural sugars, consists of crystallisable matter, and of an uncrystallisable syrup; and to separate them, Sir Robert Christison recommends that the extract be agitated with rectified spirit, and the residue boiled in another portion of the same fluid, when, on cooling, the crystallisable sugar will separate in light-greyish grains, like grape-sugar. Again, if sugar should be suspected to exist, even in minute quantity, a small portion of yeast should be added to a small quantity of the urine in a tube graduated to tenths or hundredths of a cubic inch, when, if sugar be present, fermentation will ensue, and each square inch of carbonic acid given off corresponds nearly to one grain of sugar. This test is so delicate, that one part of diabetic urine may be detected in 1,000 parts of urine of the density of 1·030. The most certain, reliable, and delicate test for sugar, is that known as "*Trommer's test*," based on the reaction of the salts of copper. A portion of urine in a test tube is to be treated with one or two drops of a solution of *sulphate of copper*, and afterwards a considerable excess of *potash* is to be added. The dark-blue solution which results is then to be held over the spirit-lamp, and boiled for a moment, when a yellowish-brown *precipitate* of the suboxide of copper is produced. Dr. Roberts, however, expresses a want of certainty in the results of most of those tests. He recommends Fehling's copper solution as the best test. It consists of sulphate of copper, gr. viii.; tartrate of potash, ʒss.; liquor potassæ, ʒi. Some of this test solution is to be boiled, and some drops of the suspected urine added to it. If sugar be abundant, a thick yellowish opacity and deposit of yellow suboxide are produced, which changes to a brick-red at once if the blue colour of the test remains dominant. If no such reaction ensues, more urine is to be added, until a quantity equal to the bulk of the test employed has been poured in. The whole must be again heated to the boiling point; and if no change occurs, it is to be set aside without further boiling. As the mixture of the urine and test cools, if no milkiness is produced, the urine may be confidently pronounced free from sugar. No quantity above a fortieth of a grain can escape such

The density of diabetic urine, however, is one of the best indications. This fluid varies in density from 1·030 to 1·074 (BECQUEREL); but on an average, and tolerably constantly, it is 1·040; and when the urinometer stands above 1·030, we may suspect that sugar is present. The quantity of sugar present has been calculated by Dr. Henry, in urine of density 1·020, to be 402 grains in every pint, while at 1·050 it contains 958 grains of sugar—the increment being, as he conceives, twenty grains, or nearly so, for every degree of specific gravity between the extremes that have been mentioned. If these data be correct, a person passing sixteen pints of urine daily, of specific gravity 1·050, actually passes nearly two pounds avoirdupois of sugar. But the amount varies greatly, amounting sometimes to *one pound or two pounds*, or even *two and a half pounds*, in twenty-four hours. In a few months patients will pass their own weight in sugar (PARKES, l. c.). Its amount is mainly

a test, and any quantity below that does not appear to be of clinical importance. It is important to bear in mind that Fehling's test solution must always be freshly prepared. Regarding Trommer's test, Professor J. Seegen, of Vienna University, makes the following remarks, and suggests a method of detecting small quantities of sugar in the urine:—"Trommer's is the most reliable and delicate test for sugar. With its aid, I am able with certainty to make out 0·3 milligramme (0·0046 grain) of sugar dissolved in 10,000 times the amount of fluid. This great delicacy of the test, however, only holds good as long as we have to do with a watery solution of sugar. If, on the contrary, small quantities of sugar are to be detected in urine, Trommer's test is neither delicate enough nor reliable, for two reasons: 1. Urine contains certain substances (colouring matters, creatine) which prevent the suboxide of copper when formed from being precipitated; no separation of the reduced suboxide of copper, therefore, takes place, the blue fluid only becoming yellow or yellowish-brown, or presenting a turbid discoloration. 2. The same processes of reduction are also brought about by uric acid; and urine, containing a considerable amount of uric acid, acts on Fehling's test fluid exactly in the same manner as urine containing 0·1 to 0·2 per cent. of sugar. The method devised by me has for its object the exclusion of those other constituents of urine which would disturb the proper action of the test, and the transformation, as it were, of the saccharine urine into a watery solution of sugar. Animal charcoal has the property of retaining most of the constituents of urine, more especially the colouring matters and uric acid. After filtering a watery solution of uric acid through animal charcoal, I could (provided the charcoal had been good), after repeated filtrations, not find a trace of uric acid in the filtered fluid. Now, in order to detect *small quantities* of sugar in urine, I proceed in the following manner:—I filter one or two ounces of the urine several times through good animal charcoal, until the urine is completely colourless. This operation only takes a few minutes. Then I wash the charcoal on the filter with a little distilled water, and to this water, when filtered off, I apply Trommer's test. The water with which the charcoal has been washed is almost as sensitive to Trommer's test as a watery solution of sugar, and in it I could detect even 0·01 per cent. of sugar by a beautiful red precipitate of suboxide of copper, whilst the original saccharine urine, when not filtered, only produces a yellow discoloration of Fehling's test fluid. With urine containing a little more sugar—say, 0·1 to 0·2 per cent.—the water flowing off from the second and third washing acts even more energetically upon the test-fluid than that of the first washing, producing an even purer deposit of suboxide of copper. The water obtained by the subsequent washings thus evidently contains the sugar in a purer form. With normal urine, the water obtained by the above process is either entirely inactive towards Fehling's test-fluid, which remains blue, or it assumes only after a while a slight dichroid (varying colour according as the light falls on or passes through) turbidity. The water obtained by a second and third washing always remains without any effect. When the *quantity* of sugar has to be determined, the urine must not be filtered through charcoal, as the latter always retains a certain quantity of the sugar, which cannot be removed again by washing. Sir Robert Christison gives the following formula for ascertaining the amount of solid matter in *diabetic urine*:—"Multiply the excess of the specific gravity over 1·000 by 2·33, the result is the number of parts of solid matter in 1,000 of urine."

influenced, in the first instance, by the saccharine and amylaceous nature of the food, which always augments the amount of sugar. The augmentation is quite perceptible about two hours after food, and continues for four or six hours, if the amount of starch taken has been considerable. It is probable, though it is not absolutely proven, that all the starch eaten is converted into sugar; and in cases accurately observed by Mr. Graham and Dr. Parkes for a considerable length of time, the quantity of sugar excreted by the urine never exceeded the amount of starch; and almost all the starchy food was accounted for by the diabetic sugar (WALSHE, PARKES). It has been supposed by some, that when the starch only is converted into sugar, it is the earliest stage of the diabetic state; and it is certain that during the progress of some cases sugar begins to be formed from other sources and in other ways, and is no longer derived solely from the starch. In some cases the sugar may recognise no other origin than the starchy food for many years. Such seems to have been the case of the late Mr. Camplin, of Finsbury Square, who kept his disease at bay for ten or twelve years, (*Med.-Chir. Trans.*, Vol. XXXVIII., p. 69; PARKES, l. c.) Complete fasting or abstinence from saccharine and starchy food for eight or twelve hours reduces the quantity of urine to the normal amount, and brings down the specific gravity to the usual figure. Sugar can then neither be detected in the urine nor in the blood, as was determined by Dr. Parkes, after numerous analyses of blood under such circumstances (*On the Urine*, p. 348). In the majority of advanced cases of diabetes, sugar is not only produced from starch, but also from *nitrogenous* foods, especially *gluten* and *animal food*. In such cases complete abstinence for a time from food lessens, but does not entirely remove, the sugar from the urine. Perhaps in all such cases the formation of sugar from albuminous food indicates a more advanced or active stage of the disease, than when the sugar is formed from starchy compounds only; and it is probable that the amount of sugar from this source increases as the disease advances. Dr. Parkes refers to a case related by Schultze, in which the amount of sugar in the urine when the patient was on mixed diet was *one-third* more than could have been furnished by the starch food alone; and the amount can always be ascertained by keeping the patient strictly on a meat diet, and feeding him with starch from time to time as an experiment (l. c., p. 348).

Thus, three conditions affecting food would seem to be present in cases of diabetes:—The earliest and fundamental one continues to act throughout, and is distinguished by complete arrest of the normal metamorphic changes of starch and sugar—an arrest probably associated with some substance in the digestive canal which absorbs the sugar. At first it affects the sugar derived from starch food, then it affects the sugar derived from nitrogenous food. Dr. Bence Jones suggests that in some cases the changes may pass one step beyond the conversion of starch into sugar, and produce “vegetable acid.” He believes a disease exists which is thus allied to diabetes, and characterised by a great amount of acid, probably *lactic*, in the system and in the urine. The chief transitional changes of starch enumerated by Dr. Bence Jones are as follow:—Starch into sugar, into vegetable acid, into carbonic acid; and he supposes the arrest to occur at the acid stage, instead of at the sugar stage, in the

morbid condition he describes. The second condition is an abnormal production (probably in the liver) of sugar from nitrogenous food—i.e., an amount of production which is abnormal—combined with arrest in the transformation of sugar normally formed (PARKES). A diabetic patient, kept on the most rigorous meat diet, has been known to pass a quantity of sugar corresponding to *two-fifths* of the entire meat food, or *three-fifths* of the *albuminate* contained in the meat (GRIESINGER, quoted by PARKES). A third condition may be also distinguished—namely, one in which the *tissues* themselves, and especially the muscles of the body, contribute to the morbid formation of sugar. This occurs under circumstances of extreme inanition, when almost no food is being supplied to the body, and yet a constant quantity of sugar is eliminated. Traube, Parkes, and Ringer have each recorded such cases. In one of Dr. Parkes' cases, the blood, after seventeen hours' fasting, was found to be still very rich in sugar (PARKES, l. c., p. 350). It is, however, a question whether this is an abnormal formation of sugar merely, or a further and more advanced stage of the disease. There are also cases which may be noticed here, in which the patients continue to lose ground although the quantity of sugar lessens, and in which the substance known as *inosite* or *muscle-sugar* is found in the urine (Fig. 108). It increases in



Fig. 108.*

quantity as the sugar lessens, and at last as much as eighteen and twenty grammes of pure *inosite* have been procured from the day's urine. The *inosite* may be obtained in the form of colourless prismatic crystals, which are efflorescent. It does not reduce the oxide of copper to the state of suboxide, as is the case with *diabetic sugar* and *grape-sugar*; and it is said to have not quite the same composition as the latter substance, but is represented by $C_2H_2O_2$; so that one atom of *grape-sugar* would thus represent six atoms of *inosite*. It may be detected by evaporation of the suspected fluid nearly to dryness in a platinum basin, when, if a little ammonia and chloride of calcium be added, a rose colour is produced, especially if the mixture be again concentrated by evaporation. M. Hohl records a case of diabetes in which, while the proportion of sugar gradually diminished, the *inosite* gradually increased in amount, till upwards of 300 grains were passed in the twenty-four hours (PARKES, BEALE). The observation is one of great interest in connection with the pathology of this remarkable constitutional disease.

Professor Sidney Ringer, of University College, has made some observations of great interest, to show the amount of *urea* and of *sugar* respectively furnished by the tissues of the body and by nitrogenous food:—(1.) During inanition one series of observations showed an

* *Inosite* or *muscle-sugar*, crystallised partly from alcohol and partly from water (after FUNKE).

enormous disintegration of tissues (48 grammes of urea and 105 grammes of sugar being passed in twenty-four hours), the relation between the urea and the sugar being tolerably constant. In the second series, when nitrogenous food was taken, the urea increased about the third hour after food, and reached its maximum about the fifth hour, after which it continued to diminish, and reached the inanition amount in the eighth hour. The sugar followed the same rule, and almost in an exact ratio; but the urea was in slight relative excess to the sugar, showing that the nitrogenous food raised the urea slightly more than it did the sugar. During inanition the urea was to sugar as 1 to 2.235, while after nitrogenous food the urea was to sugar as 1 to 1.9. It thus appears certain, as Dr. Parkes observes, that there is some close connection between the amounts of urea and of sugar in such cases as diabetes. The amount of urea may be double or even treble the normal amount, and that to an extent much greater than can be accounted for by the food taken, and due probably to some peculiarity in diabetes, causing heightened metamorphosis of tissues, such as might arise from the excessive action of oxygen on them (PARKES, l. c., p. 342). The amount of sugar is still further increased if diabetic patients take more water than their thirst demands; and it is probable that the urea is also increased, although exact experiments are wanting. The urine of diabetics often contains albuminoid matters, and sometimes albumen, in chronic glycosuria, which, as a general rule, is an unfortunate complication. Bright's disease, in its widest acceptation, is, however, rarely found added to diabetes, for when it complicates this the sugar diminishes. This is the gravest of forms. In most cases, as a result of simple fatigue, the secretory apparatus allows of the passage of albumen, and the prognosis in cases of this passive albuminuria is of somewhat less gravity (LASÈGUE). On standing, diabetic urine soon begins to ferment, with the appearance of *lactic, butyric, acetic, or formic acids*, and develops the yeast plant; and during this fermentation the urea entirely decomposes.

Prognosis.—Some of the grounds of prognosis have been already anticipated under the head of symptoms. Diabetes is "chronic in its course, usually lasting from one to two years, though it may run on for six, eight, or ten years." Becquerel mentions the case of a boy, nine years old, who died in six days, and Roberts a child of three years, who died in three weeks. Of 100 fatal cases collected by Griesinger, the duration was :—

Under 3 months,	1
Between 3 and 6 months,	2
" 6 " 12 "	13
" 1 " 2 years,	39
" 2 " 3 "	20
" 3 " 4 "	7
" 4 " 5 "	2
" 5 " 6 "	1
" 6 " 7 "	2
" 7 " 8 "	1
Undetermined,	12

The most common *complication* is a form of destructive lung disease, sometimes described as tuberculosis. It occurs in one-half of the cases

of diabetes protracted to the first year (ROBERTS). It is often quite acute; but the whole subject of phthisis engrafted on diabetes requires a careful examination, which is difficult in the present confused state of knowledge concerning phthisis. A low and fatal type of inflammation of the lungs, pleura, or peritoneum is not infrequent after the disease has lasted some time. In every tissue of the body there exists a tendency to asthenic inflammation, apt to run into abscess, diffuse suppuration, sloughing, phagedenic ulceration, or gangrene. Boils in successive crops, and even carbuncle, may appear in the course of the disease. In one of Dr. Roberts's cases, boils were an initial symptom. Spontaneous gangrene of the lower extremities, with obstruction of one or more arteries of the limb, is also not unfrequent (MARCHAL DE CALVI). Defective vision is met with in a certain number of diabetic patients. Cataract occurs in some long-standing cases; its frequency is variously stated. Of 225 cases collected by Griesinger, there was cataract in 20; but of his own 7 cases, it was present in 3; Von Gräfe states the proportion as 1 in 5; Bouchardat, 1 in 38; of 45 cases treated by Roberts, there was cataract in only 1; and Garrod says that in the large number of cases of diabetes he has treated he has never once seen it. Diabetic cataract is nearly always soft, though examples of the hard kind have been reported by Wilde, Von Gräfe, and Guersant. It comes on generally after the diabetic state has lasted eighteen months or two years; but it has been known to appear in six months. Its course is rapid; the two eyes may become completely cataractous in a few days; sometimes it is developed more slowly (ROBERTS). It begins in one eye—generally in the right—but soon involves the two. Drs. S. Weir Mitchell, of Philadelphia, and B. W. Richardson, of London, have endeavoured to show that it is due to physical imbibition by the lens of the saccharine matter of the aqueous humor; but there is no proof of this. Hoppe and Fischer both failed to find sugar in cataractous lens removed from diabetic subjects; and there are other reasons against the reception of this theory, particularly the infrequency of its occurrence, the long delay of its appearance, and its being occasionally unioocular. Dimness of sight, from supposed functional disorder of some of the internal structures of the eye (amblyopia) happens, according to Bouchardat, in about one-fifth of the cases of diabetes. Fauconneau Dufresne found the sight more or less affected in 20 out of 162 cases. Generally it is slight, temporary, and often recurrent. It is rarely a permanent affection; and when so, it ends in total blindness, and betokens a speedy fatal termination (CLYMER).

The ultimate issue of every case of diabetes is probably fatal; at least the number of cases in which the urine is rendered permanently natural is extremely small, and many of them, at the moment the disease seems to have yielded, die of destructive lung disease. Even when the presence of the saccharine principle has been so far conquered that it alternates with lithic acid deposit, or that lithic acid becomes the prominent feature, the circumstance is anything but favourable; for such individuals generally die of some sudden and overwhelming attack of internal inflammation or of apoplexy (PROUT). On the other hand, where the source and nature of the malassimilation can be discovered, and either rectified or held in check, and if the patient will submit to regimen, a favourable result may be hoped for

in some cases. A "not inconsiderable number, however, recover completely, and many more attain to a state of conditional amelioration—that is, an amelioration which is conditional on the observance of a certain diet and regimen." Dr. Trousseau says,—“By hygiene and a proper regimen, aided by the action of certain remedies, wisely and prudently used, we may hope to cure a few and to relieve a large number of diabetic patients, if the disease has not reached the last period.” The younger the patient the less is the chance of ultimate recovery. Dr. Roberts states that all the cases under twenty which he has seen have eventually died of the disease. In all persons the symptoms may continue for years, with a tolerable state of health. In corpulent persons the prognosis is more favourable than in those of spare habit. Saccharine urine, without excessive urinary secretion, is less serious than when the secretion is very great. “*Cæteris paribus*, the longer the disease has existed, the more unfavourable the prognosis; *cæteris paribus* also, the greater the general severity of the symptoms, the less is the hope of amendment. Cases which can be traced to mental anxiety and traumatic lesions appear to be somewhat more hopeful than those for which no tangible cause can be assigned” (ROBERTS). Great diminution in the amount of, or the total disappearance of, sugar in the urine, when saccharine and amylaceous articles of diet are not used, is a favourable sign; but “variations of more or less sugar are only of importance on condition of their duration—months, not weeks, being required to pronounce an amelioration as definitive. During the treatment of the disease, the practitioner has still more need than the patient to exercise unceasing watchfulness; and nothing is so wearying as chronic disease. The day the practitioner ceases to be exacting, the patient abandons the effort, and both commence to be contented with—the whereabouts. The daily measuring of the quantity of urine, and of the proportion of sugar it contains, maintains regularity of procedure, and with it that powerful element of success—determination” (LASEQUE). A soft, perspirable skin, with a moderate appetite, or the symptoms generally remaining stationary, are also favourable conditions. Albuminuria, thoracic and intestinal complications, and permanent cataract or amblyopia, are always fatal symptoms. Dr. Pavy’s clinical experience has led him to conclude that diabetes presents an alliance as regards the nature of its progress to *locomotor ataxia* and *progressive muscular atrophy*,—in each of the three diseases there are different manifestations of disordered nerve action; and that diabetes is a truly progressive affection, especially in young persons.

Treatment.—There are few diseases in which the treatment has been more varied than in *diabetes mellitus*. Every conceivable medicine has been given, but exact determinations of their effects on the sugar have rarely been made. The emaciated state of the patient presents an insurmountable obstacle to bleeding. Nevertheless, this mode of treatment has often been practised: but the pathology of diabetes, as given in the text, does not sanction general blood-letting as a mode of cure.

Mercury alike with *lead*, *antimony*, *zinc*, *silver*, and *copper*, are indifferent as to their influence on the formation of sugar (PARKES). *Opium* has been given to the extent of 100 grains in the twenty-four hours; but with an equal want of success, although it does seem to lessen the sugar,

probably by lessening the appetite and hindering the taking of food (PARKES). Nevertheless *opium* and its preparations, especially *codeia*, have among all known remedies the most decisive influence on the excretion of sugar; but the duration of the action is only exceptionally an enduring one. (SEEGEN). The whole *materia medica* has been exhausted in search of a remedy for this disease. The metals, the fixed and the volatile alkalies, the vegetable and mineral acids, the astringents, purgatives, tonics, diaphoretics, and diuretics, have each in their turn been administered, and each has perhaps afforded some relief; but the disease has proceeded, and finally, it may be said, nearly every patient dies whose treatment is left entirely to drugs. Dr. Prout, who considered diabetes merely as a form of dyspepsia, conceived that each case requires a special treatment. In the early stages of the disease some have found great benefit from the *tincture* of the *muriate* of iron, now called the *tincture of the perchloride of iron*. Dr. Camplin speaks of the *citrate of ammonia* in the effervescent form, generally combined with the *citrate* of iron, as more useful than any other medicine; while bitters and alkaline remedies did him great service at one period of his attack. Amongst remedies alkalies, as recommended by Miahle and Contour, and mineral waters have proved themselves the best. The use of Carlsbad water has always a favourable influence, the amount and duration of the influence varying with the severity of the disease (SEEGEN). The quantity of sugar in diabetes decreases under the use of alkalies, and at the same time the patient is better nourished. This action of the alkalies depends probably upon an increased oxidation of the hydrates of carbon in the blood of the non-muscular tissues, by which partly the want of oxygen in the muscles may be compensated. If the unknown cause cannot be removed, in consequence of which the muscles cease to decompose sugar, there are no remedies by which we might decompose the sugar in a diabetic diseased organism. But we may partly supply, in the blood of diabetic patients, what is wanted. Besides alkalies, we may use paralactic acid; and as glycerine acts in a similar manner as paralactic acid,—*i.e.*, is easily consumed in the blood—it deserves to be taken into consideration in such cases. It has been given with benefit to the extent of six to eight drachms daily; and its administration should be continued uninterruptedly for at least one month. In place of paralactic acid, there may be used lactic acid, as it is also consumed in the blood.

Opium, combined with ipecacuanha, is eminently useful as a sedative, especially in the form of Dover's powder; while exercise, warm clothing, friction of the surface, hot bathing, and diaphoretics, improve the cutaneous functions. The functions of the skin must always be inquired into, and must be kept active. The nitro-muriatic acid baths, and the internal administration of the acid, might also be employed with some prospect of benefit in cases requiring acid tonics. This is more generally the case in the young, and in the early stages of the disease, in which the *tincture of the perchloride of iron* is also of service, prescribed in the *infusion of quassia* or of *calumba*. But each case requires to be made a special study, considering that many, or at least several, organs are concerned in the disordered working of the system (PROUT, CAMPLIN). The *mistura ferri composita* is another of the iron preparations which has been found of

service. With the view of diminishing the morbid ferment in the stomach, Drs. W. Ebstein and Julius Muller, of Breslau, have prescribed *carbolic acid* with advantage. It was given dissolved in *peppermint water* (the strength being 1 gramme to 300 grammes), about six to seven tablespoonfuls of this solution being taken in a day. It has also been found of use in the treatment of the *acetonæmia* (B. FOSTER). One to three grains may be given in *glycerine* or in a pill, twice or thrice a day. *Valerian* has also been found of use in diminishing the excretion of urea (BOUCHARDAT), and so lessening thirst. He gives it in repeated doses, and gradually increases the amount taken until he has, in some cases, reached a dose of 30 grms. (450 grains) in twenty-four hours (*Med. Times and Gazette*, Vol. I., p. 568). With regard to the *acetonæmia*, *transfusion* has failed in this country and abroad. So also has the injection of a saline solution into the blood, as practised by Drs. E. H. Fagge and F. Taylor, of Guy's Hospital (*Guy's Hosp. Report*, XIX., p. 173 and 521). *Salicylic acid* and its salts are also worthy of trial, so is *thymol* (B. FOSTER, BURDON SANDERSON). In the severe form of *cardialgia*, I have found great benefit to result from the use of *Seller's Bismuth mixture*, prepared by Messrs. Mackay, Sellers, & Co., 1 and 2 Bouverie Street, London, in half-drachm doses, mixed with water, three or four times a day. Dr. Garrod writes favourably of the *sulphide of ammonium* as useful, in so far as it controls the morbid appetite; and Dr. S. Ringer finds *sulphide of calcium* of use. In the affections of the genitals in the female, I have found the intense pruritus to be best relieved by bathing in solution of *Condy's fluid* (*permanganate of potash*) in copious and frequent ablutions, or by vaginal injections *without soap*. Winckel has found a solution of one part of *salicylic acid* in 300 of water; but *glycerole of lead*, *zinc ointment*, *white precipitate ointment*, *glycerine and tannin*, *sitz baths with bran*, *carbolic acid solution* (1 grain to 4 ounces of water as a vaginal injection, or 5 grains to the ounce as a lotion), may each in their turn be of use in relieving these most distressing of the symptoms and complications of diabetes.

The little benefit derived from medicine induced Dr. Rollo to try the effects of an entirely azoted or animal diet; and now it is found that the regulation of the diet is of the first and prime importance in the treatment of diabetes. A full and generous diet is unquestionably useful; but the patient soon gets disgusted with mutton or beef, or both, for breakfast, dinner, and supper: he consequently nauseates a meat diet, and abandons it altogether. A diet of salt fish has been attempted, but the patient in a short time so loathes it that it has to be given up. A mixed diet, therefore, if contraindicated by some theories, is at least the best to adopt in practice, and is consistent with the remarks made under pathology, if duly regulated and aided by other means. It will be evident, however, that those vegetables which contain a large quantity of saccharine matter should be avoided, as potatoes, grapes, or other very ripe fruit, and *à fortiori*, sugar itself.

"However surely an exclusive animal diet may lessen, or entirely remove, the sugar in the urine of a diabetic patient, it is certain that it cannot long be tolerated." In this opinion Niemeyer, Trousseau, and

many other physicians now concur :—"Under its use the appetite fails, and a loathing of all food soon happens. The necessity of a mixed diet for man is as necessary for him when suffering from diabetes as in a state of health. In this disease the nervous system is undoubtedly implicated, and a rigid adherence to animal food alone, were it practicable, would soon be followed by an aggravation of the nervous troubles. There is, from the very beginning, and throughout the course of the disorder, a strong tendency to devitalisation, and this too must be guarded against. Fortunately the sugar in the urine may be kept down, and at the same time the general strength of the system maintained, by a properly adjusted diet of mixed food. Along, then, with the carnivorous dietary, whose importance is not to be undervalued, certain vegetables may be permitted, not only with impunity, but with advantage. These are, cabbage, cauliflowers, onions, spinach, water-cress, sorrel, endive, lettuce, &c. Trousseau has found no ill consequences from eating acid fruits, as strawberries, gooseberries, cherries, and he has allowed apples, pears, and grapes. He even suffers his diabetic patients to eat a small quantity of bread, if they greatly crave for it. As he remarks, there are many persons who are unable to make a meal without it. Such a regimen is more likely to keep the disease stationary, and secure the general comfort of the patient, by upholding his strength, than by confining him to one kind of diet, which his stomach soon revolts against, and which must result in innutrition, general debility, the development of intercurrent affections, and sooner or later death. Enforced daily exercise in the open air, when possible, just short of fatigue, is as of much importance in the treatment of diabetes as diet. In Professor Bouchardat's estimation exercise is the most efficacious of agents. Gymnastics should be practised. Trousseau says that he has repeatedly seen, during the hunting season, diabetic patients, abroad with their gun and dog, cease both to drink and urinate to excess, and regain their strength, and even their virile powers. A suit of flannel or buckskin should be worn next the skin. Warm alkaline baths should be frequently taken, and an occasional Turkish bath will be found serviceable, if it produces no disposition to boils; or daily packing with the wet sheet may be tried, carefully watching the effects. The whole body should be hand-rubbed daily. Cream, so long as it agrees, may be permitted, and cod-liver oil is in many cases well borne, and would seem, as a nourisher, to do good. The benefit of small doses of the alkalis, not continued longer than a week or ten days at a time, is incontestable. They are best given in the form of the Vichy, Marienbad Kreutzbrun, or Carlsbad Sprudel water. Legroux thought he derived benefit from the administration of arsenic. Dr. Richardson, of London, has recommended the respiration of oxygen, and Béranger-Féraud has shown that during its use there is great diminution in the amount of sugar in the urine (*Bul. de Thér.*, t. lxvii). The *ethereal* solution of the *peroxide of hydrogen* (ozonic ether) has been given with asserted benefit in half-drachm doses in an ounce of distilled water. Dr. T. K. Chambers has prescribed *iodide of potash* on purely empirical grounds. The preparations of *iron* are indispensable, and should be varied from time to time. To lessen the thirst and craving for food, small doses of *opium* are useful. By the use of the mixed diet recommended, particularly if fatty articles can be taken, the obstinate constipation, so common in the earlier stages of the disorder, may be obviated without a recourse to drugs. Should this not be the case, *castor-oil* in capsules, or *rhubarb* and *podophyllin*, must be prescribed.

"This mode of treatment, which secures a proper but varied diet, one that will not disgust the patient, but will nourish his body, though it may not remove the symptom glycosuria, will, in a large number of cases, give the diabetic a fair share of conditional health; so that, as Dr. Trousseau remarks, he would not know he had sugar in his urine if an occasional chemical analysis did not remind him of its presence" (Dr. CLYMER's American edition).

Nevertheless, there are, as Dr. Camplin justly observed, "certain fixed broad principles upon which the disease is to be treated." In all cases the various influences of the stomach, liver, skin, and kidneys on the nervous system and on each other, ought to be sought out and determined, and the basis of treatment arranged accordingly. *It is necessary to abstain from all amylaceous food, as well as from every solid and liquid containing sugar, or any substance readily convertible into sugar.* Fat meat and eggs may be taken, if biliary derangement is not induced by them, and fish is a most important article with which to vary the monotony of the dietary. Milk also may be indulged in occasionally, as it is not found that the sugar it contains is readily converted into glucose. Its influence, however, requires watching. The name of Dr. Donkin is associated with a special treatment of this disease in the use of skim-milk as the sole and only diet. He shows that in accordance with the teachings of chemistry and physiology we ought to expect advantageous results from it. Dr. Donkin contends that milk-sugar differs so essentially from cane sugar, grape sugar, and pure diabetic sugar, that it is not converted into it, and does not appear again in that form in the renal excretion. "Belonging to a different class of sugars, milk-sugar does not undergo *alcoholic* fermentation in contact with yeast; and, in addition, it does not precipitate the oxide of copper when treated with the reduction test. On the other hand, it is subject to *lactic* fermentation by the action of ferments. On account of these intrinsic differences, lactose, as an ingredient of milk, cannot undergo the same metamorphic changes as glucose in the processes of digestion and assimilation in health (nor be converted into it in diabetes); its conversion into lactic acid being *direct* and *immediate*, not by *intermediate* changes, through which the latter pass into this substance. For this reason milk-sugar, unlike vegetable glucose, is assimilated. It is necessary to notice, however, that milk-sugar, in the presence of acetic acid and some other vegetable acids, and of several mineral acids and other substances, was long ago observed by Simon to be changed into grape-sugar, and so became liable to alcoholic fermentation, and hence presumably as injurious to the diabetic as glucose. Hence milk-sugar may be thus perverted and rendered poisonous by admixture with other articles in the stomach, whereas taken quite alone, it may be the best of foods to the hungry patient. It is not, then, considered to be a fair trial of Dr. Donkin's skim-milk treatment, if any other food be allowed to mix with it in the stomach. It must be made the exclusive article of diet. The use of lactic acid by Professor Cantani, Dr. Balfour, Dr. Foster, and Dr. Ogle, is quite distinct from the treatment recommended by Dr. Donkin. They prescribed lactic acid as a remedy intended to make other foods wholesome; he prescribed skim-milk as a sole nutriment, which the mixture of

other foods nullifies. As to the quantity demanded, it appears that a patient can drink as much as a gallon and a half *per diem*: but that is an extreme ration; and when the appetite is so keen, Dr. Donkin advises some of the milk to be made into curd or "junks." But much less than a gallon and a half will suffice, for much less will supply the calculated daily wants of the digestive organs. Each pint of skim-milk contains half its weight of casein in a fluid state, which, in fact, is liquid bodily tissue. There is 72 per cent. of water in combination with it, and there is 72 per cent. of water in the primeest beef. Thus, in five pints of skim-milk a man would get as much nutriment as in $3\frac{1}{2}$ lbs. of beef-steak, which, in an exclusively meat diet, may be reckoned sufficient even to do a day's work upon. Dr. Donkin has, no doubt, made out a case for at least a temporary trial of this remedy in all but exceptional instances of diabetes. But the treatment must be commenced in the earliest stages. I have not found it of any use when the disease is persistently established.

It is desirable to vary the food as much as possible during the day, taking the lighter kinds in the later meals. When soups are taken, they ought to be really good, and flavoured with aromatics or onions, to the exclusion of carrots, turnips, and pease. They may be thickened with some bran *finely powdered*. Lettuces Dr. Camplin found to agree well, when eaten sparingly with oil and vinegar, or with a little salt only, if the vinegar is likely to disagree. Pickles in small quantities may be permitted to convalescents. If cocoa agree, it may be taken, prepared *from the nibs* only. With regard to drinks: if milk is found to agree, it may be used as a drink combined with half its bulk of lime-water or soda-water, or in the form of what is known as "buttermilk" in Scotland and in Ireland, but which in England is generally given to pigs, not yet being sufficiently appreciated by natives south of the Tweed. Dr. Camplin eventually found it necessary to abstain from all alcoholic drinks; but, where they are found desirable or necessary, a selection may be made from those wines and spirits which are freest from sugars. Of these, clarets may be chemically considered the best, then Burgundy. The so-called "fruity wines" must be entirely interdicted; and of all alcoholic beverages *weak* brandy and water is the safest. The amount of brandy must be always *measured*, and taken as directed by the medical attendant. From a *teaspoonful* to a *tablespoonful* in a tumblerful of water is generally sufficient for an ordinary dinner drink; and Dr. Camplin candidly and feelingly observes, from his own experience, that no diabetic need expect to recover or continue well who cannot exercise self-control, and make up his mind to be temperate *in all things*. Seeing that under this system of diet the patient is deprived of the use of ordinary bread, Dr. Camplin devised a form of bread prepared solely from *bran*; and the great value of *bran cakes* as a substitute for bread in cases of *diabetes* has now been established by the experience of so many individuals that its use ought to be insisted on. The bran used should be *thoroughly washed*, so that it may be as free from starch as possible, and *finely powdered*, so that it may not irritate the susceptible mucous membrane of the intestines. Such carefully-prepared and finely-powdered bran may be obtained from Mr. Batchley, of 362 Oxford Street, London, near the Pantheon; also of Mr. Donges, Gower Street, London, North. But if it is desirable to prepare the powder at home as it is

required, a special mill and sieve for the purpose are necessary, and may be obtained of Messrs. Evans Brothers, 54 Bricklane, Spitalfields, London, E. (Dr. CAMPLIN'S* *Monograph on Diabetes*.)

Consistently with the experiments of Bernard, "cod-liver oil holds out some prospects of a natural plan of treatment by its use." Dr. Pavy recommends ground almond powder, made into biscuits, rusks, and bread, with eggs, as a substitute for ordinary bread. Mr. Hill, 60 Bishopsgate Street, London, makes such biscuits.

An abstinence from water lessens the formation of sugar; but it probably accumulates in the body, so that when fluid is again given, an excessive elimination of sugar occurs (RINGER, GRIESINGER); and patients become extremely depressed and ill if water is withheld from them, probably from the impregnation of the body with sugar (PARKES). Coffee lessens the sugar, but increases the urea. Rennet, as recommended by Dr. Gray, of Glasgow, at first lessens the sugar and water; but they afterwards increase again. Warm baths lessen the amount slightly. When the diabetic symptoms subside, congestions, especially of the head, are apt to supervene. Such congestion Dr. Camplin found to subside gradually under the use of *citrate of ammonia* and small doses of *colchicum wine*. Warm flannel ought to be worn next the skin in all cases, and residence in a warm climate will often be of service as an aid to the means of cure employed.

The great difficulty in the treatment of diabetes is to manage the dyspepsia and impaired digestion, and, at the same time, to diminish and keep in check the formation of sugar. The first principle consists in varying the alimentation so as to prevent disgust; the second is to add fatty aliments in proper proportion to the alcoholic aliment, the exaggerated use of the latter acting injuriously on the nervous system; and the third is to suppress or considerably diminish for some time the use of feculents.

* The *formula* for bran cakes is thus given by him:—"Take a quantity of wheat bran (say a quart). Boil it in two successive waters for a quarter of an hour, each time straining it through a sieve; wash it well with cold water on the sieve, until the water runs off perfectly clear. Squeeze the washed bran in a cloth, as dry as possible, then spread it thinly on a dish, and place it in a slow oven. When it is perfectly dry and crisp it is fit for grinding into fine powder. The bran thus prepared is ground in the mill for the purpose, and must be sifted through a wire sieve of such fineness as to require the use of a brush to pass it through, and what remains on the sieve must be reground till it is sufficiently soft and fine. To prepare a cake, take of this bran powder three or four ounces, three new-laid eggs, one and a half or two ounces of butter, and about half a pint of milk. Mix the eggs with a little of the milk, and warm the butter with the remainder of the milk; stir the whole well together, adding a little nutmeg and ginger, or any other spice that may be agreeable. Bake in small tins (patty-pans, which must be well buttered), in a rather quick oven, for about half an hour. The cakes when baked should be a little thicker than a captain's biscuit. These cakes may be eaten with meat or cheese for breakfast, dinner, and supper, and require a free allowance of butter; and the cakes are more pleasant if placed in the oven a few minutes before being placed on the table. When economy is an object, when a change is required, or if the stomach cannot bear butter, the cakes may be prepared as follows:—Take of the prepared bran four ounces, three eggs, about twelve ounces of milk, with a little spice and salt, to be mixed and put into a basin (previously well buttered). Bake it for about an hour; the loaf may then be cut into convenient slices and toasted when wanted; or, after slicing, it may be rebaked, and kept in the form of rusks. Nothing has yet been discovered of equal utility to these bran cakes, combining, as they do, moderate cost with freedom from starch, and sufficient pleasantness as an article of food" (CAMPLIN, *On Diabetes*, third edition).

PURPURA.

LATIN EQ., *Purpura*; FRENCH EQ., *Purpura*; GERMAN EQ., *Purpura*;
ITALIAN EQ., *Porpora*.

Definition.—*A disease characterised by purple spots, patches, or merely points of effused blood, which are not effaced by pressure. They are usually met with on the skin alone; but they may appear simultaneously on the mucous membrane, when they are sometimes accompanied with more or less hæmorrhage.*

Pathology.—(a.) *History.*—By some this disease has been considered identical with scurvy, and been named the “land scurvy.” Others have considered it simply a disease of the skin—the *purpura simplex* of Willan and Bateman. Authors generally have spoken of the disease under the name of *petechiæ sine febre*, because the spots are not connected with any specific continued fever.

(b.) *Causation.*—The disease is very closely related to hæmorrhages as a morbid state on the one hand, and to scurvy on the other. Its causes are not well known. It is a disease more common in women than in men; and in boys before than after puberty. It has appeared under the long use of a poor diet, much sedentary occupation, watching, mental distress, and anxiety. It has also appeared under very opposite conditions; for example, in persons accustomed to the use of nutritious food and free living, but in whom there is reason to believe the digestive functions and the constitutional powers have been impaired by the persistent abuse of spirits or malt liquors. It has not been traced to any special dietetic default, not even to insufficiency of vegetable food. A predisposition to this disease often exists, which is also sometimes hereditary. Those families who suffer from the hæmorrhagic form are known in Germany by the name of “bluters.” It is very apt to recur, especially with children. The appearance of purpura is also sometimes connected with more or less disease of the heart, especially with hypertrophy or dilatation of the ventricles.

(c.) *Morbid Anatomy.*—It has not been determined what is the nature of the morbid state essential to purpura. In some cases the urine has been observed to contain an excess of albuminous matter with a deficiency of urea (COMBE).* The blood has been found deficient in coagulating power. An examination of the blood in two cases of purpura, by Dr. Parkes, showed that, with a somewhat diminished proportion of the solid constituents in general, there was a remarkable increase in that of iron. The lungs, the brain, the liver, and the alimentary canal are the most frequently affected. Purple spots and effusions of blood are to be found on the serous membranes, as in the *arachnoid*, the *pleura*, the *pericardium*, the *peritoneum*; and occasionally the blood lies coagulated in the cavities inclosed by these membranes. It has also been found in the bladder, and in the calyces and pelvis of the kidney (CRAIGIE). Instances occur in which blood oozes, or is discharged more or less copiously, from the

* An interesting case may also be referred to, related by Dr. Goodfellow in his admirable *Lectures on Diseases of the Kidney*, p. 74.

mucous membranes without affection of the skin; and to this class of cases, as a form of disease now under consideration, are to be referred those cases of profuse or fatal hæmorrhage from slight causes, recorded under the name of "*hæmorrhæa*," which have been ascribed to a *diathesis* termed the *hæmorrhagic*, and which is supposed to be hereditary, (*Edin. Med. and Surg. Journal*, Vol. XXV.) When the disease is protracted the patient becomes of a sallow complexion, waxy coloured, and dingy; anasarca swellings, gangrenous and bad sores appear on the feet and legs, general anasarca prevails, and he dies exhausted.

Symptoms.—Two varieties are to be distinguished, namely:—(a.) *Simple*, and (b.) *Hæmorrhagic*; which latter is thus defined by the College of Physicians of London—"The disease when accompanied by hæmorrhage from a mucous surface." Various vague symptoms, denoting general disorder of the constitution, precede the appearance of the petechiæ, generally by some weeks, such as languor, which is oppressive, weariness, faintness, and gnawing pains at the pit of the stomach. The appetite is variable, generally weak, but sometimes there is an inordinate craving for food, which, when eaten, is described as lying like a weight upon the stomach. The tongue is yellowish, and coated with a viscid fur, the countenance is sallow or dingy, or has a bloated, pale appearance, with swelling underneath the eyelids. The purple spots appear first on the legs, and afterwards, without any certain order, on the thighs, arms, and trunk of the body, and their presence is attended with much weakness and great depression of spirits. No degree of pressure alters the colour of the spots, and they are distinguished from flea-bites by the absence of a central puncture. At first the spots are bright red, but in a day or two they become purple, afterwards brown, and when they are about to disappear they assume a yellowish tint. When the disease continues for a long time, all these varieties of colour may be seen on a patient at the same time. The pulse is feeble, and generally a good deal quicker than natural. Deep-seated pains are felt about the epigastric region, as well as in the chest, loins, or belly. In some instances giddiness and lightness of the head prevail, especially when attempting to move or to stand erect, and there may be even dull pain in some part of the head. Constipation of the bowels, palpitation and irregular action of the heart, with a tendency to frequent syncope, are the most distressing and dangerous symptoms.

Dr. Graves has described an *exanthema hæmorrhagicum* in which febrile symptoms ushered in the malady, with an exceedingly dry and brown tongue, and a pulse which conveyed the sensation of small sharp vibrations with each beat—a kind of *wiry trembling*, with an extraordinary *thrill and hardness*, "yielding only to the withering influence of approaching death." Bleeding from the intestines and urinary system quickly supervened. These phenomena continued to spread till the whole mucous system of the patient became the seat of copious hæmorrhage. A peculiar exanthematous eruption appeared upon the skin, lasting for several days (five), and never presenting any signs of extravasation. Death followed in about four weeks. The pulse did not exceed its natural frequency (70), and there was no disturbance of the nervous system, such as headache, delirium, or want of sleep; and in the beginning of the disease the

strength was not remarkably impaired, but subsequently yielded to loss of blood. The disease seemed almost exclusively confined to the arterial and capillary systems of vessels.

Diagnosis is generally between *Purpura* and *Scurvy*. The following are the principal distinguishing features of each contrasted:—(1.) Scurvy is most common late in the winter or early in spring; purpura in the first seasons of summer and autumn. (2.) In scurvy the gums are uniformly soft, sore, spongy, swollen, and hæmorrhagic: this is no common feature in purpura. (3.) In scurvy tumors of a painful kind form in the extremities, with stiffness or contractions of the joints: such symptoms do not attend purpura. (4.) Scurvy is marked by extreme debility, and is rendered worse by blood-letting and by mercury, and is cured by the administration of lemon-juice: not so with purpura, which may require blood-letting for its cure, is not benefited by antiscorbutic remedies, and is sometimes speedily cleared away upon the supervention of mercurial salivation and hypercatharsis (WATSON and WOOD).

Prognosis.—A peculiar source of danger attends this disease in the occurrence of extravasation of blood into some vital internal organ, where a slight amount of hæmorrhage suffices to extinguish life. Such cases are described by Sir Thomas Watson. During the course of one week, in the year 1825, he was present at two inspections in the dead-house of St. Bartholomew's Hospital.

“The subjects of examination were both of them women of middle-age, who had been brought into the hospital covered with purple spots and bruise-like discolorations, and suffering hæmorrhage from the mucous membranes. Each of these women declared that the apparent bruises were marks of beatings received from her husband. One of them became suddenly hemiplegic a little while before she died. Of the manner of dissolution in the other case I am not sure. In both instances a considerable quantity of blood was spread over the surface of the brain between its membranes; and in one of them blood had been shed also into the cerebral substance, which it had extensively lacerated. It may be worth mentioning that in one of these corpses there were indications either of unusually rapid putrefaction after death, or (what I think more probable) of some degree of decomposition even before life was extinct. This woman died in the evening, and the body was examined next day, twelve or fourteen hours afterwards. A quantity of fetid gas escaped from the cavity of the abdomen as soon as it was opened, and small bubbles of air were seen to ooze from the areolar tissue of various parts of the body. Even when incisions were made into the *liver*, air frothed up, as it might do under ordinary circumstances from a section of the lungs” (*Lectures*, Vol. II., p. 866).

Treatment.—To treat this disease with success, it is necessary to ascertain the circumstances under which it becomes developed in each particular case. Its treatment may be comprised in the following measures:—The bowels ought invariably, and without exception, to be first thoroughly and effectually evacuated by means of *senna*, *aloetics*, or *calomel* and *jalap*. *Oil of turpentine*, administered in moderate and repeated doses, has also been recommended. Dr. Hardy, of Dublin,

recommends the tincture of *larch bark*. He has long used it as a styptic and carminative tonic; and it is "one of the most elegant forms of prescribing a terebinthinate" (MOORE). *Fifteen drop doses* of the tincture may be administered every hour, or *eight or ten drops three times a day*, afterwards increasing the dose according to the age of the patient and the necessities of the case. In young subjects otherwise robust, when the disease has appeared after violent exercise or excess, blood may be drawn from the arm, as practised by Dr. Parry of Bath. Under such circumstances also the young patient should abstain from animal food in every form, and should subsist on boiled rice with whey, or the light subacid fruits, as grapes, oranges, strawberries, gooseberries, baked apples, and the like. His drink may consist of tamarind-water, or water acidulated with sulphuric acid. Under this plan most cases of the disease in the young will be speedily and readily brought to a favourable termination. If symptoms of local uneasiness continue after the urgent phenomena have disappeared, leeches should be applied in the neighbourhood of the part; and it will be proper to continue the periodical and regular evacuation of the alimentary canal. When the spots have disappeared and the hæmorrhage has ceased, the constitution recruits rapidly under the gradual but cautious use of light soups and fresh fruits and vegetables (CRAIGIE).

SCURVY.

LATIN EQ., *Scorbutus*; FRENCH EQ., *Scorbut*; GERMAN EQ., *Scorbut*;
ITALIAN EQ., *Scorbuto*.

Definition.—*A morbid state characterised by sponginess of the gums and the occurrence of livid patches under the skin, of considerable extent, which are usually harder to the touch than the surrounding tissue. An altered state of the albumen of the blood is associated with this condition, and the phenomena are brought about by a deficient supply of the organic vegetable acids, or of the salts of fresh vegetables. It is ushered in by debility, lassitude, lowness of spirits, attended by fetor of the breath, and the gums swell by irritation, till they overhang the teeth in palmated excrescences. The livid subcutaneous patches and spots appear upon the skin of considerable extent, especially on the lower extremities and among the roots of the hair. Spontaneous hæmorrhages may take place from the mucous canals; contractions of the muscles and tendons of the limbs occur, with pains, and sometimes superficial ulcerations.*

Pathology.—(a.) *Historical Notice.*—The old English word is *Scorbie*, probably derived from *Scorbs*, a Slavic root-word signifying disease in general; and although there is considerable difference of opinion as to the origin of the word "*scorbutus*," it seems at least certain that it is only a Latinised variation of the vernacular name of the disease among northern nations, whether "*scorb*," "*schorbuk*," "*scharbock*," or "*skorbut*." It is mentioned by Pliny as having occurred in the Roman army commanded by Germanicus after a long encampment in Germany beyond the Rhine. It prevailed to a frightful extent in the army of Louis IX., when he was made prisoner in Egypt, in 1260; and the narrative of the campaign furnishes the earliest unequivocal description of the disease. With fish

only for fresh provisions, dispirited by being obliged to act on the defensive, and harassed unceasingly by the Saracens, the Christian army was almost annihilated by a most terrible outbreak of scurvy. It was observed to exist much earlier on land than on sea; and it unquestionably existed in the north of Europe from the most remote antiquity. It is not till 1555 that we have a particular description of it on land, when Olaus Magnus tells us that it infected chiefly soldiers in camps, and persons shut up in prisons or in besieged towns. In 1645 it prevailed among the Danes and other northern nations; in 1775 it was common on the borders of the Baltic among the peasant population, artificers, and miners; also in several parts of Scotland (where it was known by the name of *black legs*); and endemic in some seaport towns of Devonshire and Cornwall, especially among fishermen and tradesmen; but was seldom met with among agricultural labourers, who drank cider and eat plentifully of vegetables and fruits (LIND). The latter part of winter, and the early part of spring was the season in which scurvy prevailed most; and it uniformly disappeared during summer and autumn. It was thus endemic in the spring of every year two centuries ago in all the northern countries of Europe. It became gradually less frequent as agriculture and gardening improved; and we have witnessed the almost complete extinction of this disease *on land* as the influence of these arts has extended to the most remote parts of Europe, and to the humblest classes (BUDD). "The causes which, in the middle of last century, had rendered scurvy less frequent on land, have continued to operate with increasing efficiency; so that at present, except under peculiar circumstances, the disease is never met with in England, and rarely in any of the northern countries of Europe. That it should, a century or two ago, have been endemic in many parts of England seems almost incredible, when we consider the circumstances under which it arises, and the present aspect of the country; but we have undeniable evidence of the fact, and it affords proof of the extraordinary change which a few centuries have wrought in the cultivation of the soil, and in the habits of the people, especially with reference to the increased consumption of vegetable food. This is confirmed by the historical fact that, up till the sixteenth century, in the dietary of the people of this country all succulent vegetables are conspicuous by their absence (FROUDE, Vol. I., p. 47); and that until the commencement of the sixteenth century no salads, carrots, turnips, or other edible roots were grown in England. The little of those vegetables that was used before that time was imported from Holland and Flanders; and in the reign of Henry VIII., Queen Catherine, when she wanted a salad, was obliged to despatch a messenger thither on purpose" (HUME, *History of England*, Vol. IV., p. 241, quoted by Dr. BUDD). Vegetables were then both scarce and dear. "From the middle of the latter end of the eighteenth century no potatoes were kept for winter use, and the only kind of garden stuff used was a coarse kind of kale, the leaves of which were prepared for the table by boiling. This vegetable was tough and unpalatable; and during four or five winter months the food consisted almost exclusively of meat, bread, and flour pudding. The change which extensive cultivation of the potato and diffusion of gardening has wrought in the nature of the food of the people generally is remarkably great, and

well deserves the attention of physicians" (Dr. BUDD). It was in seasons of scarcity, however, during the desolation of war, and during long sieges, that the ravages of scurvy were chiefly felt. In 1625, during the siege of Breda, in North Brabant, by the Spaniards, which lasted eight months, the inhabitants and the garrison suffered severely; and when, on the 16th of March, an account was taken of the sick, 1,608 soldiers were found affected. The numbers of sick increased daily till June, when the town surrendered. In 1703, during the siege of Thorn in Prussia by the Swedes, which lasted only five months, during the heat of summer 5,000 of the garrison, besides a great number of the inhabitants, died of scurvy—the besiegers being, at the same time, quite free from it. In the early part of last century the disease was very common and very fatal in the Russian armies (LIND, BUDD). In the spring of 1760 it prevailed to a great extent among the English troops that formed the garrison at Quebec. These troops, at first 6,000 men, suffered so much from cold and want of vegetables and fresh provisions, that before the end of April 1,000 men were dead of scurvy, and twice that number unfit for service (SMOLLETT'S *History of England*, Vol. V., p. 198, quoted by Dr. BUDD). In the spring of 1795, scurvy was very general among the French soldiers in the army of the Alps. Fodère then treated between seven and eight hundred soldiers affected with it. In 1801, during the siege of Alexandria, it prevailed amongst the inhabitants and garrison to a frightful extent. The siege only lasted four months; but during that time 3,500 scorbutic patients were received into the military hospitals (LANEY, quoted by Dr. BUDD).

From the earliest times scurvy has also been observed to occur occasionally in persons long confined in prisons, asylums, and even in bedridden patients, and patients in hospitals whose diet has not been properly attended to. In the spring of 1823 the inmates of Millbank Penitentiary thus suffered, and in conjunction with dysentery—a complication occasioned by a diet of which fresh succulent vegetables formed no part, and the quantity and quality of which were not adequate to the support of health (LATHAM). The reports of the Inspection of Prisons for 1836, 1837, and 1838 abound with instances of the occurrence of scurvy in our gaols and prisons. At Norwich, for example, in 1836 it assumed a very malignant form in the county gaol.

Thus during the sixteenth, seventeenth, and earlier part of the eighteenth centuries, the disease was endemic in towns, fortifications, camps, armies, prisons, gaols, asylums, and hospitals, and originating under one common influence—namely, the prolonged use in confinement of a diet in which fresh succulent vegetables formed no part. "Many thousands were often cut off within a few months in single armies and garrisons; and it is probable more men perished from scurvy alone than from all other causes combined, whether sickness, tempest, or battle" (WOOD). In this country and in America it is now very little known on land, and many have never seen the disease, unless they have lived in seaport towns. Dr. Clymer states, in the American edition of this work, that scurvy has always prevailed in the United States army to a considerable extent, and under the same circumstances—a deprivation of fresh vegetables. In the column which marched on the city of Mexico

the men for some time previous to their landing at Vera Cruz, during the siege, and afterwards, could obtain no vegetables; and on their arrival at Jalapa, although there had been plenty of fresh beef, there was scarcely a man who did not have a scorbutic taint. During the late civil war, scurvy in some form or another was generally prevalent in the American armies. It first appeared to any extent in the army of the Potomac, at Harrison's Landing, Va., in July, 1862; and from that time forward until the end of the war, in 1865, it continued an increasing and formidable disorder. "It occurred in all the armies subjected to hardships, especially in the West; and its worst and most fatal manifestation was made after the war had closed, and during the occupancy of the frontier of the Rio Grande by the Twenty-fifth Army Corps," and in consequence of the want of fresh vegetable food (*U. S. Sanitary Commission Memoirs*). "What has been written of scurvy in the British army during the Crimean War, is equally applicable to our own during the late American War. The returns convey but a faint conception of the disastrous part which it [scurvy] acted among the troops; for though it comparatively rarely presented itself in well-defined forms, and as an independent affection, yet the prevalence of scorbutic taint was widespread, and in a vast proportion of cases evident indications of it existed as a complication of other diseases, especially fevers and affections of the bowels" (*Medical and Surgical History of the British Army*, 1858). "In the first years of the war scurvy did not prevail amongst the Confederate troops apparently to any extent. But it progressively increased with a diminishing commissariat and increased hardships" (J. JONES, in *San. Com. Memoirs*). The same authority, in his report on the diseases, &c., of the Andersonville Prison, states that scurvy, arising from sameness of food and imperfect nutrition, caused either directly or indirectly nine-tenths of the deaths amongst the United States army prisoners confined there."

But it was not till navigation was improved and long voyages undertaken that this disease became well known from its general prevalence and formidable character. The earliest account of the occurrence of scurvy at sea is to be met with in the narrative of Vasco de Gama, who discovered a passage to the East Indies by the Cape of Good Hope, in 1497. He lost 100 men out of 160 by this affection. James Cartier in his second voyage to Newfoundland, in 1535, speaks of sufferings still more severe. "With such infection did the sickness spread in our three ships, that of 110 persons that we were there were not ten whole, so that one could not help the other. Eight were already dead and more than 50 sick, and, as we thought, past all hope of recovery. This malady being unknown to us the body of one of our men was opened, to see if by any possible means the occasion of it might be discovered and the rest of us preserved. But in such sort did the malady increase, that there were not above three sound men left. Twenty-five of our best men had died; and all the rest were so ill that we thought they would never recover again." A decoction of the leaves and bark of a tree, supposed to be a species of spruce fir, was the remedy which they found restored health and recovery. "It was scurvy which used to decimate our navy, and render long sea voyages almost impossible. It was mainly by scurvy

that Anson, in his celebrated voyage of 1740-44, lost within the first ten months nearly two-thirds of his crew, and during the remaining period about half of the survivors. Scurvy continued to prevail with little abatement till 1764. The narratives of the voyages of Sir Richard Hawkins, Hozier, Anson, Sir Francis Drake, Cavendish, Dampier, Byron, and of numerous other navigators, furnish similar details, and show how recklessly the lives of sailors were sacrificed (COPLAND).

But the early history of navigation, as it records the greatest ravages of scurvy, so does it also record the best antidote to the disease. Of four ships which sailed from England on the 2nd of April, 1600, with 480 men, under Commodore Lancaster, for the establishment of the East India Company, all were so severely visited by scurvy as to have lost nearly one-fourth of their crews when they arrived at the Cape of Good Hope, and the merchants who had embarked in the adventure were obliged to do duty as common sailors. The crew of the Commodore's ship was not attacked. This immunity arose from three tablespoonfuls of lemon juice having been served every morning to each of his men; while the other ships were so sickly that the Commodore was obliged to send men on board to take in their sails and hoist out their boats; and there died at sea and on shore at Saldanha 105 men, nearly one-fourth of their whole number (PURCHAS'S *Collection of Voyages*, quoted by Dr. BUDD). But notwithstanding this evidence of the success of lemon juice in preventing scurvy—evidence the most conclusive—this valuable remedy and preventive was altogether slighted for a hundred and fifty years afterwards (COPLAND). Lord Anson's people, in 1740, on reaching the Island of Tinian, were recovered principally by eating oranges. At the end of two years after leaving England, they had lost a larger proportion from scurvy than four in five of the original number of their crew. That noble, brave, and experienced commander was so convinced of the usefulness of oranges, that before he left the island he ordered one man from each mess to lay in a stock for future security. Sir Charles Wager's people, also, were terribly afflicted with scurvy in the Baltic in 1740. Sailing, however, in the Mediterranean, and having heard how effectual oranges and lemons were in the cure of this disease, he took on board at Leghorn a large quantity of them, ordered a chest each day to be brought on deck, and allowed the men, besides eating what they chose, to mix the juice with their beer, and to pelt each other with the rind, so that the deck was strewed with the fragrant liquor. By these means he brought his men home in good health (MEAD'S *Discourse on Scurvy*). But it is mainly to the sagacity of Captain Cook that we are indebted for the first impulse towards those improvements in the treatment of sailors by which scurvy is now so effectually preventible in our navy. In 1772 he sailed from Plymouth on the 13th of July, in the "Resolution," with a company of 118 men, performed a voyage of three years and eighteen days in all climates, from 52° north to 71° south, with the loss of one only of his crew from disease. In the year 1747 Dr. Lind made some comparative trials between this and some other modes of treatment (as vinegar, sulphuric acid, and tamarinds) on board the "Salisbury," at sea. As a general conclusion from his experiments, he affirms that orange and lemon juice, or more properly the citric acid obtained from all the species

of the botanical genus *citrus*, or the natural order of fruits called *Hesperidæ*, are greatly more efficient than any other remedy in the cure of scurvy. Notwithstanding this strong opinion of Dr. Lind, the navy continued to suffer severely from scurvy for half a century, till the Admiralty gave a general order for the supply of lemon juice. This salutary measure was accomplished by a representation from the Medical Board of the Navy, in the year 1795, when Lord Spencer was First Lord of the Admiralty, after a trial made on board the "Suffolk," a seventy-four gun ship which sailed from England on the 2nd of April, 1794, supplied with a quantity of lemon juice sufficient to serve out two-thirds of a liquid ounce daily to every man on board, and this was mixed with their grog, with two ounces of sugar. She arrived at the Madras roads, on the 11th September, after a passage of twenty-three weeks and one day, without having had any communication with the land, without losing a man, and having only fifteen on the sick list. Scurvy appeared in a few of the men during the voyage, but disappeared on an increased dose of lemon juice being administered. "Let this fact," says Sir Gilbert Blane, "be contrasted with the state of the channel fleet in 1780, when Admiral Geary's fleet returned into port, after a ten weeks' cruise in the Bay of Biscay, with 2,400 men ill of scurvy; and let the state of this fleet be contrasted with that of the channel fleet in 1800, which, by being duly supplied with lemon juice, kept the sea four months without fresh provisions, and without being affected with scurvy."

In 1780 the number of cases of scurvy received into Haslar Hospital was 1,457; in 1806 *one* only; and in 1807 also *one*. The mortality in the navy had in fact been gradually decreasing since 1780, when various improvements were made in the victualling of the fleet and in the general treatment of the men; but in 1795, when a regular supply of lemon juice was first granted, the mortality fell *suddenly*, and to a degree scarcely credible (Dr. BUDD). Between the year 1779 and 1813 the diminution of sick and of deaths in the British Navy was in the proportion of four to one nearly (Sir J. BARROW, *Supplement to Encyc. Brit.*, Art. "Navy"). While it is notorious that many medical men have never seen the disease, it is, as Dr. Budd has assured Dr. Watson, by no means rare in the Hospital Ship at Greenwich, which is often full of cases of scurvy, most of the patients so affected having just arrived in *merchant ships* from a long voyage. Rumours are even now abroad to the effect that scurvy is more frequent in the merchant service than heretofore; and, if so, the least that can be said is, that it is a disgrace to the merchant princes of a nation like Great Britain. With regard to the prevalence of scurvy among sailors in the merchant service, Dr. Barnes (when Physician to the "Dreadnought" hospital ship) reported in 1864 to Mr. Simon that "of the entire number of cases admitted during the last twelve years into the Sailors' Home at Poplar, nearly half are, at the time of their admission, suffering more or less from scurvy, and of these perhaps a twentieth part seriously diseased with it; that of the entire number of cases admitted during the last twelve years into the hospital ship 'Dreadnought,' cases of scurvy have formed a twenty-fourth part; that to these must, of course, be added sufferers who are not taken into such establishments—a large but uncertain number of cases taken into the low lodging-houses of the water-side; but

that this is for London alone, and is not nearly all due to London ship-owners; that of eighty-six cases of scurvy treated in the 'Dreadnought' during 1863, only fourteen came from ships which had issued from the port of London, while thirty-one came from foreign ships, twenty-one from Liverpool ships, eight from Sunderland ships, and twelve from Glasgow and other British ships; that Liverpool ships, besides furnishing to London a large proportion of the scurvy which is treated here, convey probably a much larger quantity of the disease to their own port of departure; that at all events, during 1863, fifty cases (all probably severe) were admitted at the Liverpool hospitals, and during 1862 a dozen cases at the Glasgow and Greenock hospitals; that shipowners of Liverpool and other northern ports, and of Hamburg and America, are those who exhibit the greatest amount of disregard of the safety and health of their crews. The plight in which the poor sailors from certain services are admitted is pitiable to witness. Disabled by hardship, semi-starvation, and ill-usage of every kind, they are cast out with the same indifference with which a worn-out block would be thrown overboard." When it is remembered that the security of this country has on several occasions been imperilled by the disablement of the Royal Navy through scurvy, it may be presumed that the same cause will imperil the safety of our merchant ships. And there can be no doubt that many ships have actually foundered at sea because the crews were so prostrate from scurvy as to be unable to handle them when overtaken by severe weather. It has been the custom to inquire what proportion of the crews were disabled from scurvy. As might be supposed in the case of a disease resulting from a cause operating upon the entire crew, this proportion is often very large. Thus there were recently admitted on board the "Dreadnought" twelve cases of severe scurvy from one ship; two others were known to be seriously affected; and the entire complement of officers and men was only nineteen, leaving but five men in all able for duty to work the ship. The proportion of crew disabled has ranged from 20 per cent. to 70 per cent., and it is certain that scurvy ships have rarely a hand to spare. Deprive such ships of a fourth, a half, or two-thirds of their force, and the peril of a ship, cargo, crew, and passengers, in stress of weather, is obvious; and it must not be forgotten that where scurvy has prostrated a large part of the crew, the vigour of the remainder is sure to be sapped, so that there may not remain a single sailor before the mast in a state of thorough efficiency (SIMON, in *Sixth Report on Public Health*, p. 19).

(b.) *Causation of Scurvy and Conditions under which it is developed.*—In the Middle Ages scurvy prevailed to so great an extent that it was said to be epidemic among the inhabitants of the low countries of Holland, Friesland, Brabant, Pomerania, Lower Saxony, and indeed all countries from the 50° to the 60° of north latitude. This has been attributed to the absolute want of winter food for the cattle, so that it was necessary to kill them on the setting in of the frost, and either to salt or dry the flesh. Food was deficient and of improper quality; and hence the large stores of salt provisions found in the larder of the elder Spencer in the days of Edward II., even so late in the spring as the third of May. Six hundred bacons, eighty carcasses of beef, and six hundred of sheep, was his abundant supply. In all these countries, however, in

proportion as agriculture has advanced, and a succession of green crops has enabled the farmer to kill his best and fattest meats in winter, and in proportion as fresh vegetables have been introduced at our tables, together with a liberal use of wine and beer, so has this disease disappeared. The history which has been given of the disease has fully laid bare the causes of scurvy; which with typhoid fever, dysentery, and putrid ulcer, up to the period of the change in the victualling of the navy, produced great havoc, became comparatively rare in occurrence and light in impression. Since 1797 the improvements have consisted in giving cocoa instead of gruel for breakfast, issuing salt meats at a much earlier period after being cured, the supply of better articles, and in greater abundance by one-third, the substitution of tea in the afternoon instead of spirits; but chief of all, the use of fresh vegetables as often as possible; and, with every improvement in these respects, there has been, as a general result, a further improvement in health, till these four forms of disease, at no distant date so destructive, are scarcely known except by name. With regard to other causes to which scurvy has been attributed, it is now completely established that salted meats are not more productive of scurvy than fresh meats, so far as concerns a monotony of diet; and that salt has no influence whatever in producing scurvy. The experience of the Russians in 1720 and 1736, of the French in 1750-60, and of our own regiments at the Cape in 1836, sufficiently establishes this point. Nevertheless salt meat has generally formed a large and important part of the food taken by the sufferers in all great outbreaks of scurvy. Besides the injurious effects of cold and moisture, as well as impure air, combined with the conditions already noticed as tending to favour the development of scurvy, it is now well known that exposure for a lengthened period to the pernicious influences of a malarious district greatly aids in developing scorbutus. By observations especially devoted to this subject, I have determined that, amongst our troops who had been in Bulgaria during the war with Russia in 1854, there were two and a half per cent. of admissions for scurvy amongst them more than among those who served in the Crimea only; and that the deaths among them reported from this disease were also greater by three per cent. than among those troops who served in the Crimea, (*Glasgow Med. Journal*, July, 1857; and *Trans. of the Royal Med.-Chir. Society*, Vol. XL.) Recent combined researches have shown that scorbutic diseases were developed amongst our troops in the Crimea under the following conditions:—(1.) Deficiency of absolute nutriment; (2.) improper adjudication of the *nutrient* and *respiratory* principles of the *diet*—its monotony; (3.) bad quality of the diet, and improper cooking, or none at all; (4.) exposure to cold, combined with imperfect clothing, and labour beyond the strength of the best fed men; (5.) the persistent pernicious influence of residence in a paludal district (Bulgaria). But these circumstances are only to be regarded as the *occasional antecedents* and *predisponent causes*, any one of which, or all combined, can never, *per se*, originate the disease. LOOKING TO THE HISTORY OF SCURVY, IT WILL BE SEEN THAT THE INVARIABLE AND INDISPENSABLE ANTECEDENT OF THAT DISEASE HAS BEEN A DEFICIENCY OR ABSOLUTE WANT OF FRESH VEGETABLE FOOD. Privation of vegetable food is its one essential cause. There is no other invariable antecedent; and there

are sufficient reasons why it may not always be followed by scurvy. It is this antecedent which is the *vera causa* of *scorbutus*; and the most successful methods of prevention and of cure are in accordance with the hypothesis which assumes it to be the cause (BUDD, CURRAN, LAYCOCK, PARKES). "The giving of vegetable food is its one essential counteractive" (SIMON). The following historical records on these points are therefore of interest:—

"In 1846, the potato crop failed in Great Britain and Ireland; and in the following year there was much scurvy amongst all classes. There was an outbreak of the disorder in Scotland, especially amongst the artisans and the labourers on the railways, in 1846–47, described by Drs. Christison, Ritchie, and Lonsdale (*Edin. Mon. Journ.*, 1847), and the general fact with regard to the food of all was, that it failed in variety and in the quantity of its animal constituents. *None had tasted potatoes after the harvest of 1846—a period of seven or more months—nor fresh vegetables*; but animal food, fresh and salted, was taken in large quantities, as well as pea-soup, suet puddings, bread, and oatmeal. At Workington, a seaport town of 7,000 inhabitants, there was no case of scurvy, turnips having been used in large quantities. Dr. Curran (*Dublin Quar. Journ.*, 1847), describing the disorder as it appeared in Ireland, says:—"In no single instance could I discover that green vegetables or potatoes had formed any part of the regular dietary;" grains, tea or coffee, flesh and fish, being the food. Dr. Shapter observes that in Exeter 'the only difference in the usual diet of the sufferers consisted in the absence of the potato,' and that many of them had abundance of the necessities of life, except fresh vegetables, (*Med. Gazette*, Vol. IV.) In the Crimean War, the allied armies suffered severely from scurvy. In the British army it first appeared in Bulgaria, where the diet was poor and the supply of vegetables scanty. When the army arrived in the Crimea there was an abundance of grapes, cabbages, &c.; and though the ration was inferior, the disorder disappeared; but as winter set in, and vegetable food could no longer be got, it began again. When the supply of fresh vegetables and lime-juice became again more constant, it again gradually disappeared, and there were but few cases during the second winter. The French army suffered still more, no less than 23,000 cases of scurvy being recorded (SCRIVE). Good but lean fresh meat was issued, at first twice, then five times a week, with rice, and occasionally dried vegetables—chiefly pease, beans, and lentils—in small proportions, and bread irregularly. In the spring, with vegetation the number of cases decreased; but in July, with a parching sun, the disease reappeared, and in the course of three months, the finest and warmest in the year in that region, no less than 5,000 cases of scurvy occurred (BUZZARD). The Sardinian army, on its arrival in the early part of the summer of 1855, was largely affected with scurvy, which was checked by the issue of fresh vegetables. Dr. Buzzard, who was a staff-surgeon in the army of Omar Pasha, says that 'the loss amongst the Turks from this disorder was still greater, and that the original force which formed part of the expedition from Bulgaria to the Crimea was almost entirely swept off by disease, of which scurvy formed an important element' (REYNOLD'S *System of Medicine*, Art. 'Scurvy,' Vol. I., 1866). During the winter of 1854–55, of Omar Pasha's troops as many as 1,000 were sent away monthly, all suffering severely from this disorder. During the summer of 1855, this army was encamped near Balaklava; and Dr. Buzzard remarks that, though 'their food was

very imperfect, they were supplied with onions, and consumed large quantities of water-melons, and no cases of scurvy appeared, nor could he detect any scorbutic taint in patients suffering from other diseases. But during the winter all vegetable supplies ceased, and 'shiploads of sick were brought to Trebizond, all of whom were severely afflicted with this disease'—in some cases 'developed to an extent which recalled the terrible descriptions of the disorder contained in the narratives of our early voyagers.' This was explained by the fact that, besides the absence during the winter of fresh vegetables, they had not had a sufficient quantity of food, their diet consisting 'entirely of biscuit, a little rice, haricot beans, and "yagh" (a coarse butter made from mutton fat), and there was absolute starvation.' In 1836, over 100 cases of scurvy occurred in the 75th Regiment while quartered at Caffreland, when not an ounce of salt provisions was issued, when the men had no harassing duties, and were abundantly supplied with fresh meat. The Hottentot troops doing duty with them were served with the same rations, but sought out for themselves pumpkins, melons, wild fruits, and esculents, and so they entirely escaped; as did also the 27th and 72nd Regiments, encamped eighteen miles distant, fed with the same rations, but supplied with vegetables in addition. Dr. Buzzard states that scurvy is common in North Wales, where fresh meat and milk are abundant, but where there is little or no garden produce; and that he has met with many cases amongst the poor of London, who have eaten no meat of any kind for weeks, but have lived on tea, bread, and butter. At the close of the Punjaub campaign (1848-49), the troops had abundance of excellent fresh meat and bread, but no fresh vegetables, yet suffered severely from scurvy; and in the Himalayan stations, during the second Burmese War, under the same circumstances, scurvy was prevalent amongst the troops (*Med. and Surg. Hist. of the British Army*, Vol. II., 1858). Scurvy prevailed to a great extent in the United States army during the civil war. This was not owing to any deficiency in the ration, or to the want of liberal provisions for fresh vegetables by the government and private organisations, or the neglect of the medical department in issuing antiscorbutics. All these were most generously provided. But there were times when they could not be obtained or used; when the men were obliged to live on marching rations; or when, through the ignorance or negligence of commanding officers, they were not distributed. Scurvy, under these conditions, was the invariable sequence. At one time, in the Army of the Potomac, when 'symptoms of scurvy began to appear,' and there was a general 'low vitality of the men,' Dr. Letterman found that he had rightly attributed it to 'want of fresh vegetables,' for 'while large supplies of potatoes had been issued, the troops received in some cases a very small quantity, and in others none at all' (l. c., p. 106). Again, he says (pp. 109, 110),—'This favourable state of the health of the army, and the decrease in the severity of the cases of disease, are in a great measure to be attributed to the improvement in the diet of the men by the issue of fresh bread and fresh vegetables, which has caused the disappearance of the symptoms of scurvy that in January began to assume a serious aspect.' The testimony of Dr. F. Hamilton, with respect to the Army of the West, is to the same effect" (Dr. CLYMER, l. c.)

Its predisposing causes are thus clearly shown to be (whether on shore or in ships) such agencies as debilitate the strength,—e.g., rations insufficient in quantity or in kind; impurity and scarcity of water; exposure to

extremes of heat or of cold in long continued dampness and moisture of air; long exposure to malaria; foul air; inadequate clothing; frequent wetting without change of garments; overwork and depressing mental conditions; and convalescence from other serious diseases, leaving the body weak. "The force and persistent continuance of scurvy are regulated and modified by the simultaneous operation of the predisposing with the essential causes of the disease. On these grounds alone can any reason be assigned why in ships there has always been found to be a very great disproportion in the remarkable fewness of attacks of officers compared with those among the common seamen. Officers are never subjected, under any ordinary conditions of service, to the same amount or combination of the predisposing causes, nor to precisely the same diet, although there may be no difference in their abstention from succulent vegetables, which it has been quite impossible to obtain. In fact, it was proved thus early, that the influence of the predisposing causes is of great weight in the origin and progress of every outbreak of scurvy; and that, without a due regard being had to them, the prevention and cure of scurvy has never been attended with satisfactory results following the measures adopted" (Sir WM. SMART, *Brit. Med. Journ.*, 1877, p. 617).

As to the exact agent in vegetable food which is so antiscorbutic various opinions have been held. Trotter always held the opinion that the real antiscorbutic principles in fruit and vegetables were the vegetable acids, particularly citric, oxalic, and malic acids; and to test the accuracy of his opinion, he made, in 1800, a series of comparative trials between *lemon juice* and pure *citric acid*. Both these remedies were furnished to eight or ten ships, and reports were then obtained of their effects. The result was that *citric acid* was found the most efficacious. Sir William Burnett, also, was in the habit of supplying convict ships with *citric acid* and *nitrate of potash* as well as with *lemon juice*, in order that comparative trials might be made of the relative value of these remedies; and the official documents bearing on this point were carefully examined by the late Dr. Parkes, who considered that nothing could be more convincing than the evidence they contained, showing that the efficacy of *citric acid* was clearly proved, while *nitrate of potash* was shown to be inferior in curative power, (*On the Pathology and Treatment of Scurvy*, l. c.) Dr. Clymer mentions that the Ottawa lumberers living on pork salted with nitre suffer severely. Dr. J. O. Grant found in one *shanty*, out of thirty-six of those men, that twenty-five were affected with scurvy. The astonishing effects of *fresh lemon juice* have been evidently underrated by Dr. Garrod; and it is more probable that its virtues are due to citric and other acids than to potash. Dr. Aldridge contends for the influence which should be ascribed to a deficiency of phosphorus, sulphur, lime, and the alkalies, in occasioning scurvy. That something may be owing—a part merely—to the causes contended for by Dr. Aldridge is not improbable. But it is unnecessary to pursue this subject any further than very briefly to state, that one of the most evident changes from the healthy condition is seated in the blood, which is altered in composition either by the addition to it of some ingredient or ingredients, or by the absence of something which ought to exist in it; and the deficient ingredient may be one of the ordinary constituents of the blood, or it may be some principle or element entering into their com-

position. This deficiency is due to the absence of certain articles of diet ; and the disease is known by experience to be at once cured by supplying those articles.

(c.) *Morbid Anatomy*.—The days when scurvy was most prevalent were not those in which many *post-mortem* examinations were made ; and our earliest knowledge of the morbid anatomy of this disease is derived principally from Poupart and Lind. They tell us that in those cases in which flux or dysentery is absent the intestines have been found perfectly sound, however copious the hæmorrhage from them may have been. The principal effects of the disease were observed in all cases in the cellular tissue of the extremities. The quantity of congealed blood effused in that part, even where no stain or mark could be perceived on the skin, was quite astonishing. “It often lies,” says Lind, “in large concrete masses on the periosteum, while the bellies of the muscles of the legs and thighs seemed quite stuffed with it, often an inch in thickness.” Haspel and Marmy confirm these accounts of Lind when they describe laminar masses of black grumous effusion in the subcutaneous and intermuscular connective tissue, compressing and breaking down the primitive fasciculi. He often found water effused into the cavities of the chest and abdomen, and no less frequently blood,—the quantity of blood effused in all parts sometimes amounting, in his opinion, to no less than a fourth part of that contained in the whole body. Poupart gives some further particulars, and says that on moving the limbs of some scorbutic patients a noise is heard ; and that on examining the joints the epiphyses had entirely separated from the bones ; and in other cases, that the cartilages of the sternum had separated from their bones ; and bones that had united after being broken very often separated again at the site of fracture. He says that if we squeezed the ribs which had begun to be thus separated from their cartilages, “there came out abundance of corrupted matter, so that nothing was left of the rib but its bony plates.” The mesenteric glands were usually enlarged ; the spleen, often three times bigger than natural, fell to pieces, as if composed of coagulated blood. In two cases examined at St. Thomas’s Hospital by Dr. Robert Williams, patches of ecchymoses were found under the pericardium covering the heart, and also under the arachnoid membranes covering the brain. In some epidemics of scurvy there is a marked tendency to the effusion of dark liquid blood into the serous cavities, and of the synovial membranes, the most common site being into the pericardium, then the pleura, and next the peritoneum (KARAWAGEN). The spleen is often enlarged, distended with blood, and soft. The texture of the muscular system is also soft and flabby. The blood appears to be deficient in red particles (BUSK, BECQUEREL, RODIER,² ANDRAL, FRICKS), and does not impart a stain to the lining membrane of the heart and great vessels. A fluid or dissolved condition of the blood has also been noticed, appearing often as “a mere gore, not separating into crassamentum and serum, and putrefying soon. It appears to be starved of some essential ingredient.” It flows with difficulty from the vein, and, after standing some hours, deposits a thick, muddy sediment, which subsides from a reddish serum ; and in the last stage of the disease it becomes quite black (ROUPPE). There does not, however, appear to be any evidence of deficient powers of coagulation in

scorbutic blood; and so long ago as 1699, Poupart noted the large coagula found after death in the cavities of the heart. Three analyses of the blood were also made by Mr. Busk, before 1840, in all of which the quantity of fibrine was found to be above its normal standard. Stoeber, in 1845, came to the same conclusion; and so did Andral, on repeating analyses which at first led him to an opposite conclusion. Two facts of great importance, if confirmed, have been observed by Chatin and Bouvier. They have observed that the *albumen* of the blood does not coagulate under a temperature of 74° Cent., or 165·1° Fahr.—that is, from 5° to 8° Fahr. above the normal standard; so that the albumen of the blood undergoes some change in scurvy which increases its solubility. They also observed that the force of cohesion of the *fibrine* was so much lessened that they were unable to isolate it thoroughly from the red corpuscles; and it was this attraction of the fibrine and the red corpuscles which the early writers on scurvy attempted to describe by the terms "*agglutinated blood*," "*viscid and thickened crassamentum*" (PARKES, "On the Pathology and Treatment of Scurvy," *Brit. and For. Med.-Chir. Review*, Oct., 1848). Thus all the phenomena of scurvy, and the conditions under which it becomes developed, point to the blood as essentially altered. What the alteration definitely consists in has not been yet determined; but it appears that the condition of the blood in scorbutus does not consist in the want of a due proportion of either of its three prominent constituents. The proportion may, in some cases, be deranged; but it appears that in all cases there is a deficiency or altered quality of some essential ingredient.

Symptoms.—The earliest is a change of colour of the skin, particularly of the face and eyelids. This is sometimes the first and only sign of the disease. The skin round the orbit may be puffed up into a bruised-coloured swelling. At the same time there are vague, wandering, rheumatic-like pains in the limbs, weariness, depression of spirits, and a longing for fresh vegetables and fruits. With these pains, however, there is no fever; the pulse is soft and natural, and the temperature of the body lower than usual, and great disorder of the respiratory function, amounting to dyspnoea, may supervene. The countenance appears pale or yellow, and bloated; there is great depression of the physical powers, followed by swelling of the gums, which become soft, spongy, and hang over the teeth in large flesh-like palmated masses, very much disposed and readily excited to bleed. The edges of the gums are purple where they are in contact with the teeth. Several old observers state (and Fauvel confirms the observation) that in old people without teeth these vegetations do not occur, and the gums remain comparatively unaffected. In one case he noticed that a single remaining tooth was surrounded by a mass of swollen gum: the tooth was extracted, and the gum immediately became level and firmer, while the other symptoms preserved their intensity. The swollen and fungous gums have always appeared to be much more intense in cases occurring on shipboard; and it is not improbable that they are affected peculiarly because they are exposed to pressure and attrition; sometimes they are severely ulcerated (PARKES). All parts suffer from pressure in cases of scurvy—the merest rub causes an ecchymosis, the slightest possible blow produces an extensive bruise, and the gums may suffer most from the causes already noticed.

A small eruption (like flea-bites) of a purple hue is next seen on the lower extremities; and about the same time the muscles of the leg or thigh become hard and painful, and in a day or two the skin over the pained part becomes first yellow and then purple. This discoloration forms patches sometimes as big as the palm of the hand, and may extend over half the leg and thigh. The popliteal regions are a frequent site of this pain and discoloration, sometimes attended with œdema, especially of the ankles and feet. The discoloration is especially apt to show itself in the situation of old cicatrices and injuries. "These extravasations are both *superficial and deep-seated*. The *superficial* are,—(1.) *Dermic*, and (2.) *Subdermic*. The *dermic ecchymomata* vary in size from small, dark-coloured spots to large blotches; they may be distinct, or may come together at some point of their circumference. The anatomical site of the spots is the hair follicles or sudoriferous canals, and their structure hinders the diffusion of the sanguine serosity. The epidermis becomes dry, rough, and raised in scales, resembling the skin of a recently-plucked fowl (H. LARREY), and the sudatory function is diminished or suspended. Subsequently desquamation takes place. The solid part of the blood may, however, remain infiltrated in the meshes of the derm, forming brown spots, which may last for months, the epidermis over these being smooth and shining. In the *subdermic ecchymomata* the blotches are larger and of variable depth. They may invade the whole limb, but the most common site is the ham, the groin above and below Poupart's ligament, the thigh, the calf and the pterygo-maxillary region. If they compress superficial veins, they cause œdema and pain. They produce swellings in the flexures of the joints, most often in the ham or shin, in the pterygo-maxillary region, and bend of the elbow. They are hard, but pit on persistent and firm pressure. When on the shin and circumscribed they may be mistaken for syphilitic nodes. Resolution is their usual termination, their hue changing from dark purple to green and yellow. When the dark colour persists with a greenish-yellow border, it is evidence that the effusion is very thick. Should their termination be by ulceration, a sort of indolent boil forms, with a deep-red base and black summit, with œdema of the adjacent connective tissue. If the surface is galled, there is constant weeping of a sero-sanguinolent fluid; the tumor flattens a little; and a small, dark, ulcerated central point, with wine-red edges, appears: this is a gangrenous mass, which gains little by little, until an ulcer of variable size is established. The most frequent site of these scorbutic ulcers are the calves of the legs, the buttocks, thighs, sacrum, shoulders, arms, and more rarely the chest and abdomen. The *deep-seated extravasations* are,—(1.) *Sub-aponeurotic*; (2.) *Parenchymal*, into the muscles, kidneys, heart, lungs, &c. These extravasations into the muscle-tissue never end in suppuration, but the tissue becomes atrophied, and undergoes a sort of gelatinous degeneration; resolution is very sluggish. When the effusion is beneath a resisting aponeurosis, as the *fascia lata*, there is no alteration in the integument immediately over it, but the swelling and discoloration happen at remote points, as the ham, about the knee, &c. The source of these extravasations would seem to be the softened capillary vessels, permitting the leakage of altered blood. There is no apparent solution

of continuity except an ulcer exists" (MARMY, quoted by Dr. CLYMER). The tongue is white, the breath fetid, and the stools generally pale. As the disease advances, all these symptoms are aggravated. The loss of physical power increases, the purple spots have a tendency to ulcerate, and the ulcers are distinguished from all others by their putrid fungoid appearance and great tendency to bleed; old sores open, and the callus of broken bones has been even dissolved and their ends separated. Profuse hæmorrhages frequently take place from the mouth, nose, lungs, or bowels, sometimes from *bullæ*, which form and burst (discharging blood) on the mucous surface. The teeth become loose, so that they either fall out or may be taken out by the finger and thumb. *Ptyalism* is not uncommon in scurvy, with swelling of the parotid and submaxillary glands. The pulse hurries on to 120 or 140; and at length the patient sinks from diarrhœa or dropsy, and with effusion so sudden that he perhaps may have walked a short distance, and then died in a quarter of an hour afterwards.

The duration of the disease is generally many weeks, and sometimes, under the most favourable circumstances, many months, the patient recovering his strength very slowly. When the disease follows ague, obscure or irregular remissions or intermissions of febrile symptoms are observable, and more or less enlargement, with pains in the region of the spleen, is often detected. From the commencement of the disease the alvine evacuations are more or less disordered, but the change may not attract attention. At first costiveness prevails, but subsequently the stools become much more frequent and very offensive. Diarrhœa, with colicky pains, supervenes, and, with more or less attendant hæmorrhage, rapidly sinks the patient beyond hope of remedy. Dysentery frequently supervenes upon the scorbutic state, more especially when dysentery is epidemic; and this combination of maladies has sometimes been termed "*scorbutic dysentery*." The tongue, pale at first, becomes broad, flabby, and indented at the edges of the teeth. The great poverty of the blood causes the mucous membrane of the tongue and inside of the lips and cheek, as well as of the skin, to have a peculiar paleness, which contrasts remarkably with the appearance of the gums. The eyesight is frequently weakened, and night-blindness (*hæmeralopia* or *nyctalopia*) has frequently been observed as a commencing and concomitant symptom; and a most distressing state is the inability to sleep at night.

"The tendency to *swoon* in the most severe cases is sometimes so great that the slightest motion, or the erect posture, or even any trifling exertion, may be followed by fatal syncope. It is stated in the account of Lord Anson's voyage, that many of the men, although confined to their hammocks, ate and drank heartily, were cheerful, and talked with much seeming vigour, and in a strong tone of voice; and yet, on their being the least moved, although it was only from one part of the ship to the other, and that in their hammocks, they have immediately expired; and others, who have confided in their seeming strength, and have resolved to get out of their hammocks, have died before they could reach the deck. And it was no uncommon thing for those who could do some kind of duty, and walk the deck, to drop down dead in an instant, on any endeavour to act with their utmost vigour" (COPLAND).

There is an intercurrent chest affection which is apt to occur during cold and damp weather in scorbutic patients.

"It begins with slight rigors, followed by feverishness, and accompanied by lancinating pains in one or both sides. There is dyspnoea, and a feeling of constriction in the chest, as if a cord were drawn tightly round it. This condition is commonly caused by intrapleural effusion of blood, but sometimes blood escapes into the pulmonary tissue; in the latter case, the expectoration becomes dark and sanious, and has a fœtor resembling that of pulmonary gangrene, but which is due to decomposition of the sanguine fluid. Cold sweats, increasing dyspnoea, anxiety, and a frequent, thready pulse, precede death—the constant termination. Sometimes there is neither pain nor cough, but rapid increase of the breathlessness, and sudden death. The physical signs of the lung lesion are often wanting, though sometimes there may be dulness, mucous rhonchi, and bronchial respiration (HASPEL). Chest dulness on percussion in scurvy may sometimes be due to sanguineous effusions into the thoracic muscles" (HASPEL, BUZZARD, CLYMER).

With our knowledge now of the phenomena of embolism, may not the suddenly fatal end of such cases be due to the morbid condition of the fibrine of the blood already referred to?

Diagnosis.—The scorbutic state of the skin is to be distinguished from *flea-bites*, *bruise*, *typhus fever*, and from *purpura hæmorrhagica*. In *purpura* the blotches occur suddenly in persons previously, to all appearance, in good health; while the dingy hue of the skin is wanting, also articular effusions, and pains in the limbs.

Prognosis.—In the present day, when the patient can command medical care and proper diet, scorbutus, though tedious, is seldom fatal. When these, however, have been wanting, the mortality has been terrible. Lord Anson, in his voyage round the world, lost above 200 men, and at last could not muster more than six fore-mast men in a watch fit for duty. At the commencement of our last war with France, on the fleet returning from sea, it often happened so many men were landed ill of scurvy that even Haslar Hospital, large as it is, could not contain them; and many were lodged in the chapel, others in tents, while others died in the boats before reaching the shore.

Treatment.—The disease, "so fatal when left to itself, is cured with the greatest facility. Symptoms apparently the most grave and serious vanish as if by magic, and without leaving behind them any serious injury to the constitution. The sanious discharge from scorbutic sores has been known to change colour, and to become healthy in a few hours after the commencement of treatment. In pure cases of scurvy the blood, and the blood only, is at fault," (PARKES, l. c.) "Lemon juice," writes Dr. Watson, "is really a specific against scurvy, whether it be employed as a preventive or as a remedy. It supplies something to the blood which is essential to its healthy properties." The potato seems to be no less efficacious as a remedy and preventive (BUDD), and all the antiscorbutic remedies are most efficient in the form of juice—from raw and uncooked fruit. The reader will find a most interesting account of the efficacy of potatoes and of onions in Dana's *Two Years before the Mast*—a book well

worth reading. The antiscorbutic principle, whatever it may be, is in greatest amount in unripe fruits; it lessens gradually as they ripen; and if the juice be obtained, the principle disappears, or is decomposed and rendered inefficient, when fermentation occurs. When lime-juice becomes musty, a mucilaginous principle is developed at the expense of the citric and malic acids; and the percentage of citric acid gradually decreases (SIR WILLIAM BURNETT, PARKES). Good *lemon* juice seems to be more effectual, however, than pure *citric* acid; probably from its containing *malic* and *tartaric acids* besides *citric*, and from the *citric acid* being in the form most easily absorbed and decomposed by the digestive organs of man. The *Materia Medica* gives numerous analogous examples of the superior efficacy of a medicine in its *natural combinations* (PARKES). This is all we can yet say, however, regarding the actions of either of these means of cure, notwithstanding the researches of the chemists of the present day. Moreover, it is sufficient: and with such remedies at command, the prevalence of scurvy in *merchant vessels*, or in any navy, ought not to exist. "The one thing wanted in order that scurvy should be entirely banished from the *mercantile marine* is proper provision for the dietary of the crew,—such provision as is enforced in *emigrant ships*, where each person's weekly allowance must have in it at least 8 oz. of preserved potatoes, and 3 oz. of other preserved vegetables (carrots, onions, turnips, celery, and mint), besides pickles and 3 oz. of lime-juice; where, also, there is considerable variety of bread stuff; and where, on two days in the week, preserved (not salted) meats must be given. With such a dietary as this, the details of which might be varied, provided its principle were adhered to, the occurrence of scurvy would be impossible. And even with dietaries inferior to that just described, scurvy would not occur as it does, if but the provisions of the 'Merchant Shipping Act' (17 and 18 Vic., c. 104) were obeyed,—that whenever a crew shall have been consuming salt provisions for ten days, *lime juice* or *lemon juice* and *sugar* shall be served out at the rate of *half an ounce* each per day; and if, during the voyage, the opportunities which offer themselves were fairly used for getting new supplies of fresh animal and vegetable food; but owners disobey the law, and captains neglect opportunities to counteract the results of this disobedience. Owners, notwithstanding the law, will send forth their ships on long voyages without any provision of lime-juice, or with lime-juice insufficient in quantity, or with lime-juice of which the quality is bad; and captains, with half their crews more or less disabled, are known to run past St. Helena or the Western Isles, when a few hours' delay would obtain sufficient provisions to repair the mischief occasioned by first neglect" (SIMON and BARNES'S *Sixth Report on Public Health*, 1864, p. 20). It is perhaps hardly fair to attribute the improved health of the navy entirely to the introduction of a daily allowance of lemon juice, considering that the quantity of the diet was greatly increased, and its quality greatly improved, contemporaneously with this addition. It is gratifying, however, to see how largely these combined measures have improved the health of the navy, and rewarded the cares of those who superintend it. During the nine years preceding these changes the sick seamen sent to the hospitals were 1 in 3·9, while in the nine succeeding years the proportion was only 1 in 8·4; so that not only

has scurvy almost disappeared from ships of war and naval hospitals, but the efficiency of the navy has actually been increased threefold.

Dr. Parkes advises that the following measures be adopted in time of war, or in prolonged sojourn on board ship, or at stations where fresh vegetables are scarce:—“(1.) The supply of fresh vegetables by all means in our power. Even unripe fruits are better than none, and we must risk a little diarrhœa for the sake of their antiscorbutic properties. In time of war *every* vegetable should be used which it is safe to use, and when made into soups all are tolerably pleasant to eat. (2.) The supply of dried vegetables, especially potatoes, cabbages, and cauliflowers; turnips parsnips, &c., are perhaps less useful; dried pease and beans are useless. As a matter of precaution these dried vegetables should be issued early in the campaign, but should never supersede the fresh vegetables. (3.) Good *lemon juice* should be issued daily (1 oz.), and it should be seen that the men take it. (4.) *Vinegar* ($\frac{1}{2}$ oz. to 1 oz. daily) should be issued with the rations, and used in the cooking. (5.) *Citrates, tartrates, lactates, and malates of potash*, should be issued in bulk, and used as drinks, or added to the food. The easiest mode of issuing these salts would be to have packets containing enough for one mess of twelve men, and to instruct the men how important it is to place them in the soups or stews. Possibly they might be mixed with the salt, and issued merely as salt” (*Prac. Hygiene*, 2nd Ed., 1866, p. 466).

Our recent Arctic expedition, in 1876, is of interest for two reasons:—(1.) As regards a peculiar theory as to the action of lime-juice which the commander of that expedition entertained and acted upon, based as it was upon a groundless and absurd hypothesis, that by doubling the dose of lime-juice for a month he believed he could, so to speak, “saturate” his men with it before they started on an important sledging expedition, so that they might be able to do without it on these journeys. Lime-juice is simply an article of diet; and is no more “cumulative” in its action than any other aliment which goes to nourish the body. Its nutritive influence continues for so long and no longer than the time when it is continued and assimilated as an article of diet. The assumption of the theory was entirely gratuitous and unfounded; and to have acted upon it against competent medical opinion was unjustifiable. (2.) Another theory was entertained to palliate the omission of lime-juice from the sledging rations, namely:—that lime-juice when exposed to an extreme degree of cold deteriorates and ceases to have any antiscorbutic properties.

“This theory was not only disproved by the evidence of the medical officers on other sledging expeditions, but was effectually answered by the fact that the whole expedition was virtually saved from the most fearful mortality from scurvy by the opportune discovery of a depôt of lime-juice left five years before by the “Polaris” expedition, lying in a cask on the ice exposed to the extremity of cold alternating with heat. It had also been much weather-beaten, and on its surface mixed with snow; but it nevertheless proved to be in no respect deteriorated in its antiscorbutic powers, and served to keep off the last extremities of disease from the sledging parties to whose relief it was sent in time. It appears, indeed, to us, and it will appear, we think, to any one who reads the whole narrative, that, but for the timely discovery of this depôt of the

“Polaris” lime-juice, and its utilisation for the sledging parties, there is good reason to fear that so large a proportion of the crews would have been destroyed by scurvy, that it is doubtful whether the whole expedition would not have been lost from the want of sufficient power to bring the ships out of the ice. However this may be, the frozen lime-juice rendered the most essential services, and the theory that freezing destroys its virtues is most effectually disposed of” (*Brit. Med. Journal*, March 10, 1877).

In fact there is nothing in the history of our latest Arctic expedition, of 1876, which does not point to the cause of the outbreak of scurvy as similar to all those which history has already recorded; and which will again produce the disease if the teachings of history are similarly neglected and set aside.

The great body of evidence taken by the Committee appointed to inquire into the causes of the outbreak of scurvy in the Arctic expedition fully shows that “the outbreak was due essentially to the omission—or, rather, to the refusal—of Captain Nares to send lime-juice with the sledge parties. It was shown that the provisions of the ships were ample in quantity and excellent in quality; and, further, the Committee having had before them the preliminary memorandum by Sir Alexander Armstrong, the Director-General of the Medical Department of the Navy, strongly and explicitly requiring the issue of a daily ration of lime-juice, and having heard Sir George Nares’ explanation of the reasons which induced him to act in defiance of that memorandum, decided that in doing so he made himself responsible for the outbreak of scurvy which occurred, and that he had no sufficient reason for not carrying out the rules laid down in the memorandum.”

“It is a satisfaction to find that throughout the whole of this inquiry the Medical Department of the Navy comes out with the utmost possible credit. Nothing could be more clear, more explicit, more absolutely prophetic than the memorandum which was furnished to Sir George Nares by the Admiralty from Sir Alexander Armstrong, on the immeasurable importance of supplying the men with a daily ration of lime-juice. The whole of his directions for the maintenance of the health of the men are indeed most able, practical, and judicious; and, had they been strictly followed out, the results would have been very different. The conduct of all the medical officers of the expedition appears to have been such as to merit and to receive the warmest approbation of their superiors from first to last, and they showed a devotion to duty which is entirely comparable with that displayed by the officers and men of this gallant expedition generally. They can seek for no higher praise, and it is on all hands conceded that they merit no less. One moral of this inquiry, which is salient on the face of it, and which we can hardly be expected to abstain from pointing, is, that combatant officers will do well always to respect the functions of the medical officers, and to attribute just weight to medical recommendations. In framing false medical theories, and in assuming the responsibility of refusing to act upon his medical instructions, Sir George Nares brought a great disaster upon this expedition; and he assumed the full responsibility of it” (*Brit. Med. Journal*, March 10, 1877).

ANÆMIA.

LATIN EQ., *Anæmia*; FRENCH EQ., *Anémie*; GERMAN EQ., *Anæmie*—Syn., *Blutarmuth*; ITALIAN EQ., *Anemia*.

Definition.—*A disease in which there is either a relative diminution of the mass of blood (ANDRAL), with the general composition and distribution of the blood altered from the normal standard; or in which the mass of blood is diminished, and the liquor sanguinis is watery, poor in albumen, and containing an excess of salts. These conditions, co-existing with relative deficiency of the red blood-corpuscles (VOGEL), and a diminution of the urine-pigment (PARKES), with altered innervation of the vascular system (LEBERT), constitute anæmia.*

Pathology.—(a.) *Causation.*—The term *anæmia* literally means absolute deficiency of blood—a condition of existence obviously not possible. A diminution in the quantity of blood, with an alteration in its composition, almost never occurs alone, but is generally a morbid state resulting from many exhausting morbid processes peculiar to such wasting constitutional diseases as *scrofula* and *cancer*.

As an independent constitutional disease, anæmia may be said to owe its origin to three sets of conditions,—(1.) Copious loss of blood, such as by hæmorrhage or venesection, or oft-repeated small losses of blood. (2.) From loss of other fluids of the body besides blood, such as lymph, and especially of such fluids as contain albuminous, fibrinous, mucinous, or caseinous substances, such as the excessive secretion of milk in protracted suckling, suppuration, profuse blenorrhœa, leucorrhœa, or diarrhœa. (3.) From insufficient and improper nutriment, or from disturbances in the absorption and assimilation of food, and the process of sanguification; or from repeated temporary interruptions to oxygenations of the blood, as by imperfect ventilation of sleeping rooms or of coal mines. (4.) It may result from the co-operation of many influences; for example, excessive bodily and mental labour; continued excitement, pain, care, grief, hardships; many acute and chronic diseases, some of which augment the consumption of blood, as when the elements of the blood are consumed faster than they can be made, as in fever of long duration. Other influences impede the formation of blood—such as acute disease under certain circumstances, when its invasion is intense and its duration prolonged, such as *scrofula*, *carcinomatous diseases*, *diabetes*; poisoning by malaria—the *malaria chlorosis* of Vogel, or *paludal cachexia* of Martin, as in the “used up” condition of our Bulgarian troops, described by the late Dr. H. Mapleton in *Parliamentary Paper* 247, for 1856, p. 253. “It is the most general of all the conditions incident to tropical invalids” (MARTIN). Anæmia is often the result of such poisons as *lead*, *mercury*, *iodine*, *aniline*, *tobacco*, and of diseases such as *syphilis*, *rheumatism*, *goitre*. Tailors, shoemakers, cooks, firemen of steam-vessels, and female operatives in large factories, are the most frequent sufferers from anæmia. Dr. Walshe has also very justly directed attention to the prevalence of anæmia amongst female servants in London, who are obliged to go up numerous and long flights of stairs very often in their daily labours; and he is of opinion that the suspended inspiratory acts have much to do with the

peculiar state of the blood that ensues and causes anæmia ("Clinical Lectures," *Lancet*, 1849). Amongst forms of anæmia to be distinguished are:—(1.) From excessive flow of lymph. Under this head Lebert has described a dilated condition of lymphatic vessels—their varices, and an excessive flow of lymph from them. Such may occur spontaneously and repeatedly, and on each occasion such quantities of lymph may be poured out that great and severe anæmia may be the result. One female patient lost at one time 1750 grammes, or nearly 57 ounces of lymph. The symptoms of anæmia in such cases are analogous to those produced by hæmorrhage—especially hæmorrhages from the uterus. (2.) Anæmia from hæmorrhage or loss of blood. Under this heading Lebert points out that there is a class of cases in which, either from the spontaneous cessation of the hæmorrhages, or from their cure by surgical means, as for example piles and uterine polypi, the patients appear to be perfectly cured after very large losses of blood, and they do indeed regain the external appearances of health, if looked at superficially. But when we study them more closely, we find them remaining for many years still feeble, easily fatigued, irritable, and an easy prey to disease of various kinds. (3.) Anæmia from insufficient nourishment—or starvation; is to be seen chiefly in schools, prisons, work-houses, asylums, and other public institutions, where sufficient attention is not paid to the quantity, quality, and due combination of the appropriate nutritive elements in the food. (See following Chapter.)

(b.) *Morbid Anatomy*.—In many of the diseases just mentioned the blood-mass is evidently diminished. We have indications of this diminution in the small pulse, in the pale bloodless appearance of the countenance and surface of the body generally, especially seen in the lips and gums, and in the small blue collapsed veins, particularly obvious by contrast on the pallid skin. In such cases one would never think of drawing blood to know whether or not its constitution was changed; but in cases where the opportunities for examination have occurred, the blood-corpuscles have almost always been found relatively diminished; and accordingly the College of Physicians of London define anæmia simply as "*deficiency of red corpuscles in the blood*." The causes which lead to this diminution are obscure; and at one time the spleen and glands have been held as concerned in bringing about the disease by some abnormal exercise of their functions. The result of this morbid condition of the blood chiefly betrays itself—(1.) Upon the *vascular system* generally; and, (2.) Upon the *metamorphoses of tissues*. The blood-vessels contract in proportion to the diminution of the blood-mass. The *arteries* contract generally; and the pulse, whenever it can be felt, is found to have become small and tense. The *capillaries* also contract, the skin and mucous membranes becoming pallid and comparatively bloodless. The *heart's* action in extreme cases becomes irregular, and the whole circulation generally is disturbed. As regards the metamorphoses of tissues, the muscles and the nervous system appear to suffer first; debility and prostration, both bodily and mental, occur; and in severe cases sensation may be lost, syncope is frequently apt to occur, and even death may result. The disease seems to go through different stages, according to the causes which bring about the anæmic condition. After mere loss

of blood in quantity, for instance, the water and the salts are renewed with most rapidity, the albumen later, then the colourless corpuscles, and last of all the red corpuscles. Hence it is that anæmia is frequently associated with a watery condition of the blood, as well as with a lessening of the number of the blood-cells. It is a matter of the greatest importance in practice to distinguish, if possible, between these conditions, with a view to a rational means of treatment. Those exhausting diseases which are attended especially with deranged nutrition and sanguification, such as *Bright's disease*, *carcinomatous diseases*, *scrofula*, and *suppuration*, lead also to the development of that form of anæmia in which the *liquor sanguinis* is in excess, in which the blood is poor in albumen, containing an excess of salts, and in which the blood-cells ultimately become deficient. A tendency to *general dropsy* or to *diarrhœa* ensues, nutrition becomes still more disordered, and new formations are apt to become developed. As regards the red corpuscles of the blood in anæmia there are slight forms in which they vary between 110 and 100 per 1000; and a more severe form, in which they are lessened to 65 per 1000 (ANDRAL), or even less. Andral and Gavarret mention even as few as 28 per 1000 in human beings. In average cases it is rare to find the red corpuscles diminished below half the normal number, except perhaps temporarily after very large and sudden losses of blood.

Symptoms.—In combination with an investigation of the blood, the symptoms and signs of *anæmia* are already read in its pathology. It is only by general symptoms, however, that the physician is able circumstantially to conclude that relative diminution of the cells of the blood co-exists with deficiency of the mass. Great debility is a prominent and striking feature. The skin is of a pale waxen colour; the “whites” of the eyes have a bluish aspect; the mucous membrane of the mouth is colourless; the gums and lips are white; and the tongue is pale, large, and flabby; the pulse in general is about 80, but very feeble, and easily excited—the least stimulus, the least mental emotion or movement of the body, produces great momentary acceleration of the pulse. The temperature in the axilla is usually normal; although anæmic patients generally suffer from cold extremities. Respiration is hurried on the least exertion. The appetite is bad, and thirst prevails. When the disease has existed for some time, œdema of the feet and ankles may supervene; and, finally, sweating, in the extreme stage of *anæmia*. It occurs most frequently in the last stages of severe and exhausting sickness of a lingering and prolonged kind, where a high degree of paleness of the skin and mucous membranes exists, with a small weak pulse, collapse of the veins, and a small volume of the heart, spleen, and liver. Mental depression is usually distressing, the temper variable, and intelligence torpid. Diagnostic characters of *anæmia* have been recognised also in various *murmurs* which may be heard in some parts of the vascular system of anæmic patients. Although the seat, the causes, and the signification of these *murmurs* have been very much disputed, yet the following account may be given of them, based on numerous observations and investigations, especially those of Vogel and of Walshe. There are three kinds of anæmic *murmurs* which may be distinguished, namely,—(1.) *Cardiac murmurs*; (2.) *Arterial murmurs*; and, (3.) *Venous murmurs*.

The *cardiac anæmic murmur* gives forth what is usually called a "bellows sound," sometimes intense, sometimes faint, and which generally accompanies the first ventricular sound of the heart, which it commonly more or less masks. It is short, and usually soft. The position where these *systolic* cardiac murmurs is best heard is of some importance, as indicating anæmia rather than disease of the heart. They are heard towards the base of the heart in anæmia, rather than towards the apex, as in organic disease of the valves (WALSHE). It is thus exceedingly local, the maximum of intensity being generally in the fourth intercostal space, near the sternum; more rarely limited between the fourth and fifth; beyond these points it is never heard. It follows the course of a line drawn from the point of greatest intensity to the inner portion of the right clavicle. The *diastolic* murmurs are really of venous origin; and their intensity is more or less increased by suspension of the respiration. The *cardiac anæmic murmur* occurs most frequently in true anæmia, especially if cardiac excitement is associated with it; and simultaneously with it we frequently hear *arterial* and even *venous murmurs*. "That the site of the cardiac anæmic murmur is in the arterial orifices, and especially in the aortic orifice, no one doubts" (POTAIN'S article "Anæmia," *Dict. Encyc. des Sciences Médicales*, t. iv., p. 392, Paris, 1866). This was the received site of the murmur by all, except Hughes, until quite recently. He (remarking that it cannot be heard below the nipple and in the axilla, as is the case in aortic murmurs depending upon organic disease, or generally on a level with the ascending aorta, or at the arch) was disposed to place it in the orifice of the pulmonary artery (*Guy's Hospital Reports*, 1851). Quite lately, Dr. Parrot has given some excellent reasons for believing the site of the anæmic cardiac murmur to be in the right auriculo-ventricular orifice (*Archives Gén. de Méd.*, Août, 1866); its maximum of intensity, he observes, is in the fourth intercostal space, near the sternum, corresponding with the right auriculo-ventricular orifice; and its line of propagation is in the course of the superior cava, and not of the aorta; and he further assigns insufficiency of the tricuspid valve as the cause of the murmur (CLYMER). It has been observed by Vogel and others, however, that while the presence of these *cardiac murmurs* may be employed as an auxiliary sign together with others in the diagnosis of anæmia, yet it is not to be concluded from their non-existence that anæmia is not present. Such *murmurs* are not heard in all anæmic patients; and although present they are not in every case due to anæmia, because they occur in organic diseases of the heart, such as valvular diseases, and endocarditis; and a physician trusting to and acting upon stethoscopic signs *merely*, without a due appreciation of general symptoms, seriously injures the patient, and does an injustice to the science of medicine.

The *arterial anæmic murmurs* are seldom heard. They consist of an intermittent blowing, sometimes soft, sometimes sharp sound, perceived to be synchronous with the beat of the pulse, which gives at the same time a thrill to the finger, so that the *murmur* may be inferred by practice from the nature of the pulse. It is only in the larger arteries in which they are heard, such as the brachial, the subclavian, the femoral, the carotid, and abdominal aorta. They are most frequently heard after great losses of blood; and sometimes also in *chlorosis*. Vogel has heard

them during the paroxysms of intermittent fever, while they are absent during the intermissions. They may be heard in typhus fever. No positive conclusion can therefore be formed from them alone regarding anæmia.

The *venous murmurs* or *hums*, significant of *anæmia*, are *continuous*, humming, buzzing, occasionally musical, singing murmurs, easily distinguishable from the blowing *intermittent* arterial murmurs. They are most frequently heard on the right side of the neck, at the junction of the external and internal jugular vein; but they may also be heard in the femoral veins in their maximum degree of intensity. They may be heard, also, over the course of the superior longitudinal sinus, and at the maximum intensity over the *torcular Herophili* (WALSHE). They are supposed to be produced by abnormal oscillations of the venous valves, or by sharp collision among the blood-discs, passing from sideward veins into a large vessel. When the venous murmurs are strong, they may not only be *heard* but also felt as a gentle thrill, by placing the finger on the part. These venous murmurs are seldom absent in well-marked anæmia. Nevertheless, anæmia is not to be positively inferred from the mere presence of any one of them. The mechanism both of the cardiac and venous anæmic murmurs have generally been ascribed,—(1.) To increased friction of blood, whose viscosity is diminished, against the walls of the vessels; (2.) to increased celerity of the blood, and consequent loss of relation between the contents of the ventricles and the area of the openings leading from them; (3.) to the passage of thin watery blood through tense veins (OGIER WARD). Dr. Flint has suggested spasm of the papillary muscles, causing insufficiency of the mitral valve, as the cause of the cardiac murmur; and Marey believes it due to diminished arterial tension, and increased rapidity of the ventricular systole. Whenever a proper examination can be made of the veins of the neck, particularly at the base, the seat of the two pulsations—and sometimes only one, synchronous with the beat of the radial artery—will be found to be limited to the course of the vein, to be from above downwards, to follow a line which makes an acute angle with the artery, and to cease to be heard immediately on the least compression above the point of pressure; all of which goes to prove that the pulsations are due to a reflux of blood into the right cavities of the heart—in fact, a true venous pulse.

One constant urinary character attends both anæmia and chlorosis—namely, a diminution in the urine pigment, which is often reduced to one-fourth or one-sixth of its normal amount. Very generally there is also lessening of the free acidity; and urine which is pale and almost neutral during the whole of the twenty-four hours, is almost as good an indication for the use of iron as the pallor of the skin itself. The amount of iron in the urine of anæmia is often very small. The quantity of urine may not be decreased, and may be in some cases large. Its specific gravity is low, (PARKES, l. c.) With regard to other constituents, such as urea and uric acid, the accounts are very contradictory.

Diagnosis.—Anæmia and consumption by pulmonary phthisis are apt to be confounded together; and Dr. Pollock protests against anæmia being considered a frequent antecedent or manifestation of phthisis. He con-

siders it a condition least frequently associated with phthisis. In none of 125 cases of anæmia was there any sign of tubercle; and although the girl with extreme anæmia is always supposed to be consumptive, the diagnostic points are,—(1.) The build of the anæmic patient is generally antiphthisical, and, in spite of her colour and delicate appearance, there is neither loss of flesh to any great extent nor fever. (2.) Mobility, percussion, and tone of respiratory sounds are normal, even when cough, dyspnoea, and hæmoptysis may have existed for months. The blood-changes characteristic of anæmia are antagonistic to tubercle; and cases of anæmia very rarely proceed to tubercle of the lung. Anæmia wastes those organs and tissues dependent on an abundant supply of red blood; phthisis wastes all tissues by furnishing them with impure blood. The phenomena of anæmia are caused by insufficiency of blood; the phenomena of phthisis are caused by impurity of blood (POLLOCK).

Prognosis.—It is very important to know, in a practical point of view, that every acute disease which occurs in an anæmic individual assumes a peculiar character: a very high degree of debility and prostration ensues, convalescence is protracted, and all severe intercurrent diseases acquire a lingering course.

Treatment.—The energies of the physician must be directed to discover and counteract the cause of the anæmia. Nutritious substances must be supplied for diet, in the shape of easily digested meats and broths. The purely tonic treatment, in the combination of air, exercise, and diet, must be carried out as far as practicable. A change of air is absolutely necessary, and generally also of diet. *Iron* is one of the best medicinal remedies; and it ought to be taken as largely diluted with water as possible. The astringent preparations are pre-eminently tonic; and are especially useful when the anæmia is associated with or dependent upon inordinate discharges. *Solution of the perchloride of iron*, in the form of *tinctura ferri perchloridi*, in doses of ten to thirty minims in *water*, or in an *infusion of quassia* or of *calumba*, has properties in common with the numerous salts of iron, and is one of the most reliable preparations. If the anæmia is associated with *diarrhœa*, or *menorrhagia*, or *leucorrhœa*, the *solution of the perntrate of iron*, in similar doses, is attended with benefit. A preparation which is no longer new to the pharmacopœia—the *syrup of the phosphate of iron*—possesses the general properties of the ferruginous compounds, and is of great service when the *anæmia* is associated with certain forms of *dyspepsia*, or with *amenorrhœa*. It invigorates and increases the powers of digestion, and may be given to the extent of one to three drachms for a dose in *water*. Another phosphatic preparation of very great value is that which was devised by the late Dr. Easton, Professor of Materia Medica in the University of Glasgow. Although it is not in the pharmacopœia, and although its mode of preparation had not been published before Dr. Easton kindly sent it to me for publication in the previous editions of this work, yet the combination has become very popular throughout the country as a valuable tonic in *anæmia* and *cachexia* generally. As such it has been largely used by my colleagues, Professors Maclean and Longmore, amongst the used-up cachectic and anæmic soldiers under treatment at the Royal Victoria Hospital at Netley;

and I would add my testimony to its being a most valuable medicine in general practice.*

In some cases the *astringent* preparations of iron are not suitable, and are apt to irritate delicate stomachs, or those in whom any inflammatory local disease exists. For delicate females and children the *saccharated carbonate of iron* is a most valuable preparation, in the form of *mistura ferri composita*, to the extent of one to two ounces for a dose; or in the form of the *pilulla ferri carbonatis*, in doses of from five to twenty grains in the twenty-four hours. The *citrate of iron and ammonia* is another remedy which possesses scarcely any astringency, and may often be given in cases of anæmia when the stomach will not bear more astringent preparations. Five to ten grains of this salt may be taken during the twenty-four hours. It is best taken during effervescence, prescribed in *solution of citric acid*, and not in *bicarbonate of potash solution*. If it is put into the latter, carbonic acid will be given off, and probably burst the bottle. *Tincture of orange-peel* is the best flavouring agent; but as the salt will not dissolve in the tincture alone, it is necessary to dissolve the salt in water first, and then add the tincture, otherwise the division into doses is impracticable (SQUIRE). When it is desirable to continue the use of iron for a long time, as in the *anæmia* of neuralgic affections or *tic-douloureux*, or to give it in large doses, the *magnetic oxide of iron* is the best preparation to administer in doses of five to twenty grains twice or thrice a day in water. The *reduced iron*—the *ferrum reductum* of the British pharmacopœia—is also a remedy which does not possess the astringent properties of the other preparations, and is one of the most powerful remedial agents in cases of anæmia. *One to five* grains may be given several times a day in powder or in pill. It has no taste, and *one* grain is equal medicinally to *five* grains of the citrate of iron. The *citrate*

* The following is the original formula devised by Dr. Easton for the preparation of the *phosphates of iron, quinine, and strychnia*, in the form of a syrup ("Syrupus ferri, quiniae et strychniae phosphatum") :—R. Ferri Sulph. ʒv.; Sodæ Phosph., ʒvi. (a little more phosphate of soda gives a better result, say ʒi.); Quiniae Sulph., grs. xcii.; Acid Sulph. Dil., q. s.; Aquæ Ammoniae, q. s.; Strychniæ, grs. vi.; Acid. Phosph. Dil., ʒxiv.; Sacchar. Alb., ʒxiv. "Dissolve the sulphate of iron in 1 oz. boiling water, and the phosphate of soda in 2 oz. boiling water. Mix the solutions, and wash the precipitated phosphate of iron till the washings are tasteless. With sufficient diluted sulphuric acid dissolve the sulphate of quinia in 2 oz. water. Precipitate the quinia with ammonia water, and carefully wash it. Dissolve the phosphate of iron and the quinia thus obtained, as also the strychnia, in the diluted phosphoric acid; then add the sugar, and dissolve the whole, and mix without heat. The above syrup contains about one grain phosphate of iron, one grain phosphate of quinia, and one thirty-second of a grain of phosphate of strychnia in each drachm. The dose might therefore be a teaspoonful three times a day. The amount of phosphate of quinia might be increased according to circumstances; and if eight grains of strychnia were employed in place of six, as in the above, the phosphate of strychnia would be in the proportion of the one twenty-fourth of a grain in every fluid drachm of the syrup. I would scarcely venture on a much larger dose. In cases of delicate children, with pale countenances and deficient appetites, I have given, with great benefit, a combination of equal parts of the above syrup and of that prepared by Mr. Edward Parrish, often called chemical food. To children between two and five years of age, the dose of this combination may be a teaspoonful three times daily. The preparation ought to be kept as much as possible from light and air, otherwise the preparation loses its characteristic *opaline* appearance, and assumes a fawn colour; a precipitate may be thrown down." It is prepared by Messrs. Murdoch, Chemists, in Glasgow; by Duncan and Flockhart, in Edinburgh; by Randall and Sons, Southampton; and by Savory and Moore, in London.

of iron and quinine, either in solution or in the form of a pill, is a new and useful preparation, in doses of five to ten grains three times a day.

Generally, such a preparation of iron must be found as will not check the digestion of other food. If loss of appetite and feverishness follow its use, the form of the remedy is probably unsuitable, and requires to be changed. Chlorine, in the form of warm hydrochloric acid baths, is highly spoken of by Dr. T. K. Chambers, as an agent of great value, and as a directly restorative medicine in anæmia (l. c., p. 332). The bowels are to be kept regular by four grains of the pills of aloe and myrrh taken at bedtime. Ferruginous remedies are always of use when we have reason to believe that there is a diminished energy in the formation of blood-cells; and if the urine be pale and almost neutral during the whole of twenty-four hours, it is as good an indication for the use of iron as the pallor of the skin in cases of anæmia (PARKES). Dr. Trousseau was of opinion, however, that for anæmia, complicated with tubercles, iron preparations were unsuitable, as tending to hasten their development. Dr. Clymer has found arsenic a valuable remedy combined in the form of chlorides of arsenic, iron, and quinine.

CHLOROSIS—Syn., GREEN SICKNESS.

LATIN EQ., *Chlorosis*—Idem valet, *Pallor luteus fœminarum*; FRENCH EQ., *Chlorose*—Syn., *Pales couleurs*; GERMAN EQ., *Chlorose*—Syn., *Bleichsucht*; ITALIAN EQ., *Clorosi*.

Definition.—*A deficiency of the blood-cells with redundancy of the serous part of the blood, occurring in young girls at the age of puberty; in the young of both sexes before the complete development of the distinctive characters of the sexes towards puberty; and sometimes during pregnancy.*

Pathology.—(a.) *Causation*—A very indefinite idea is associated with the name and nature of this disease. Many employ the term precisely in the same sense as anæmia; or they limit the term *chlorosis* to those forms of anæmia whose causes are unknown. The original use of the term is expressed in the first part of the definition as limited to that form of anæmia which occurs in the female at the period of puberty. It is to be classed amongst the “functional diseases of the female organs of generation in the unimpregnated state.” The conditions which bring about this constitutional affection are not well understood. Almost all pathologists now admit that the nervous system generally is always more or less implicated in these cases, but the real nature of the nervous influence is unknown. The disease is frequent in females between the ages of sixteen and twenty-five years, is often of long duration, lasting for months and even for years, with a tendency to relapse after cure.

There also appears to be no direct proportion between the percentage of the blood-discs and the functional troubles of chlorosis. It seems to arise even when hygienic conditions are most favourable, and it is by no means confined to the poor. The formation of the blood seems to be at fault. A form of chlorosis has also been described by Lebert under the name of “*Essential or Idiopathic Anæmia*” (*progressive pernicious anæmia*). Its causes are unknown; but the disease appears to have a great proclivity

or preference for pregnant women. This peculiar chlorotic condition, which Lebert described, is acutely febrile, supervening quickly after delivery. The absence of any definite organic lesion, and the presence, on the other hand, of the clearest symptoms of chlorosis in women who have scarcely lost any blood at all in their labour or after it, oblige us to look on this condition as a chlorosis due to the puerperal condition. One patient had general anasarca, without any albuminuria and without any signs of Bright's disease. There were three women in all who got well in from three to six weeks, under treatment by tincture of *malate of iron*, of which thirty to fifty drops were taken thrice daily. This condition appears to be developed *during the pregnancy* in some cases. One was a woman, aged thirty-three, in good health till her fifth pregnancy. During this she had slight symptoms of anæmia. A few days after delivery (in which she lost hardly any blood, and the labour itself was natural) alarming symptoms of severe anæmia set in. When admitted to hospital, sixteen days after the birth of the child, there were no signs of organic disease, but she had a quick pulse, hot skin, and a hæmic bruit. The urine (specific gravity 1017) appeared perfectly normal. For many weeks she remained in a restless, sleepless, and dangerous condition, spite of all treatment. But after rather more than two months she improved, and left the hospital in another month greatly better. There is in these cases a real rise of temperature not easy to account for. The second case, in 1853, of this anæmia was in a pluripara, aged twenty-nine, a cotton-spinner. Her symptoms began in the third month of her third pregnancy, but were not alarming till three or four weeks after apparently natural labour, without hæmorrhage. Her state continued alarming till nearly three months had elapsed. After observing three more cases, in 1854, Lebert again drew attention to these cases in his reports of the medical department for that year. Again he published a fatal case in the *Weiner Medicinische Wochenschrift* (1858, No. 34, 'Ueber essentielle Anämie'). This patient was only aged twenty-four. She had been married five years. She had had one child before, and the last labour, ten weeks before admission to hospital, was apparently quite normal. The lochia were rather scanty. There were, however, several attacks of epistaxis, short, and of no great severity, yet they seemed to greatly exhaust her. There were no signs of organic disease. She had a quick pulse (108 to 120); rapid breathing; hot, dry skin; extreme pallor of skin and mucous membranes, and all the rational signs of extreme anæmia, including the usual nervous symptoms. In spite of all treatment she died fifteen weeks after her labour, in most complete collapse. All the viscera, even the brain, were extremely anæmic; the blood in the cerebral vessels, and indeed everywhere, was paler than it should be (pale red, or pale brown). The large vessels everywhere were nearly empty. There were scarcely any clots or blood in them, or in the heart. The liver was somewhat enlarged. The heart's substance was pale and soft (fatty?). Other cases are reported by Lebert in his reports for 1867, and by M. Lépine in 1876; but the late Dr. Addison accurately described this disease, and distinguished it from that which bears his name (ADDISON'S Works, Syden. Soc., p. 212). Much confusion prevails regarding these cases of "*essential or idiopathic anemia*," as distinct from the various

forms of chlorosis and symptomatic anæmia which occur in women (*The London Medical Record*, July 14 and September 15, 1876).

Symptoms.—A change in the complexion constitutes the most striking symptom of chlorosis. A marked pallor of the skin prevails, sometimes perfectly pale, at other times yellowish, greenish, or waxen coloured. The lips and mucous membranes are also pale—symptoms which are the result of the deficiency of blood-cells, and so of blood-pigment. Slight dropsical swellings occur, such as œdema of the feet and ankles, the face and the eyelids, and a bluish halo sometimes encircles the orbit. The temperature of the body is generally diminished—the breath is cool, the lips, nose, ears, hands, and feet are cold; and chilliness of the body prevails, which is morbidly sensitive to external cold, and seeks warmth. As in other forms of anæmia, the patient suffers from great prostration of strength and debility; she is tired by the slightest exertion, and the weakness sometimes becomes so great as to lead to fainting. The patient is languid, listless, sedentary, and indisposed to exertion. She is nervous, low-spirited, and frequently a prey to irregularities of temper. *Hysteria* may prevail with *chlorosis*. The patient frequently suffers from vertigo, headache often recurrent, *tinnitus aurium* especially of the right ear, sparks in the eyes, tendency to fainting, neuralgia, spinal irritation, and convulsions, or a “heaviness for sleep.” The mind is sometimes morbidly impressed with grief, while despondency and frightful dreams prevail. There is the apprehension of nightmare, melancholy thoughts predominate, mania may become confirmed, and the insanity may assume a suicidal tendency.

Disorders of digestion attend the disease. Appetite is diminished or perverted, or even depraved. Acids and highly flavoured foods are craved for; and sometimes such substances as chalk, paper, ashes, coal, plaster of Paris, hair, earth, and even excrements, are desired to be eaten. Substances very difficult of digestion are longed for, and are sometimes digested better than simpler kinds of food. Constipation frequently and obstinately co-exists; but afterwards diarrhœa may alternate, and lead to *hæmatemesis* or *melæna*, when the evacuations are usually scanty, dark-coloured, and fetid. A singular and peculiar pain of one or both sides, which suffer together or alternately, is a frequent symptom. It is referred generally to the region over the false ribs and the *ilia*. The recurrent nature, the particular situation, and the alternating character of the pain, are peculiar and characteristic. It is not aggravated by a deep inspiration, although it may seem to be so at first.

Other functions are no less disordered; for instance, the respiratory, the generative, and the circulatory. Respiration is oppressed, or performed with difficulty, and the breath is offensive. Breathlessness prevails, and is experienced especially on any exertion. Sometimes also fits of dyspnoea; and sometimes a sonorous cough occur. Menstruation is generally absent, or performed imperfectly, irregularly, and with pain, and the flow is thin and watery, or leucorrhœal. The condition of the menstrual discharge is generally very easily impaired. The catamenia become irregular in their return, inconstant or of short duration in their flow, deficient in quantity, and pale in colour, terminating in a state of leucorrhœa. In some cases each return of the catamenia is preceded

and attended with much pain in the back and in the region of the uterus. Later in the disease there may be complete *amenorrhœa*. The conditions of the vascular system are variable. The pulse is slow, feeble, and soft, but sometimes frequent, and always easily accelerated. The heart and great vessels are irritable; palpitation is common; or, more frequently, a sense of fluttering in the *præcordia*, with irregular action of the heart, or imperfect syncope and murmurs, as already described, may prevail. It is to be observed, however, that, apart from mere lassitude and palpitation, patients are sometimes chlorotic for weeks, without any other marked symptoms; and the same complication of intercurrent acute diseases is apt to happen as described under anæmia. The condition of the urine is similar to that which prevails in anæmia.

Diagnosis.—It is especially necessary that the physician should not confound *chlorosis* with *jaundice*, on the one hand, or with *disease of the heart*, on the other; or with *scrofula* with *incipient deposit of tubercle*; or with inflammation within the chest or abdomen; or with the chlorosis of Addison's disease.

Treatment is chiefly conducted by change of air and diet, by moral and physical hygiene, and by medicines. All the functions of the body must be carefully observed and regulated. The residence of the patient must be a reputed healthy spot, in the pure air of the country, rather than the town. Dry frictions of the back and limbs are also recommended, with bathing in the sea, where it can be borne. The food must be regulated so as not to be too stimulating nor disgustingly bland; frequent change is demanded, with a due attention to the proper apportioning of nutritive and respiratory elements. The diet should be generous, and carefully apportioned to the powers of digestion. Any symptoms of dyspeptic oppression, impaired digestion, or offensive stools, demand a careful readjustment of the dietary. Three meals, or perhaps four, may be taken during the day—the intervals between the meals being equal; and, half an hour before each of the meals, two grains of *capsicum*, with one grain of *quinine*, may be given in a pill. The breakfast should consist of biscuit, dry toast, or stale bread, with fresh butter, and perhaps an egg, and one small cupful—not more—of hot black tea. Five or six hours after breakfast, and half-an-hour after the pill, a dinner of *well-done* meat, such as mutton chops, may be eaten with potatoes—beef-tea, or milk being taken for drink, according to taste and the powers of digestion. The evening meal may be similar to the morning. At first there may be a loathing of all kinds of food in the form of regular or "ordinary diets." In such cases no meals should be prescribed, and no solid food; but a cup of milk, with a third part of lime-water in it, may be given every two hours, prescribed as a medicine; and a pint of beef-tea in divided doses may be taken as a drink during the day. This diet may be gradually added to as the appetite improves. Trousseau observes that "there are some chlorotic girls who would rather die from inanition than eat ordinary food. We should not hesitate in such cases to make those therapeutic concessions which we are so often obliged to submit to in the practice of our art. I allow alimentary substances acknowledged to be very indigestible—caring not for the kind of food so long as it is eaten—as radishes, salads, fruits hardly ripe, highly

seasoned sausage meat, old cheese, vegetables, meats prepared with vinegar, acid drinks, and the like. All I require in this whimsical diet is, that there be sufficient variety. By such means we often succeed in rousing the digestive functions, bringing back the sensation of hunger, and gradually leading the patient to proper food." Lebert insists on the importance of artificial feeding in some cases. A mixed diet is essential.

Change of air and bathing will also greatly aid in restoring health. If the patient lives in a town, she ought to be sent into the country; and, best of all, to the sea-side, where sea-bathing may be prescribed, if the strength of the patient is sufficient. Should the patient have been living in the country, a change of air and scene to a lively town, with cheerful society; and, sometimes a little good wine with the food, will be no less useful as remedial agents. The much-be-quacked use of water, in its varied modes of appliance to particular cases, will be found a powerful restorative agent, if judiciously used by the patient under medical advice.

Of medicines, the preparations of *iron* have most reputation. They seem to act by promoting the formation of the red blood-corpuscles; and they operate best in those cases in which the blood is rich in albumen. They also act as stimulants to digestion; and, from what has been stated at page 1098 *et seq.*, it is not altogether immaterial which of the numerous *officinal* preparations of iron are to be prescribed. Almost every physician has some preparation of iron he fancies better than another; and some of them seem to be aided in their good effects by combination with *carbonate of potash*, such as the *sulphate of iron*, in doses of *three to five grains*, or of the *ferri sulphas exsiccata*, in doses of two to five grains, prescribed in pills. The use of ferruginous remedies must be persevered in for months, and the general indications for prescribing the preparations are the same as those given under *Anæmia*, page 1098. In pills, the iron preparations may also be combined with *extract of nux vomica*, or with *strychnia*, in suitable doses; or the *syrup of the phosphates of iron, quinine, and strychnia*, may be given (see page 1099); or the eliminative action of the colon is to be promoted by four grains of the *pill of aloes and myrrh* taken every night at bedtime, or by a grain of the *watery extract of Barbadoes aloes* with a little *gingerine* as a pill. Simple bitter tonics are useful adjuncts to the chalybeate treatment, such as *gentian*, *calumba*, and the preparations of *cinchona*. They aid feeble digestion. *Tartarated iron* (*Ferrum tartaratum*) is also a useful remedy, and may be prescribed with alkalies, in doses of six to twenty grains, dissolved in water.

Where a high degree of serous plethora exists, and produces violent excitement of the vascular system, palpitation of the heart, and congestion of the head, venesection may be practised. It not only acts as a sedative, but aids the radical cure of the disease, inasmuch as it causes the subsequently administered ferruginous preparations to be borne more easily. Both general and local blood-lettings may be used: but the blood must be taken in small quantities, a couple of ounces at a time being quite sufficient (VOGEL).

GENERAL DROPSY.

LATIN EQ., *Anasarca*; FRENCH EQ., *Anasarque*; GERMAN EQ., *Wassersucht*;
ITALIAN EQ., *Idropisia Generale*.

Definition.—*An accumulation of serum in the areolar tissue throughout the body; a general œdema or anasarca with effusion into one or more of the large serous cavities.*

Pathology.—(a.) *Causation.*—It is a form of extensive or universal œdema, and means an infiltration of the general areolar or connective tissue of the body with fluid serum, to which the name of *anasarca* has been given, from the Greek ἀνά, “through,” and σὰρξ, “the flesh” (see page 110, ante).

The disease usually begins to manifest itself by œdema of the feet and ankles—appearing towards evening, and diminishing, if not disappearing altogether, towards the morning. Such œdema is known from its “pitting” under pressure—that is to say, it retains the impression of the thumb and finger when they are made to grasp firmly each side of the ankle over the swollen part. The fluid surrounding the ankle may have gravitated there; but by and by, as the morbid condition persists which gives rise to the collection of fluid in the connective tissue, the swollen condition extends from the ankles up the limbs, encroaches on the abdominal and thoracic parietes, and, lastly, reaches the head and upper extremities. The whole body then exhibits a bloated and turgid appearance. Where the connective tissue is loose, the local swelling from fluid is generally the greatest; for example, over the dorsum of the foot and back of the hand, the eyelids, the scrotum of men, and the *labia pudenda* of females. The lower extremities are often so enormously swollen as to become shining and tense; when the cuticle may rise like a blister, or even giving way, an erysipelatous inflammation may pervade the limb. Sloughs are then apt to form in the connective tissue; open sores follow, and become the outlet of excessive discharges of serum, which greatly relieve the patient. Thus it is seen that watery accumulations are those which are properly to be accounted dropsical, and which are rather symptoms of disease than a disease itself. But it is a symptom which cannot always be traced to its source when the patient is yet alive, nor to its antecedent morbid action, nor to any organic change discoverable after death. Hence, practically, such a dropsy must be regarded as a substantive disease—the dropsy being then the sole object of treatment. In all cases it is also something more than a symptom; it is a cause of various other symptoms, which may constitute the greater part or the whole of the patient's distress. By its weight or pressure the dropsy may embarrass important functions, or even extinguish life. In general dropsy, which is the result of organic disease, there are two sets of symptoms to be distinguished:—namely, those which depend upon the primary disease, and those which are caused by the collection of the fluid; and the following account of them is condensed from the excellent and exhaustive Article “Dropsy,” by Sir THOMAS WATSON, in Tweedie's *Library of Medicine*, Vol. V., p. 96.

I. *Cardiac Dropsy.*—That the general dropsy is of cardiac origin is suggested,—(1.) By the presence of thoracic symptoms, such as cough and

dyspnoea, occurring prior to the dropsy; (2.) by the direct signs of cardiac disease, such as distended jugular veins, irregular movements of the heart, unnatural impulse, altered sounds; (3.) by the history of some previous acute disease, affecting especially the left side of the thorax, or particularly by an account of antecedent arthritic rheumatism; (4.) by the advanced age of the patient, rendering it probable that some of these organic changes in the heart and large blood-vessels are in progress which are almost natural in the decline of life; (5.) by the absence of all evidence of renal disease. Then comes the question, "Of what kind of heart disease is dropsy a consequence and symptom?" It is always found to be such a disease of the heart as produces a certain amount of permanent obstruction to the passage of venous blood—an obstruction which must be sufficient to distend the veins beyond their natural capacity. Such dropsy is usually associated with dilatation of the right chambers of the heart. This dilatation is at once an effect and a sign of impeded transmission of the blood from the right side of the organ; and the dropsy is not dependent on the dilatation. The actual conditions which imply an impediment to the flow of blood are such as mutually affect the heart and the lungs; and the order in which the diseases of these organs are connected as cause and effect, is a definite and constant order, taking place in the directions opposite to that in which the blood circulates. When dropsy supervenes we may then expect the signs which denote the dilatation of the right chambers. The heart is heard and felt to beat beyond the præcordial limits, the pulsations become feeble and unequal, if they were not so before. The patient is liable to fluttering palpitations, to extreme and panting dyspnoea on the slightest exertion, even on taking food into the stomach, or adopting the horizontal posture; his skin assumes a dusky hue, and his lips and extremities are apt to be livid. Anasarca first becomes manifest about the feet and ankles; and during the earlier stages the oedema disappears during the night's rest, and returns towards evening. It may be confined, for a considerable period, to the legs; but by degrees it ascends towards the trunk of the body, the integuments of which become doughy, the scrotum fills, and water collects in the serous bag of the abdomen and thorax. In extreme cases the dropsy is universal, pervading the cellular tissue of the head and face, and upper extremities. From continued pressure and stretching the areolar tissue loses its elasticity; the oedema is soft, and readily "pits" on pressure. Sometimes the fluid continuing to increase, the cuticle is separated by it, and large vesications appear on the limbs; or some part of the areolar tissue may slough, and a breach of surface being thus made, the fluid drains off by this vent in great abundance to the signal relief of the patient, and furnishes a valuable hint to the physician as a guide to treatment. The disease of the lungs which most commonly and certainly, though often very slowly, leads to dropsy, is dilatation with or without rupture of the air-cells (*emphysema of the lungs*). In this morbid state many of the smaller blood-vessels of the lungs become gradually obliterated, so that when the lesion is extensive and advanced the pulmonary texture is visibly white and bloodless. The blood does not readily pass through the pulmonary blood-vessels. A certain amount of accumulation becomes

habitual in the right side of the heart and great veins, and at length the capillary vessels feel the congestion, and more or less anasarca takes place.

General dropsy may also arise from mere debility of the muscular tissue of the heart, such as occurs in anæmia often; in cachexia; in exhaustion from habitual loss of blood; and whenever the muscular tone is considerably reduced. The heart-muscle is ill-nourished by the thin and watery blood. It cannot tighten on its contents with sufficient force to drive them effectually forwards. It may even dilate somewhat under the centrifugal pressure of the resisting fluid. The alternation of the blood concurs to facilitate transudation of its serosity through the coats of the minute vessels, and so dropsy ensues. This is a common source of anasarca in young, feeble, and delicate women; but it seldom goes beyond œdema of the legs. The trunk and arms may be occasionally slightly infiltrated, and the face puffy and bloated. It is rare that effusion takes place into serous cavities (see under Anæmia and Chlorosis). Tumors of various kinds (aneurism of the aorta, cancerous growths, &c.) occupying the thorax, and causing pressure upon the large veins, may also be a cause of general dropsy.

II. *Renal Dropsy*.—We may suspect renal disease as a cause of dropsy in a given case,—(1.) By the absence of any direct symptoms of cardiac disease; (2.) by the complexion of the dropsical patient; (3.) sometimes by certain parts of his previous history; (4.) by certain conditions and qualities of his urine. Here, again, the question suggests itself,—“With what kind or kinds of renal disease is dropsy apt to be associated as a consequence?” The cortical (or secreting) portion of the kidney is the primary and chief seat of the lesion; the medullary (or excreting) portion being sometimes implicated, but in a less degree. The size, figure, and consistence of the organ is altered; so also is the colour and condition of its surface and interior. The appearances vary greatly in different cases—varieties probably connected with different stages of the disorganising process. The average weight of the human kidney being *four* ounces, some morbid kidneys may weigh as much as twelve. Some are much larger, and some much smaller than natural: and such alterations of bulk belong principally, if not altogether, to the outer secreting portion of the gland. If a longitudinal section is made of a large kidney, the cortical part is seen to be unduly broad: and the same part is evidently narrow when the whole organ is smaller than common; and the radiating medullary parts approach nearer the surface than in a healthy kidney. Enlargement is more commonly coincident with the earlier, and contraction or shrinking with the later periods of the renal disease. *Consistence* also varies: sometimes the morbid kidney (mostly in the earlier periods) is soft and flabby; at other times (later periods) it is remarkably compact and hard. When the investing tissue is stripped off (less distinctly shining through it before separation) the surface appears mottled, marbled, or stained,—of a yellowish-grey colour in one place, and of a dark or purple tint in another. It may be pale throughout its whole extent, more commonly of many hues, and variegated with little streaks, which are portions of veins containing red blood. Sometimes the surface is curiously speckled by stellate congestion of the superficial veins; often it is uneven, as if strewed with prominent grains, and in some instances it is quite rough and granulated.

The most uniform and characteristic morbid appearance is presented by the cut surface when the kidney is cut longitudinally into two symmetrical halves. The cortical substance is then seen to be the main seat of the disease. It has lost, in a greater or less degree, or almost entirely, its natural red colour and uniform aspect. Sometimes it has a speckled or granular appearance; more often it has a pale homogeneous surface, generally streaked by linear markings, bearing, when well pronounced, a near resemblance to the section of a parsnip. The appearance is that of a deposit having taken place, whereby the natural texture of the part is obscured, and its blood-vessels emptied or obliterated; while the healthier medullary masses are displaced and pushed aside—sometimes compressed and encroached upon, sometimes exhibiting the same yellowish appearance interposed between, and opening out their radiating striæ. Firm coagula of blood may be found blocking up the veins emerging from the gland. Sometimes small cysts, full of clear fluid, stud the surface of the kidneys and throughout its interior. Another condition of the kidney is expressed by the term "*Sanguine Congestion*." The whole organ is then gorged with blood, which drips from it when it is cut into. It is generally somewhat flabby, of a deep and dark red, or even of a chocolate or purplish colour, nearly uniformly diffused, except that the exposed surface is usually diversified by still darker tuft-like spots, which have been ascertained to be the Malpighian bodies filled with blood.

Although dropsy is not a constant or essential symptom of any one of these lesions in the kidney, the circumstances which, during the lifetime of the patient, conduce to the conclusion that the dropsy under which he is seen to suffer is renal, are of the following kinds:—(1.) When the renal disease sets in suddenly, with acute symptoms, dropsy generally supervenes soon; and during its chronic progress anasarca is apt to show itself, or to increase wherever inflammation of any part or febrile disturbance happen to ensue. The more copious and rapid the effusion, the less do the dropsical parts pit on pressure; and an accumulation of fluid in the larger serous cavities is not in general a prominent feature. (2.) In all cases of general dropsy it is usual to look for evidence of disease in the thorax, especially of the heart; and most particularly of retarded circulation in the veins. "If we found no material or adequate embarrassment of the respiratory functions, no deviation from the natural sounds of the heart, no derangement of its regular movements, no alteration in the force of its pulsations, or in the space over which they may be heard and felt, no distension of the large veins of the neck, we then have reason to suspect, at least, that the anasarca is connected with some vice of the kidney." (3.) Our judgment is also guided or assisted, in some degree, by the complexion of the patient. When the general dropsy depends upon disease of the heart, the cheeks and lips are occasionally florid, often purplish or livid, frequently dusky, and loaded sometimes (as in chlorotic women, where the heart may be temporarily distended without any strictly organic disease, and the blood is thin and poor), the face and mucous membranes are pale; but in the renal variety of dropsy there is commonly a strikingly characteristic hue—an evident want of red blood, indeed, in the capillaries, but an unhealthy dingy sallowness withal—an aspect such as reveals to an experi-

enced eye the existence of some visceral disease in the abdomen. (4.) Certain accidents in the history of the patient will strengthen the suspicion that the kidney is the organ at fault. An attack of illness, for example, attended with perhaps temporary swelling of the body and disturbance of the urinary functions (*acute dropsy*), soon after some exposure (under unfavourable circumstances) to the influence of cold, either applied to the external surface or by a draught of cold drink to the stomach. There is reason to believe that in acute dropsy is laid the foundation of those peculiar changes in the kidneys which, since they were first pointed out by Dr. Bright, have been chiefly studied in their connection with chronic dropsy; that, as rheumatic carditis may occur and become latent as to its effects for some time, and yet implant the germs of future cardiac dropsy, so the stress or strain that befalls the kidney in cases of febrile dropsy may set on foot a morbid process which long works silently and unobserved, but which at last gives notice of its operations by symptoms—reproduction of dropsy in a more chronic form being the most significant symptom of all. The acute attack may have been forgotten. There may have been no obvious (though these may have been ill understood) indications of renal affection, and its existence have been unsuspected. The previous history of intemperate habits would be of importance also in diagnosis, if these same habits had not a like influence in causing disease of other organs (especially of the heart) as well as of the kidneys. Suppression of the catamenia and blows on the loins have also each been precursory of this form of dropsy. (5.) But the most remarkable and sure evidence of the existence of renal disease is to be found in the state of the renal secretion (see under Diseases of the Kidneys, Vol. II. of this work).

III. *Acute or Febrile Dropsy* is more nearly related to renal than to cardiac causes. It sets in suddenly and with violence, usually under recent exposure to cold under unfavourable circumstances, so that the function of some large secreting organ has been suspended, or materially checked. Its typical form is to be seen in dropsy after scarlet fever (see Scarlet Fever, p. 512, *ante*, and also under Diseases of the Kidney, Vol. II. of this treatise).

The emaciation caused by such cases is often extreme; but it is only manifest on the *anasarca* subsiding from any cause.

Diagnosis.—The progress of general dropsy is, as a rule, continuous, with longer or shorter intervals, when the disease does not seem to advance. It may resemble general emphysema, which is distinguished from *anasarca* by its crackling underneath the fingers on handling the parts.

The **Treatment** of general dropsy is regulated by the nature of the disease which causes the dropsical state, and will be considered under *Heart Diseases* and *Kidney Diseases* in the next volume.

BERI-BERI.

LATIN EQ., *Berberia*; FRENCH EQ., *Béri-Béri*; GERMAN EQ., *Beri-Beri*; ITALIAN EQ., *Beri-Beri*.

Definition.—*A constitutional disease, expressed in the first instance by anæmia, and culminating in acute œdema. It is marked by stiffness of the limbs, numbness, and sometimes by paralysis of the lower extremities, oppressed breathing (anxiety in paroxysm), and a swollen and bloated countenance. The urine is secreted in diminished quantity. The œdema is general, not only throughout the connective tissue of the muscles, but throughout the connective tissue of solid and visceral organs in every cavity of the body. Effusion of serum into the serous cavities very generally precedes death.*

Pathology.—(a.) *Historical notice.*—The name *Beri-beri* was given by the Malabar physicians (a name which has been handed down to us by writers on Indian diseases) to almost every fatal form of debility—paralyses of various kinds, reflex paraplegia, dropsy, anasarca, cachexia, scurvy, and anæmic rheumatism, with various diseases of the heart and pericardium. As these have all at some time been included under the common name of *beri-beri*, it is necessary to give to the term some degree of precision, by a definition which will embrace the leading phenomena of this obscure and very remarkably fatal disease. Practically it is unknown to pathologists in this country. Though common in various parts of India, the territorial range of its endemic prevalence seems limited in a peculiar manner. It is met with chiefly in the tropics; but also in China and Japan up to 40° of north latitude; on the eastern coast of Australia, and southwards of the Cape of Good Hope to 36° of south latitude (M. VAN LUNT, *International Congress Med. Science, Amsterdam*, Sept., 1879). It has been chiefly met with on the Malabar coast, in Ceylon, and in that tract of country reaching from Madras as far north as Ganjam (HAMILTON). It is principally endemic in that portion of Hindostan called the Northern Circars—a province lying on the west side of the Bay of Bengal, extending from 15° to 20° north latitude. Madras, in north latitude 13° 6', appears to be the southern limit of the disease in Hindostan. Towards the north of Madras, in the jails of Guntoor, Nellore, Masulipatam, Rajahmundry, Vizagapatam, Chicacole, Bellary, and Cuddapah, the disease is known to prevail. Towards the south it is never seen. It is said to extend from the coast not farther inland than forty to sixty miles. It has recently appeared in Bahia, where it is now tolerably common, and in 1871–72 it was rapidly increasing (DRS. PATERSON and DE SILVIA).

(b.) *Causation.*—A residence of several months in a district where *beri-beri* prevails is necessary to its development (CHRISTIE, *Edin. Med. Journ.*, Vol. III., p. 116); and the greatest predisposition to the disease exists when troops have been about eight or ten months in a settlement (W. HUNTER, Naval Surgeon, *Diseases Incident to Indian Seamen on Long Voyages*, 1804; EVEZARD, *Madras Quart. Journ. of Medical Science*, Vol. IV., p. 44). The influence of season in promoting the development of *beri-beri* seems to be remarkable. Towards the close of the rainy season the

admissions to hospitals are far more numerous than at any other period of the year: damp and moisture, combined with cold, seem very favourable to the production of the disease; and the most severe cases at Trincomalee occur during the change from wet to dry weather (RIDLEY, *Dublin Hospital Reports*, Vol. II., p. 227), when a strong and hot land wind prevails, when the atmosphere is extremely dry, and when the night temperature is many degrees lower than that of the day. From August to December, when heavy falls of rain occur, with occasional sultry days, and when the alternations of temperature are at the greatest, and the exhalations the most concentrated, then the admissions to hospital for beri-beri are the most frequent (WRIGHT).

Although the etiology of beri-beri is but little known, there are two points that seem well established in the history of the disease, and which must have a prominent recognition in all investigations relative to the causes which develop the disease. These points are,—*First*, The limited geographical range of *beri-beri*; *Second*, The fact that the morbid train of phenomena is never developed till the patient has resided upwards of eight or twelve months in the settlement where the disease is endemic. Nevertheless Dr. Paterson, of Bahia, writes me to say that he finds *beri-beri* “to attack persons in the most favourable conditions of life as regards means, residence, and regularity of life; and yet it is not sufficiently confined to our locality, or even to our houses, as to enable me even to suspect any local cause. Its favourite victims are parturient women, alike before and after their confinement” (see under Chlorosis—*essential* or *idiopathic anæmia* of Addison and Lebert, persistent progressive anæmia of the Germans, page 1101, *ante*). Malaria, alternations of climate and temperature, noxious material in the waters of districts, have all been indicated as operative agents in bringing about the disease. But, looking to the fact that all the phenomena of the disease point to anæmia, it may be generally stated that whatever tends to induce this state will favour the development of beri-beri. Accordingly, the disease will owe its origin in one place and in one person to the operation of a series of conditions which may not obtain in another place and with another person. Mr. Christie found in Ceylon that the aged and debauched were those most liable to the disease. A great proportion of his patients were men accustomed to lead a sedentary and debauched life, such as soldiers’ tailors, shoemakers, and the like, who, working at their trade, were often excused military duty, and who, by double earnings, were able to procure a larger quantity of spirits than other men. Generally it may be stated that all those who have studied this affection in the countries where it occurs most frequently, attribute it to the want of proper food, from which the inhabitants of such countries almost constantly suffer. To this are to be added such other causes as have been already noticed (under *Anæmia* and *Chlorosis*),—namely, overwork, pregnancy and lactation, dampness of climate, and the like. With regard to œdema there is a great tendency, in almost every disease in Europeans, to result in serous effusions after long residence in India. Indeed, Sir Ranald Martin observes, in his admirable introductory chapter to the second part of his work on the *Diseases of Tropical Climates*, that of all conditions incident to tropical invalids anæmia is the most common. With regard to some places—such, for

example, as Masulipatam (where *beri-beri* abounds, in the jails especially)—it has been observed that *œdema* is frequently one of the latest symptoms of *anæmia* outside the jail; that *anæmia* appears to be almost the normal state of about the third of the population, particularly the higher castes—a circumstance which is partly due to the constitution of the Telugoo, and partly to the eliminating power of the drinking-water, which contains so much of *chlorides* and no *iron*. The natives are well aware of the effects of the brackish water which the majority of them are obliged to drink at the end of the rainy season; and when they find themselves suffering from *anæmia*, they procure water from Goodoor, a village about six miles due west of Masulipatam. The water at Masulipatam seems to abound in saline material, such as chlorides, lime, magnesia, and a trace of alumina and iron, in the form of a carbonate (EVEZARD). The sanitary condition of prisoners in jails is, it is presumed, much worse than the free population outside. The criminals in the jails of India have just sufficient food to keep them alive, and scarcely variety enough. They have not sufficient food to store up for fat or warmth, and none of those luxuries which are supposed to check waste of tissue, such as tea, coffee, tobacco, betel, and the like; and they have not sufficient clothing, many of the prisoners having only one cloth. But the persistence of the *anæmic* state is more due to the damp and foul atmosphere of unventilated cells than to food. The soil is for the most part salt. The water of the tank in the jail compound is salt, and its recession leaves a wide margin of saline substance in the soil, which reattracts moisture like a cloth that has been wetted in sea-water, and which afterwards retains and absorbs the moisture. The *chunam* and mud of the jail walls, and the walls of the cells too, are salt; and in the morning they may be seen dripping with moisture, even in the dry season. Since improvements have been made in drainage, and since the introduction of a mass of iron, which is allowed to rust and corrode, into each *chatty* of water used by the prisoners for drinking and cooking, not a single case of *beri-beri* has occurred during the wet season. It is lying on the wet ground in wet weather which brings on *beri-beri* among Sepoys who have previously rendered themselves predisposed by voluntary starvation (EVEZARD). In Bahia Dr. Paterson is not disposed to consider *beri-beri* as primarily a dropsy; but rather as an affection of the (sympathetic) nervous system.

Under conditions such as these, it is not difficult to understand how a state of ill-health is insidiously established, during which period some *inbred* morbid material is developed in the constitution, which at last produces the phenomena of *beri-beri*, when certain endemic circumstances favour the full expression of the disease.

(c.) *Morbid Anatomy*.—On examining the blood under the microscope, the number of the red corpuscles is seen to be greatly diminished; they also seem to have lost their tendency to form rolls. It contains a large number of microcytes and of granulated brilliant bodies. The solid principles of the blood (albumen, fibrine, fat, salts) are greatly decreased in proportion. In short, the blood has become *hydræmic* (M. VAN LUNT, l. c.) More or less fluid is found in one or all of the cavities of the chest, most commonly in the pericardium. The areolar tissue of

the heart is in some instances loaded with fluid, and hypertrophy of its substance is not unfrequent. The areolar substance of the lungs is loaded with water, likewise the substance of the brain. The ventricles contain an increased amount of fluid; and fluid is effused over the surface of the brain. The abdominal cavity and the general connective tissue of the body abound in fluid. In general, it may be said that in every case examined after death serous fluid in one or more cavities was found to exist; and the pericardium alone, or in conjunction with some other cavity, was the seat of effusion in above 83 per cent. of the cases—the quantity of fluid varying from two ounces to a pint. The spinal cord is variously altered, either by minute effusion of fluid into its substance, or by congestion of its vessels, thereby accounting for the phenomena of paralysis. The unsteadiness of gait and the paralysis of the lower limbs may be also in some measure explained by the mechanism of the spinal canal, combined with the presence of serous effusion in its connective tissue. When it is remembered that the spinal cord more completely fills the vertebral canal in the dorsal region than it does either above or below, it will be understood how symptoms of compression from serous effusion will most early manifest themselves by paralysis or unsteadiness of gait in the lower limbs. The kidneys have been found enlarged, softened, and anæmic; the heart is generally found enlarged, pale, flabby, and softened; the lungs are œdematous, and serous effusion into minute connective texture is the only uniform appearance which exists in every case (WARING, *Notes on Diseases of India; Indian Annals*, 1856, III., p. 490).

Symptoms.—The majority of the phenomena which characterise the well-recorded cases of this disease are undoubtedly referable to *anæmia*, or to chlorosis, and by the Germans it is regarded as a variety of *pernicious progressive anæmia* (see p. 1101, *ante*). An impoverishment of the blood exists, with all the symptoms of serous anæmia. The debility and increasing prostration, the cold extremities, palpitation, dyspnœa on exertion, frequent, small, and quick pulse, the bruit occasionally heard in the neck, the scanty urine, the torpid bowels, the deadly pallor of the tongue, all indicate a condition of anæmia (EVEZARD). The disease makes its advances in an insidious manner, as all forms of anæmia do, without any primary or well-marked train of symptoms; and the indisposition appears to be comparatively slight which exists as a stage precursory to the visible invasion of the fully expressed disease (WRIGHT, EVEZARD). The approach of the final and characteristic features of the disease appears to be very gradually brought about; a constitutional state or diathesis is gradually established, and a form of anæmia sets in, combined with the cachectic dropsy of Andral—a condition allied to that of chlorosis in the female (EVEZARD). Pains and formication of the lower extremities supervene, the limbs become stiff, and motor paralysis at last comes on. At the same time the lower limbs become anæsthetic; and the anæsthesia gradually spreads over the whole of the cutaneous region, coinciding with muscular hyperæsthesia. The patient finally has anasarca, and effusions take place in the serous cavities. The body temperature is low (97·8° Fahr.) The affection is essentially chronic; sometimes there is an apparent amelioration of the symptoms. Many of the more early observers of the disease concur

in regarding the fully expressed phenomena of beri-beri as the result of exhaustion and debility (FARRELL, DICK, DUNCAN'S *Edin. Medical Commentaries*, Vol. X., p. 207, RIDLEY). Like rheumatism and gout, the disease expresses itself under several forms, of which three may be noticed, namely:—(1.) The *Acute, severe, or inflammatory form* is generally the culmination of the constitutional and local phenomena in a first paroxysm. Numbness, paralysis, and œdema of the extremities are the leading symptoms, followed by dyspnœa and oppression at the *præcordia*. For a short time previous to any other obvious symptom, the patient, though robust-looking, may not have been able to exert himself in consequence of the partial loss of the use of the lower limbs. This rapidly increases, till he finds that there is inability to walk, accompanied with œdema of the extremities, which very soon passes into general anasarca, affecting the innermost recesses of the textures—if such an expression may be permitted. Febrile symptoms are associated with this acute anasarca. The skin is hot and dry, the urine is scanty and high-coloured, the bowels are costive, and the stomach irritable. There are rapid and full pulsations of the large arteries, while the pulse may be variable at the extremities, accompanied generally with dyspnœa and symptoms of effusion within the chest. In other cases there may be headache, restlessness, and delirium, with a slow and full pulse, indicating serous effusion and pressure on the brain. When the œdema is general, and becomes rapidly developed, the condition of the blood is changed from its anæmic character. It becomes dark and ropy, resembling in some degree the appearance of the blood taken from a patient affected with cholera (WRIGHT). (2.) In the *second, the asthenic, or chronic form* of beri-beri, the patient is very often more or less worn out by some previous disease; or he may have had a previous acute attack, of which there may be a relapse; and it appears that men in whom the disease has once manifested itself are the more subject to future attacks (CHRISTIE), for it is found that one attack predisposes to another (WRIGHT); and then the dropsical symptoms more generally resemble those observable after protracted fevers or other debilitating causes. Abdominal dropsy is most prevalent, accompanied with symptoms of general relaxation—a small and quick pulse, constipated bowels, scanty urine, loss of appetite, universal œdema, much pitting on pressure, and paralysis of the extremities. The heart partakes of the general debility. It is flabby, and the venous circulation becomes retarded. Soon, perhaps, it dilates, when a temporary bellows sound may be heard. After several such attacks and recoveries the heart becomes thickened, and hence we have the *post-mortem* appearance of either a large and flabby heart, or of one eccentrically hypertrophied (EVEZARD). (3.) In the *third and mildest form* the patients are first attacked with some stiffness or rigidity of the legs and thighs, succeeded by numbness, slight œdema, and sometimes paralysis of the lower limbs. The œdema is in general limited, with slight pitting on pressure. There is no unnatural heat of skin; the pulse is seldom above the natural standard; the urine is scanty; and the appetite unimpaired. There may be occasional palpitations of the heart, with costiveness, blanched conjunctivæ, flabbiness and paleness of the tongue, and whiteness underneath the nails. Although such patients generally say that they are well, they will sometimes acknowledge a slight feeling of

numbness and coldness of the extremities—symptoms which would readily disappear under appropriate treatment; but after a close night, with either a fog or a shower of rain, such a patient would apply for medical aid in the morning, with a scared aspect, sighing breathing, violent palpitation of the heart, sometimes with a diffused impulse, pain in the præcordial region, and a variable fluttering pulse. In such cases there are also dyspeptic symptoms, with acid eructations and puffiness of the stomach. The scanty and high-coloured urine has an acid reaction when voided, shows a specific gravity of from 1025 to 1040, and contains an excess of urea. It is such cases which are apt *suddenly* to become aggravated, and to pass into the acute form of the disease. The oedema then progresses from the lower limbs to the hands; the throat becomes swollen, and the face bloated. A sense of numbness is experienced round the mouth, and a general sense of numbness is experienced all over the body, particularly over the extremities, which appear to be unusually weighty and rigid (hence, when walking, the gait is unsteady). The urine, secreted in diminished quantity, is extremely hot when passing through the urethra; and as the disease progresses, a total suppression of the urine generally takes place. A sense of pain and tightness is felt immediately beneath the inferior edge of the sternum, which sometimes becomes so distressing as to induce the patient to solicit that the part may be cut open, hoping to relieve the tightness by that means. Dyspnoea becomes so urgent as to prevent the patient from lying down; heavy sighing (*suspirous breathing*) occurs, with great anxiety and restlessness. The lips and tongue may now be seen to be livid, and the extremities become cold (RIDLEY). Universal debility, extreme prostration, anxiety, dyspnoea, numbness, oedema, anasarca, and paralysis, are the most characteristic phenomena of the disease. And in some instances, when these phenomena are fully expressed, the advent of the fatal event is so rapid that the patient may die within six, twelve, twenty-four, or thirty-six hours; and in other instances the disease is more frequently prolonged over several weeks. Death is in some cases extremely sudden; and from the anxietas, and the fact that obstructions sometimes occur in the veins (EVEZARD), it is not improbable that death in such instances may be due to *embolism*. Mr. Ridley especially notices the suddenness of dissolution in most of the cases which he observed among the troops in garrison at Ceylon in 1814. "Very frequently, when speaking to one man," he says, "I have been called to another, whom I had just before left under promising circumstances, and have found him gasping, his eyes protruding, his hands clenched, and in a few minutes he was dead; and it has sometimes happened that the man I was addressing has been taken off in the same manner" (*Dublin Hosp. Reports*, Vol. II., p. 234). It is clear, therefore, that the disease cannot be referred to granular disease of the kidney with *albuminuria*, although albumen is sometimes present in the urine in a high degree, as Dr. Ranking attempts to show; nor yet with scurvy, as Dr. Morehead teaches. It seems rather to be a constitutional disease *sui generis*.

In Bahia Dr. Paterson recognises three forms:—(1.) In which dropsy exists as the prominent symptom; (2.) in which paralysis is the leading feature; (3.) mixed cases, in which the dropsy and paralysis are equally predominant.

Diagnosis.—The most uniform phenomena in the fully expressed disease is the characteristic numbness of the surface generally, and the paralytic affections of the lower extremities which accompany all the forms of the disease. The disease has been confounded with the *barbiers*—a chronic disease in which paralysis, tremors, spasms, contractions of the limbs, and emaciation are the most remarkable symptoms. Some, however, are inclined to regard the acute or first form described as the true *beri-beri*, while others consider the second form described as the disease known by the name of *barbiers*. It is admitted, however, that the one is often associated with the other, either of them being the primary affection; for cases commencing in the form of *barbiers* often suddenly take on the more fatal and acute form of *beri-beri*, while the latter frequently presents the symptoms characteristic of the former (MALCOLMSON, *A Practical Essay on the History and Treatment of Beri-beri*, Madras, 1835). The two classes of cases prevail in the same places, seasons, and circumstances, and require the same treatment. It is, therefore, more consistent to consider the disease under the three forms described in the text.

The Prognosis is generally unfavourable, the disease approaching insidiously, the anæmia and constitutional state having often advanced so far as to be beyond the reach of repair. A temporary recovery may be established, but relapses are frequent, convalescence is lingering, and as a rule the patient never recovers, (M. VAN LUNT, l. c.) A first attack generally leaves unpleasant phenomena behind. In the case of Mr. Ridley, a surgeon of the Royal Artillery, who wrote an excellent account of the disease as he saw it in Ceylon, and who suffered from it himself, the memory became considerably impaired, and an extraordinary fluttering of the heart, almost producing faintness, occurred at intervals after convalescence was established. Violent palpitation succeeded, which, on subsiding, left extreme lassitude and faintness; and when these phenomena first occurred, a violent beating of the carotids came on; and when this rapid pulsation ceased, he was left so extremely weak and languid as to excite considerable apprehension of a fatal result. In all cases of *beri-beri* a suddenly fatal result may be apprehended at any period after the full expression of the phenomena, probably from *embolism* or *cardiac affection*. The symptoms which denote danger are general œdema, much *anxietas*, and palpitation of the heart, the respiration becoming more quick as the fatal termination approaches. It is laboured, quick, and interrupted with sighs and groans, the pulse becoming weak and irregular at the extremities, with palpitations of the heart. Death generally comes on in the midst of nervous complications, or from exhaustion following vomiting; and if coma, delirium, or convulsions supervene, a fatal event may be expected. Much nausea, obstinate costiveness, a dry and black tongue, are all symptoms of danger. The prognosis is more favourable if the œdema sets in very gradually, and is confined to the lower limbs, when the skin is moist and moderately warm, when the patient enjoys sleep, and when the principal natural functions are unaffected.

It is a disease of a peculiar nature, which has been extremely frequent and fatal amongst all the troops, both Europeans and natives, at Ceylon (CHRISTIE); and although *beri-beri* is a far more common disease among the natives of India than among the Europeans, yet the rate of mortality

is nearly twice as great among Europeans as it is among the natives. Indeed, next to cholera beri-beri must be regarded as the most fatal disease (judging by the proportion of deaths to admissions) to which Europeans in India are liable (WARING); while Mussulmans appear to be more subject to the disease than Hindoos (MALCOLMSON). Among European soldiers in India the ratio of mortality, or of deaths to admissions, from beri-beri is above 26 per cent., and amongst Sepoys it is nearly 14 per cent.; and large as these ratios seem, they are small compared with the ratio of mortality which obtains amongst the convicts in the Indian jails, where the percentage is as high as 36·5 (WARING).

Treatment.—*Bleeding* has been considered applicable to those cases where there is extreme difficulty of breathing and delirium, when the patient is robust, and when the œdema does not pit much on pressure, where there is rapid and full pulsation of the large arteries, and if the urine shows the existence of albumen (WRIGHT); but, judging from the pathology of this disease, the constitutional influence of stimulants, of a generous strengthening diet, of tonics, diuretics and analeptics, ought undoubtedly to be the basis of treatment. Should there be irritability of the stomach, an *effervescing draught*, with doses of *laudanum* and *camphor mixture*, are useful. Saline drinks should be administered, and the extremities should be rubbed with stimulating liniments, and rolled in flannel bandages. In the *asthenic* or *chronic* form of the disease, the strength must be supported by the most nourishing diet that can be given in small bulk, aided by tonics, and wine if necessary; while doses of equal quantities of *squill* and *digitalis* (ten to fifteen drops of each) may be given twice or thrice daily. In the *third* and mildest form, a native remedy called *Treeak Farook* is very useful. The ingredients of this medicine are unknown; but it professes to be the "*Theriaca Andromachi*" of old writers. It is prepared in Venice, and transmitted to India through Arabia, and was first recommended by Dr. Herklotts, of the Madras Presidency, as a remedy in beri-beri. It is a thick extract (in which some terebinthine material largely enters), which is only to be procured from the Moghuls, and in those towns which still keep up some communication with the Arabian Sea. Many observers bear testimony to its good effects in removing the œdema and subduing the pulse (WRIGHT, TRAILL, *Madras Quarterly Med. Journ.*, 1842, Vol. IV., p. 154, GEDDES, MALCOLMSON). In some recorded cases the pulse has fallen in four days from 108 to 84 beats per minute under its use. The prescription most approved of consists of pills of the following ingredients:—

R *Treeak Farook*, ʒss.; *Pulv. Rhei.*, ʒiiss.; *Confectio. Aromat.*, ʒss.; *Mellis*, q. s., misce, et divide in pill xlvi. (MALCOLMSON).

The electuary form is also much used in India. The remedy does not seem to be an active medicine, except in combination. Four or five stools are obtained daily under its use, and its action is not accompanied by any violent purging, increase of pulse, or determination to the surface; and after it has been used from one to two weeks, the œdema generally disappears, when the numbness and paralysis subside. If it purges, the

quantity of rhubarb must be diminished. The patient should feed on animal food, wheaten cakes, and milk. In instances where the native remedy has failed to produce a beneficial effect, *nux vomica* has been more successful, commencing with doses of two grains of the *extract* daily, and increasing the dose gradually according to the physiological result. The *extract of nux vomica*, in doses of half or a quarter of a grain, combined with iron or gentian in a pill, is the most convenient form. Local abstractions of blood from the spine have also proved useful; and a blister applied over the loins has given relief in many obstinate cases. No single rule of treatment will apply in all cases. The anæmic condition must be counteracted on the principles of treatment explained under that disease; and any specially abnormal state, such as diseased heart, must be treated accordingly. When the disease prevailed very generally in the Carnatic, in 1782 and 1783, some cases were most successfully treated by a pill containing a quarter of a grain of *extract of elaterium*, combined with *extract of gentian*, given every hour until copious watery evacuations were procured; and this plan was repeated every third or fourth day, till with others this plan of treatment was not so successful; the cases recovering best under large doses of *spirits of nitre*, *antimonial wine*, *frictions with warm camphorated oil*, *aperient medicines*, *wine*, and a *nourishing diet*. Mr. Evezard's method of medicinal treatment consists mainly in the administration of *acetate of potash* in gin, in the following formula:—*R. Gin, ʒss. ; Potass. Acet., gr. v. ; Aquæ, ʒiii. ; misce.* To be given three times a day. If vomiting persists, *hydrocyanic acid* may be useful in relieving it, to the extent of one drop three times a day, given in milk. In the experience of Dr. Paterson of Bahia, a trip to Europe (if not had recourse to too late) is a certain cure: and medicinally, his patients have derived marked benefit from a combination of *ergotine*, *iron*, and *extract of belladonna*, in the form of a pill. Sea-bathing is also acknowledged to be of great service.

CHAPTER X.

INFLUENCE OF FOOD ON SOME CONSTITUTIONAL DISEASES DESCRIBED IN THE PREVIOUS CHAPTER.

RIGHTLY to understand the nature of many of the GENERAL DISEASES of a CONSTITUTIONAL kind which have now been described, it is necessary to consider the influence and effects of food on the human frame; and it is not less necessary to define what is here meant by "Food." Food must be taken to represent a combination of alimentary substances containing certain elements suitable for digestion and assimilation, all of which are necessary and sufficient to maintain healthy nutrition; and by its potential or latent energy, goes to maintain the body in all its functions of healthy life, under extremely variable circumstances; such as, (1.) *Living at ease on a merely "subsistence diet," just sufficient "to keep body and soul together;"* (2.) *living and working—(a.) mentally, (b.) bodily, or (c.) both*

mentally and bodily; (3.) hard work—(a.) mentally, (b.) bodily, or (c.) both mentally and bodily.

Three ways of studying the influence of food in health and disease are thus called for on the part of the student of medicine—namely, (a.) chemico-physical, (b.) physiological, (c.) clinical. Combined researches in chemistry, physics, physiology, and pathology, during recent years have cleared up much that was doubtful, and established certain principles on which proper scales of diet may be founded, so as to maintain the health of the body under a great variety of conditions as to labour, confinement, freedom, and exercise. Statistics have shown, more decidedly now than hitherto, how intimately disease and mortality are associated with the supply of food to the people. The records of the Registrar-General of England, and those of Sir William Wilde in Ireland; the evidence of the Commissioners relative to the supplies in the Crimea during the war against Russia in 1854, 1855, and 1856; many valuable reports relative to the health of the Navy before and after the diet was changed; and, still more recently, the knowledge relative to the famines of India and China, fully substantiate these statements.

While physicians were well aware of the intimate connection that existed in a general way between food and disease, it is due to the more recent labours of chemists and physiologists especially, that this connection has been reduced to an intelligible form, and that the principles which they have established are now put to a practical use. The great fact which recent chemical and physiological investigations have established may be expressed thus:—“*That the various alimentary substances made use of by man and animals contain at least four classes of constituents, each of which performs its own assigned function in the living animal economy. If the substance contains nitrogen, it seems most fitted for the nourishment of tissue, and has been called plastic or nitrogenous; if it is deficient in nitrogen, and has an excess of carbon or hydrogen, it appears to undergo combustion in the body, and is called a non-nitrogenous or a respiratory element of food (hydro-carbons); if it is fatty in its nature, it performs the double duty of maintaining animal warmth, and of assisting in the assimilation of nitrogenous compounds; and, lastly, if it is saline in its quality, it goes to build up the solid textures of the animal frame, and aids the important work of carrying new materials into the system and old or effete matter out of it*” (LETHEBY). Man and animals cannot maintain health if their food does not contain all of these constituents. A proper combination of these elements of food is necessary to maintain perfect nutrition; and common instinct, with experience, tells us that these classes must be associated in due proportions, under a variety of modifying circumstances. There are undoubted *habits* of feeding which, while they appear to be dictated by common instinct, are also sanctioned by science. For example, white meat being deficient in fat, bacon is eaten with veal and with fowl; melted butter is used with fish and vegetables; eggs and butter are mixed with sago, tapioca, and rice; cheese is eaten with macaroni; salads and vinegar are eaten with cold or salted meat; a vegetable is mixed with an animal diet; bread is eaten with butter, bacon with greens, pork with pease pudding, and so on. Old habits and instincts not only declare that these combinations are compatible, but science informs us now why such combinations are demanded for the maintenance of health;

and when they cannot be obtained, health is endangered, the constitution is gradually altered, temperament is modified, life is shortened, families extinguished, armies are swept from their encampments, and races of men from the face of the earth. The experience of Sir Robert Christison has shown,—(1.) That the most successful dietaries for bodies of men, deduced from practical observation, contain carboniferous and nitrogenous food in proportion of about three of the former to one of the latter by weight. (2.) That while *nitrogenous* may replace *carboniferous* food for supporting respiration, *though at a great loss, carboniferous* food (without nitrogen) cannot replace *nitrogenous* food for repairing textural waste. (3.) The daily amount of nutritive principles of both sets must increase with exercise and exposure, otherwise the body quickly loses weight, and ere long becomes diseased. If the above proportion between the two sets be maintained, the weight of real nutriment per day varies, for adults at an active age, between seventeen and thirty-six ounces; the former being enough for prisoners confined for short terms, the latter being required for keeping up the athletic constitution, or that which is capable of great continuous muscular efforts. (4.) Dietaries ought never to be estimated by the rough weight of their constituents, without distinct reference to the real nutriment in these as determined by physiological and chemical inquiry. As the late Dr. Parkes has not only given the results of those who have practically worked at this important and difficult subject; but has to a large extent verified those results, and made many original experiments on the subject, the remainder of this chapter will embody a summary of his observations (*Practical Hygiene*, Fifth Ed., edited by Dr. DE CHAUMONT).

Milk being held to contain all the necessary aliments, and in the best form for the maintenance of human life (PROUT), its analysis has been used as the basis for an enumeration and classification of the foods, or aliments necessary for the maintenance of life in its most perfect state. The substances in milk are—(1.) The nitrogenous matters—namely, the casein principally; and, in smaller quantities, albumen, lacto-protein, and perhaps other albuminous bodies; (2.) the fat and oil; (3.) sugar in the form of lactine: water and salts, the latter being especially combinations of magnesium, calcium, potassium, sodium, and iron, with chlorine, phosphoric acid, and in smaller quantities sulphuric acid. The different members of each class *inter se* have a remarkably similar composition, while there are broad lines of physical and chemical demarcation between the classes: and, while the different classes appear to serve different purposes in nutrition, all are necessary for the maintenance of perfect health. The nitrogenous aliments are blood fibrine, muscle fibrine or syntonin, myosin, vegetable fibrine, albumen (in its various forms), and globulin. Their composition is remarkably uniform, containing between 15.4 and 16.5 per cent. of nitrogen. They are now conveniently distinguished by the common term of “albuminates;” and they can replace each other in nutrition. The second class consists of the various animal and vegetable fats, wax, and the like, the composition of which is also very uniform, and the chief nutritive differences of which depend on physical conditions of form or aggregation, which cause some fats when acted upon by the alimentary fluids to be more easily absorbed than others.

The group of the starchy and saccharine substances (the *carbo-hydrates*), or of their allies or derivatives (*dextrine, pectin*), is equally well characterised by chemical resemblances *inter se*, and differences from the other groups. The several dietetic starches, sugars (including lactine), cellulose, and the various derivatives of the starches, are all closely allied. The fourth class consists of the salts already noted, and of water. There is also considerable physiological evidence to show that these classes of aliments serve different purposes in nutrition; and with regard to the necessity of all the four classes, it can be affirmed with certainty that (putting scurvy out of the question) men can live for some time and be healthy with a diet of *albuminates, fat, salts, and water*. But that special conditions of life, such as great exercise, or exposure to very low temperature, appear to be necessary; and under usual conditions of life health is not very perfectly maintained on such diet. It has not yet been shown that men can live in good health on *albuminates, carbo-hydrates, salts, and water*, without *fat*. In some experiments which the late Dr. Parkes made with Liebig's essence of meat, and Hassall's dried food with bread, he was much struck with the bad effect produced on the health of the experimenter, and with the immediate relief given by the addition of butter and a larger supply of starch, without augmentation in the amount of nitrogen. The exact effect produced by the deprivation of any one of these classes is not yet known. Health cannot be maintained on *albuminates, salts, and water* alone; and, on the other hand, it cannot be maintained without them. The *albuminates* cannot take the place of starch or fats in the case of human nutrients, and fat must be taken in its own form. The *salts* and *water* are also quite as essential as the *nitrogenous* elements. Lime, chiefly in the form of phosphates, is absent from no tissue; and there is reason to think no cell growth can go on without it. In large morbid growths, and in rapidly growing cells, it is in large amount. The lowest forms of life (bacteria and fungi) will not grow without earthy phosphates. Magnesia is probably also an essential constituent of growth in some tissues. Potash and soda, in the form of phosphates and chlorides, are equally important: they seem to be especially concerned in the molecular currents. Forming parts of almost all tissues they are less fixed, so to speak, than the magnesian and lime salts. It is also now certain that the two alkalies do not replace each other, and have a different distribution; and while potash seems to be the alkali for the formed tissues, such as the blood-cells and muscular fibre, the soda salts are more largely contained in the intercellular fluids which bathe or encircle the tissues. The chlorine and phosphoric acid have also very peculiar properties, the former apparently being easily set free, when it gives a very strong acid, which has a special action on albuminates; and the latter has remarkable combining properties with alkalies. Both are supplied in almost all food; the *sodium chloride* to a large extent separately. Carbonic acid is both introduced and made in the system, and probably serves many uses. Iron is, of course, also essential for certain tissues or parts, especially for the red blood-corpuscles, and for the colouring matter in muscle; and in small quantity it is found almost in every tissue and in every food. The sulphur and phosphorus of the tissues appear to enter as such with the albuminates. The salts which form carbonates in

the system, such as the lactates, tartarates, citrates, and acetates, give the alkaloids to the system which seems so necessary to the integrity of the molecular currents. That state of malnutrition, which in its highest degree we call scurvy, appears to follow inevitably on their absence; and as they exist chiefly in fresh vegetables, it is a well-known rule of dietetics to supply these with great care, though their nutritive power otherwise is small. So important are these substances from this point of view, that they might well be placed in a separate class.

In addition to the substances comprising these four classes of alimentary elements, there are others which enter into many diets, and which have been termed "accessory foods," or "force regulators," like the salts. The various condiments which give taste to food, or excite salivary or alimentary secretions; also tea, coffee, cocoa, alcohol, and such like, furnish the chief substances of this class. As to the exact action in nutrition of these substances but little is definitely known; and it is impossible at present to assign to them their true action.

Admitting, therefore, the necessity of a certain amount of each of the four classes of alimentary elements for a perfect diet, the question necessarily follows,—“What quantity of each class is necessary for the daily diet? and how far the latent energy in each class may be best secured for the purposes of the body?” The daily quantity of food has been determined partly by observation of the results of a great number of dietaries, and partly by physiological experiments, the general results of which are given in the following tables:—

TABLE I.—AMOUNT IN OUNCES (AVOIR.) AND TENTHS OF OUNCES DAILY FOR MALE ADULTS (Playfair).

WATER-FREE SUBSTANCES GIVEN DAILY.	Subsistence Diet (PLAYFAIR) i.e. sufficient for the internal mechanical force necessary to carry on the work of the body to keep a man merely alive.	Diet at rest in quietude.	Adults in full health, but with moderate work (MOLESCHOTT).	Adults in very laborious work (PARKES).
Albuminates,	2	2·5	4·587	6 to 7
Fats,	0·5	1	2·964	3·5 to 4·5
Carbo-hydrates,	12	12	14·257	16 to 18
Salts,	·5	·5	1·058	1·2 to 1·5
Total water-free food, :	15·0	16·0	22·866	26·7 to 31·0

These diets are estimated for a male European adult, of the average height, (5 ft. 6 in. to 5 ft. 10 in.) and average weight (140 lbs. av. to 160 lbs. av.) Dr. Parkes thus sums up the general results:—Assuming the water-free food to be 23 ounces, and a man's weight to be 150 lbs., each pound weight of his body receives in 24 hours 0·15 ounce, or the

whole body receives nearly $\frac{1}{100}$ part of its own weight of dry *water-free* food. But a certain amount of water (between 50 and 60 per cent. usually) is contained in it; and adding this to the water-free solids, the total daily amount of so-called dry food (exclusive of liquids) is about 40 ounces. In addition to this, from 50 to 80 ounces of water are taken in some liquid form, making a total supply of water of 70 to 90 ounces; or, on an average, 0·5 ounce for each pound weight of the body.

But this average daily amount of food and water varies considerably from the following causes (some of which are recognised in the above Table I.):—(1.) Individual conditions of size, vigour, activity of circulation and of the eliminating organs. No men eat exactly the same, and no single standard will meet all cases. The usual average range in different male adults is from 34 to 46 ounces of so-called solid food, and from 50 to 80 ounces of water. (2.) Differences of exertion.—If men are undergoing great exertion they take more food; and if they can obtain it, the increase is especially in the classes of albuminates and fat. The amount of water, too, is increased; but is very various according to circumstances, and is not so much augmented apparently as the solid food. On the other hand, men at rest in quietude will usually eat less; at any rate, the weight and health of the body can be maintained on less. The subsistence diet given in the table, though it will keep a man alive, will probably not prevent him from losing weight, and therefore it is not really sufficient. (3.) Difference of climate.—More food is taken in cold seasons and in cold countries than in hot. On an average, then, it is found that a man requires *four or five* ounces of chemically dry NITROGENOUS food daily (albuminates); that in a state of rest he will require *three and a half* ounces; under a state of considerable exertion *five and a half* ounces; and under extraordinary exertion he may require as much as *six and a half*, or even *seven* ounces of dry NITROGENOUS aliment daily. The quantity of CARBO-HYDRATES required to keep a man in health cannot be less than from *fourteen and a half* to *fifteen* ounces in twenty-four hours; and even *nineteen* to *twenty-two* ounces under great exertions. The amount of FATTY MATTERS ought to equal about half the quantity of the *nitrogenous* aliment. But in a state of rest about *one* ounce in the twenty-four hours will be sufficient; while under great exertion *two and a half* ounces may be required daily. The amount of WATER required varies from *seventy to one hundred and thirty* ounces; and the SALTS supplied in the food should amount to from *half an ounce* to *an ounce* daily, consisting of *chloride of sodium, chloride of potassium, salts of lime and magnesia, carbonates, citrates, lactates, and acetates* (PARKES, l. c.)

To determine by calculation the amount of these different alimentary principles, and therefore the nutritive value of a given diet (the articles of which are known), the following table (II.) of mean composition is given. It shows the mean amount of water, albuminates, fats, carbo-hydrates, and salts, which are contained in 100 parts of each of the substances in common use.*

* This table was devised by the late Dr. Parkes, and is in use in the Laboratory of the Army Medical School. He kindly permitted me to give it in the previous edition. It has been greatly more amplified since that time, as it appears in the fifth edition of *Practical Hygiene*, edited by Dr. De Chaumont.

TABLE II. (WITH FOOTNOTES), FOR CALCULATING DIETS (Dr. Parkes).

Articles.	IN 100 PARTS.				
	Water.	Albumi- nates.	Fats.	Carbo- hydrates.	Salts.
Meat of best quality, with little fat, like beefsteaks,	74.4	20.5	3.5	...	1.6
Uncooked meat of the kind supplied to soldiers,—beef and mutton. Bone constitutes one-fifth of the soldier's allowance,* . .	75	15	8.4	...	1.6
Uncooked meat of fattened cattle. Calculated from Lawes' and Gilbert's experiments. These numbers are to be used if the meat is very fat,	63	14	19	...	3.7
Cooked meat,† roast, no dripping being lost. Boiled assumed to be the same,	54	27.6	15.45	...	2.94
Salt beef (Girardin),	49.1	29.6	0.2	...	21.1
„ pork (Girardin),	44.1	26.1	7.0	...	22.8
Fat pork (Letheby),	39.0	9.8	48.9	...	2.3
Dried bacon (Letheby),	15.0	8.8	73.3	...	2.9
White fish (Letheby),	78.0	18.1	2.9	...	1.0
Poultry (Letheby),	74.0	21.0	3.8	...	1.2
Bread, white wheaten, of average quality,	40	8	1.5	49.2	1.3
Wheat flour, average quality, . .	15	11	2	70.3	1.7
Biscuit,	8	15.6	1.3	73.4	1.7
Rice,	10	5	.8	83.2	0.5
Oatmeal (Letheby),‡	15	12.6	5.6	63.0	3
Maize (Poggiale),	13.5	10	6.7	64.5§	1.4
Pease (dry),	15	22	2	53§	2.4
Potatoes, 	74	1.5	.1	23.4	1
Carrots (cellulose excluded), . .	85	.6	.25	8.4	.7
Cabbage, 	91	.2	.5	5.8	.7
Butter,	6	.3	91	...	variable. taken as 2.7
Egg (10 per cent. must be deducted for shell from the weight of the egg),	73.5	13.5	11.6	...	1
Cheese,	36.8	33.5	24.3	...	5.4
Milk (sp. gr. 1030 and over), ¶ . .	86.7	4	3.7	5	.6
„ (sp. gr. 1026),	90	3	2.5	3.9	.5
Cream (Letheby),	66	2.7	26.7	2.8	1.8
Skimmed milk (Letheby),	88	4.0	1.8	5.4	0.8
Sugar,	3	96.5	.5
Pemmican (De Chaumont)** . . .	7.2	35.4	55.2	...	1.8

* The gelatine of the meat is reckoned with the albuminates: it is not certain what deduction should be made on account of its lower nutritive value, which is about one-fourth that of albumen (Bischof).

† These numbers are taken from John Ranke's analysis.

‡ I have determined the nitrogen in good Scotch oatmeal, and the results are almost identical with Letheby's. I have therefore omitted Von Bibra's analysis given in the 3rd edition.

§ There is also some indigestible cellulose in maize, pease, and carrots, which is not included in the table.

|| Professor Attfeld (*Report of Committee on Scurvy*, 1877, p. xlv. et seq.) gives rather different figures, reckoning the albuminates a good deal higher.

¶ These numbers are probably rather high.

** The sweet pemmican used in the Arctic expedition of 1875-76 was similar to the above (the ordinary pemmican used in the same expedition) with the addition of about 5 per cent. of cane sugar. In other cases, particularly in the American pemmican, raisins and currants are added. (See *Report of Committee on Scurvy* for analyses by Professor Frankland and Dr. De Chaumont). A little pepper is added, not reckoned quantitatively in the above analysis, but probably included in the "loss," i.e., the difference between the sum of the above constituents and 100.

In the following table (III.) the calculation of these four ingredients per ounce has been made; the substance being supposed to be in its natural state, and to have the composition already assigned to it in the former table (Dr. PARKES).

Substance.	One ounce (=437·5 grains) contains in its natural state in grains.			
	Water.	Nitrogen.	Carbon.	Salts.
Best hosp. steaks and chops,	14·22
Uncooked meat (beef), . . .	328	10·35	64	7
Uncooked fat meat (beef), . . .	275·6	9·6	98·3	16
Cooked meat, . . .	236	19	117·7	13
Salt beef, . . .	215	20·4	69·7	92·3
„ pork, . . .	192	18·0	85·0	99·7
Fat pork, . . .	170	6·8	192·0	10·1
Dried bacon, . . .	65·6	6·1	273·8	12·7
White fish, . . .	341	11·5	52·4	4·4
Poultry, . . .	324	14·5	62·0	5·2
Bread, . . .	175	5·5	119	5·6
Wheat flour, . . .	65·6	7·6	169	7·4
Biscuit, . . .	35	22·7	183	7·4
Rice, . . .	43·7	3·5	176	2·2
Oatmeal, . . .	65·6	8·7	172	13
Maize, . . .	59	7	176	6
Pease, . . .	65·6	15	161	10
Potatoes, . . .	324	1	49	4·4
Carrots, . . .	398	·4	18	3
Butter, . . .	26	·2	315	11·8
Egg, . . .	321	9·3	71·5	4·4
Cheese, . . .	161	23	162	23·6
Milk, . . .	380	2·75	30·8	2·6
Cream, . . .	289	1·9	93·5	7·9
Skimmed milk, . . .	385	2·8	25·0	3·5
Sugar, . . .	13	...	187	2
Pemmican, . . .	31	24·3	273·5	8·0

The nutritive value should be calculated on the raw substance, as the analyses of cooked food are more variable. The mode of using the table is stated by Dr. Parkes as follows :—The quantity of uncooked meat or bread being known, and it being assumed or proved that there is no loss in cooking, a rule of three brings out at once the proportions. Thus, the allowance of meat being 12 ounces—of not the best parts—twenty per cent., or 2·4 ounces, are deducted for bone. The quantity of water in the remaining 9·6 ounces will be $\frac{7·5 \times 9·6}{100} = 7·2$, and the water-free solids will be 2·4 ounces. The albuminates will be 1·44 ounce; the fats, ·8064; and the salts, ·1536 ounce. The precise amount of the mineral matters of the various articles can be calculated from the analyses given under the head of each article. Thus the usual range of a standard daily diet for an adult man, calculated in this way, is from 250 to 350 grains of nitrogen; and the extreme range is from 2 to 7 ounces of dry albuminate; or from 138 grains of nitrogen (the smallest amount necessary for the bare maintenance of life, as calculated by Dr. Lyon Playfair) to 483 or 500 grains, the amount taken under very

great exertion. The carbon ranges in various diets from 3,600 to 5,800 or 6,000 grains; and the amount of salts when it reaches 461 grains is large, but it is difficult to estimate them, as so much sodium, chloride, and lime salts are lost through the skin. The salts seem to be made up of chlorine, 120 grains; phosphoric acid, 50 grains; potash, 40 grains; lime, about 4 grains by the urine and some by the bowels; magnesia, 4.7 grains by the urine, and a considerable amount by the bowels; and iron, the amount of which is quite uncertain (PARKES, l. c., p. 198). A certain proportion between the carbon and the nitrogen ought to be maintained. In the best diets this is,—Nitrogen 1 to Carbon 15 (DE CHAUMONT).

The preceding very interesting and valuable tables are given here to show the actual proportions in which various substances used as food may be associated in dietaries, and as a guide to the student of medicine when, as a practitioner, it may often be his lot to devise and construct scales of diet suited to various conditions of existence. They will also aid him in the investigation of the causation of the constitutional diseases, when the following order of inquiry should be methodically followed:—(1.) Is the food excessive or deficient in quantity, as a whole, or in any of its primary classes of aliments? (2.) Are the different articles digestible and assimilable; or, from some cause inherent in the food, or proper to the individual, is there difficulty in primary digestion or want of proper assimilation? (3.) Is the quality of the food altered either before or after cooking? (PARKES, l. c.) The calamities which happened to our soldiers in the Crimea (in 1854) show that the dietaries of working men cannot be safely reduced below the physiological standard; and, in the words of Sir Robert Christison, “any person conversant with the science of the present subject could have foretold, as a certain consequence, sooner or later, of their dietary, that the British troops would fall into the calamitous state of health which befell them in the Crimea.”

In cases where preserved food must be used for want of fresh food, there are difficulties to be overcome which experience and science have made to disappear in a great measure. It is known that salt meat has a tendency to favour the development of disease under certain circumstances; but it is not known, either physiologically or chemically, what is the exact nutritive value of salt meat in a scientific point of view. There are three circumstances connected with its use which tend to diminish its value as an article of diet—namely, (1.) The process of salting meat is very deteriorating, for the brine extracts the juices of the flesh, and so removes most of the important constituents, organic and mineral (phosphoric acid, lactic acid, and magnesia.) Extract of meat may be got by dialysis from the brine (WHITELOW); and Liebig has reckoned the nutritive loss at one-third, or even one-half. Myosin is also found to be soluble in a 10 per cent. solution of chloride of sodium, and hence a large quantity of this substance also passes into the brine. Dr. Parkes concludes that salt beef may, perhaps, be reckoned as equal to two-thirds the quantity of fresh beef—an estimate he considers quite high enough (l. c., p. 194). (2.) The salt meat for the navy and for soldiers in the field is always highly salted, in order to keep for two years or more in every climate; and such highly salted meat must be so thoroughly steeped

in cold water, to remove the salt, before it is eatable in large quantity, that much of its most nutritive constituents must be washed out—namely, its albumen and sapid extract, called osmazôme (CHRISTISON). (3.) The sarcolemma of salted meat is always hardened; and hence those foods require a long time for digestion, and frequently disagree with the stomach (LETHEY). Few can eat a pound of salted meat daily for any length of time, even when fed on rations by no means liberal. Nitrogenous elements must therefore be added to a diet where salted meat predominates, rather than increase the amount of that substance; and this is best done by adding pease, flour, currants, raisins, and oil, butter or lard, to the ration. It is not certain how the value of salted meat should be calculated. Dr. Parkes has given the following analysis per cent. by Girardin of uncooked American salt beef:—Water, 49·11; fibrine and cellular tissue, 24·82; extractive matters, 3·28; albumen, ·70; fats, ·18; soluble salts, 21·07. “Experience has shown,” says Dr. Letheby, “that there are certain articles of food which are not particularly nourishing in themselves, but which serve some very important purposes in the animal economy. This is the case with tea and coffee: in fact, the use of a vegetable infusion, containing astringent matter and an active principle rich in nitrogen, has been almost universal among mankind from the earliest times.” “The physiological action of these beverages appears to be of a somewhat singular kind; for while they excite the brain, they calm the nervous system generally, and though they produce a state of wakefulness and activity, yet they also induce a species of languor and repose. Lehmann has ascertained by experiment that coffee greatly diminishes the wear and tear of the system: it oils the machinery, as it were, and checks the waste of friction. Those who use it find that during active exercise the destruction of tissue is prevented, and that there is less demand for food; in fact, with a maximum of work to perform, and a minimum of food to accomplish it, he will best sustain his vital power who resorts now and then to a cup of tea or coffee. Hence its value as a means of economising food, and hence its importance to the poor labouring man.” In many of our large merchant-ships the crews are engaged on the condition that coffee shall take the place of grog; and those captains who are careful of the health of the men give them warm coffee before and after they have been aloft in cold and stormy weather—a practice which cannot be too much overvalued. As an article of diet coffee stimulates the nervous system, and in too large quantity may produce tremors. It increases the frequency of the pulse in men, and removes the sensation of commencing fatigue during exercise. It appears to increase the urinary water; and it increases the action of the skin. It is invigorating, without producing subsequent collapse; and the hot infusion is almost equally serviceable against both cold and heat. Tea has a stimulative and restorative action on the nervous system, and is useful under circumstances of great fatigue.

EFFECTS OF OVERFEEDING OR EXCESS OF FOOD.—Food may be taken in such excess that it is not absorbed. It then undergoes chemical changes in the alimentary canal, and at last putrefies, when much gas is formed (carbonic acid, sulphuretted and carburetted hydrogen). As much as 30 pounds of a half putrid mass have been got rid of by purgatives (ROUTH, quoted by

PARKES, l.c., p. 202). Dyspepsia, constipation, and irritation, causing diarrhoea, which does not always empty the bowels, are produced. Sometimes such putrid substances may be absorbed, as shown by signs of evident poisoning of the blood, a febrile condition, torpor and heaviness, fœtor of the breath, and sometimes possibly even jaundice. When excess of albuminates continually passes into the system, congestion and enlargements of the liver, and probably of other organs, and a general state of plethora, are produced. If exercise is not taken at the same time, there is a disproportion between the absorbed oxygen and the absorbed albuminates, which must lead to retention of some substances in the body, or to irritation of the eliminating organs by the passage through them of products less highly elaborated than those they are adapted to remove. It is highly probable (though not proven) that gouty affections arise in this way. A great excess of albuminates without other food produces in a short time (five days—HAMMOND) marked febrile symptoms, malaise, and diarrhoea; and if persevered in, albumen appears in the urine. Excess of starches and of fats delays the metamorphosis of the nitrogenous tissues, and produces excess of fat (corpulence). Acidity and flatulence are caused by the use of much starch; and if excess of starch be taken under any circumstance much passes into the fæces; and the urine often becomes saccharine. By studying the tables given at pp. 1124-25, the economy of meat would be greatly promoted, especially by a substitution for it (to a greater extent than has ever been the custom in this country) of lentils, pease, haricot beans, green vegetables, especially in stews with meat—*pot au feu* (see "The Doctor in the Kitchen," *Brit. Med. Journal*, Sept. 27, *et seq.*, 1879), and of fish, rice, cheese, macaroni, hominy, and oatmeal. A meat meal (except in very exceptional cases of hard work) once daily ought to be sufficient for any man. Too much respiratory food favours the development of fat, and checks the proper nutrition of the muscular tissues; hence it is that rice feeders and potato eaters, and those who indulge in fermented liquors, are often bloated in their appearance, become extremely fat, and are not capable of prolonged exertion. The brewer's drayman is a bad subject for the wards of an hospital; for though he usually has all the appearance of a man possessed of great muscular strength and vital endurance, yet he is not so in reality, for the muscular tissues have been encroached upon by fat, and the general power has been weakened by an undue influence of the respiratory element. Most of the animals in our menageries, from a too liberal allowance of respiratory food, die from fatty degeneration. Accumulation of the nitrogenous elements in the blood is often also a prolific source of disease, and their non-elimination is conducive to the propagation and development of many general diseases. Attention has now been drawn to the influence of such a condition in establishing the characteristic diseases of overfed convicts (LETHEY, THOMPSON).

CORPULENCE.—One of the most distressing results of overfeeding from a wrongly adjusted dietary is *corpulence*. It is to be considered a disease when "it renders persons, from difficult respiration, uneasy in themselves; and, from inability to exercise, unfit for discharging the duties of life" (CULLEN). Dr. T. King Chambers, from observations of thirty-eight persons, whose weight varied from 224 to 504 pounds, comes to

the following conclusion:—In the corpulent, (1.) The bony framework of the body is less massive than in the spare, as indicated especially by the smallness of the hands and feet. (2.) The skin is usually fresh coloured and thin. (3.) The respiratory function presents the following well marked peculiarity—namely, the volume of air the lungs are capable of containing in the chest and expiring from it is considerably less than the average quantity contained and expired by healthy persons of the same height. The vital capacity is diminished and less carbon expired. Hence, the wind becomes short and the mental powers inactive from *corpulence* (*Gulstonian Lectures*, 1850). Much has been written on the subject of *corpulence*; and the attention of the public was successfully arrested by one who appreciated the relief he obtained by following the directions of a physician who happened to take a scientifically correct view of his case. Mr. Banting (whose pamphlet on the subject of “Corpulence” is well known) managed to reduce his physical proportions under the following dietary:—

Breakfast—Four or five ounces of beef, mutton, kidneys, boiled fish, bacon, or cold meat of any kind (except pork), a large cup of tea (without milk or sugar), and one ounce of dry toast. *Dinner*—Five or six ounces of any fish (except salmon or eels), any meat (except pork), any vegetables (except potatoes or rice), one ounce of dry toast, fruit out of any pudding, any kind of poultry or game, and two or three glasses of good claret, sherry, or Madeira (champagne, port, or beer forbidden). *Tea*—Two or three ounces of fresh fruit, or a rusk or two, and a cup of tea without milk or sugar. The tea may be very much enjoyed when taken in the Russian fashion—i.e., with a thick slice of lemon floating on the top, instead of milk. *Supper*—Three or four ounces of meat or fish, similar to dinner, with a glass or two of claret. *Nightcap*, if required, a tumbler of grog (gin, whisky, or brandy, without sugar), or a glass or two of claret or sherry.

The quantities of the different articles specified in this liberal diet-roll, Mr. Banting states, must be left to the natural appetite; but for himself he took at *breakfast* six ounces of solid and eight of liquid food; at *dinner*, eight ounces of solid and eight of liquid; at *tea*, three ounces of solid and eight of liquid; at *supper*, four ounces of solid and six of liquid; and the *nightcap* he introduces to show that it is not injurious; whilst, for the consolation of smokers, it may be mentioned that tobacco is allowable.

When Mr. Banting began this treatment in August, 1862, he weighed 202 lbs.; and after a year's perseverance in it, in September, 1863, he had lost 46lbs.; and had reduced his girth $12\frac{1}{4}$ inches. The sound dietetic principles on which corpulence ought to be treated had been previously enunciated by Dr. T. K. Chambers, in his excellent monograph on that subject; and Mr. Banting's dietary is in exact accordance with what is there stated, and with what has been stated in the text. A still more varied dietary might be prescribed on similar principles by reference to the tables at pages 1124 and 1125. The intervals between meals ought not to be too long; but in this respect there are great individual differences as to slowness or rapidity of digestion.

EFFECTS OF DEFICIENT FOOD.—“A deficiency of food, especially of the

nitrogenous part, quickly leads to the breaking up of the animal frame. If nitrogen be cut off from the body, the various functions languish. The complete deprivation of albuminates, without lessening of the other classes, produces marked effects only after some days,—*e.g.*, five to seven days (PARKES). If such abstention be prolonged, however, there is eventually great loss of muscular strength, often mental debility, and some feverish and dyspeptic symptoms. Then follow anæmia and great prostration. The elimination of nitrogen in the form of urea greatly lessens, though it never ceases, while the uric acid diminishes in a less degree. If starch be largely supplied the weight of the body may not lessen for seven or eight days (HAMMOND, quoted by PARKES, *l. c.*, p. 204). If the deprivation of albuminates be less complete (70 to 100 grains of nitrogen being given daily) the body gradually lessens in activity, and passes into more or less of an adynamic condition,—thereby predisposing to the attacks of all the specific diseases which have been considered in this volume (especially malarious affections and typhus), also to pneumonias; and also modifying the course of some of these diseases, as, for instance, typhoid fever, which runs its course with less elevation of temperature than usual, and with less or no excess of ureal excretion. The deprivation of starch can be borne for a long time if fat be given; but if both fat and starches are excluded, though albuminates be supplied, illness is produced in a few days. Men can be fed on meat for a long time, as a good deal of fat is then introduced; but the deprivation of fat does not appear to be well borne, even if starch be given; but the exact effects are not known. The great remedial effects produced by giving fats in many of the diseases of obscure malnutrition prove that the partial deprivation of fat is both more common and more serious than is supposed, and in all diets for soldiers and prisoners in every country it is greatly deficient (PARKES, *l. c.*, p. 204).

Famine, plague, and pestilence, are associated with each other in the public mind, and the records of every country show how closely they are related. The medical history of Ireland is remarkable for the illustrations of how much mischief may be occasioned by a general deficiency of food. Always the habitat of fever, it every now and then becomes the very hotbed of its propagation and development. Let there be but a small failure in the usual imperfect supply of food, and the lurking seeds of pestilence are ready to burst into frightful activity. The history of the famine of the present century is but a too forcible illustration of this. It fostered epidemics which had not been witnessed in this generation, and gave rise to scenes of devastation and misery which are not surpassed by the most appalling epidemics of the Middle Ages. The principal form of the scourge was known as the contagious famine fever (typhus), and it spread not merely from end to end of the country in which it had originated, but, breaking through all boundaries, it crossed the ocean, and made itself painfully manifest in localities where it was previously unknown. Thousands fell under the virulence of its action, for wheresoever it came it struck down a seventh of the people, and of those whom it attacked one out of nine perished. Even those who escaped the fatal influence of it were left the miserable victims of scurvy and low fever. Another example, not less striking, of the terrible consequences of what may be

truly called famine, was the condition of our troops during the early part of their sojourn in the Crimea, in 1854. With only just enough of food to maintain the integrity of the system at a time of repose, and at ordinary temperatures, they were called upon to make large muscular exertions, and to sustain the warmth of the system, in the midst of severe cold (LETHEBY). India in recent years has furnished a similar history. The famine minutes of Sir Richard Temple, published in the "Gazette of India" of Feb. 3, 1877, showed that *one pound* a day of grain was sufficient, in his opinion, to sustain life, and that the experiment ought to be tried. It was fortunate that Surgeon-Major Cornish, F.R.C.S., the able Sanitary Commissioner for Madras, deemed it his duty to record his protest against such an opinion as being in direct contradiction to the accumulated testimony of scientific observers in every country in which this question of the quantities and variety of food essential to keep a labouring man in health and strength has been the subject of investigation. He showed that sixteen ounces of cereal grains are insufficient to maintain a labouring adult in health; that the evidence of the district medical officers in the Madras Presidency, submitted to Government long before any famine difficulties and controversy had arisen, established the fact "that the minimum grain allowance of a man in health and in work is not less than twenty-four ounces; while from twenty-four to forty-eight ounces *per diem* are very commonly consumed by the natives of Southern India." And, after showing the varying amount of nitrogen in sixteen ounces of the different kinds of grain used in India (*rice* varying from sixty-eight to eighty grains, *raggy* or *cholum* about one hundred, *wheat* one hundred and sixteen grains), Dr. Cornish adverts to the well-known fact that, with a view to supplement this low amount of nitrogen in *rice* and *cholum* (the grains in common use), the natives use "flesh-meat, milk, cheese, &c., and the vegetable feeders *pulses*, which are highly nitrogenous in their composition." Dr. Cornish then turns to the experience gained in the Indian jails, and shows that five things are essential to the health of prisoners other than the bare allowance of one pound and a half *per diem* of cereal grains. These are—(1.) pulses; (2.) meat or fish; (3.) oil and condiments; (4.) fruit or vegetable; (5.) salt. "With a regulated minimum of these articles we are enabled," he writes, "to keep the prisoners in fair health;" but "if this minimum of food, from any cause, is withheld or lessened for a time, the jail inmates begin to drop off from the very same causes that tell so severely on the famine-stricken people in the relief-camps of India." After exposing the fallacy, that a diet which will barely keep a man alive who is at rest, will sustain him when employed on work requiring physical exertion, Mr. Cornish arrives at the obvious conclusion that with one pound of grain *per diem*, and half an anna in cash, the labourer on the famine-works cannot make his daily ration equivalent to *two hundred grains of nitrogen*; that, in short, sixteen ounces of grain is insufficient for the maintenance of a labouring man; and he warned the authorities that an attempt to feed men on Sir Richard Temple's famine ration would end in great loss of life—not, indeed, by immediate starvation, but from the diseases that tread in the footsteps of famine (*Brit. Med. Journ.*, May 26, 1877). The actual mortality of the famine-stricken in the Madras camps for ten weeks ending March 31, in a mean strength of 11,005,

was 1,971 total deaths), showing an annual ratio per 1000 of 930·8—a death-rate which wipes out nearly the whole of the living within a year. “The deaths were almost entirely due to diseases which invariably in India attack underfed and starved people—namely, extreme wasting of tissue and destruction of the lining membrane of the lower bowel.”

When privations of clothing and lodging are added to insufficient diet, long exertion, insufficient repose, intemperance, and the miseries of poverty, the symptoms already detailed are of the most aggravated kind. “Long before insufficiency of diet is a matter of hygienic concern—long before the physiologist would think of counting the grains of nitrogen and carbon which intervene between life and starvation—the household will have been utterly destitute of material comfort;—clothing and fuel will have been even scantier than food; against inclemencies of weather there will have been no adequate protection; dwelling-space will have been stinted to the degree in which overcrowding produces or increases disease; of household utensils and furniture there will have been scarcely any,—even cleanliness will have been costly or difficult; and, if there still be self-respectful endeavours to maintain it, every such endeavour will represent additional pangs of hunger. The home, too, will be where shelter can be cheapest bought,—in quarters where commonly there is least fruit of sanitary supervision, least drainage, least scavenging, least suppression of public nuisances, least, or worst, water supply, and, if in town, least light and air. Such are the sanitary dangers to which poverty is almost certainly exposed, when it is poverty enough to imply scantiness of food” (SIMON, *Sixth Report on Public Health*, 1864, p. 14). But a multitude of cases of minor degrees of suffering occur in which the symptoms are less marked than those described. Such cases are indicated by a sallow and dingy appearance of the skin, a soft and flabby feeling of the flesh, more or less emaciation, general debility, feebleness of the circulation, and frequently swelling of the ankles. The stomach becomes disordered, the appetite defective, and digestion impaired. The individual feels languid and desponding, is soon fatigued, incapable of exertion, and has an irresistible desire to fall asleep, from which he is apt to awake suddenly and in a fright. The body is easily chilled, breathlessness and palpitation are experienced after slight exertion, attacks of *vertigo*, *tinnitus aurium*, and transient blindness, are common, and there is a peculiar forlorn and dejected aspect of countenance which is very characteristic. This state of things is commonly soon succeeded by some specific disease; though it may continue, with only slight variation, for a very protracted period, until the patient falls by slow degrees into a state of mental as well as physical incapacity; and, being no longer able to procure any employment, is completely invalided, and applies for medical relief. It may perhaps be thought that these remarks apply to cases of deficient nourishment, which are less frequent; but the experience of those who have practised extensively among the wretched purlieus and miserable abodes which exist in every large metropolitan town can testify to the contrary. I well remember listening to the interesting clinical lectures of Professor Christison, of Edinburgh, on the cases of *scurvy* which prevailed in that town and its vicinity in 1847, and hearing the melancholy recitals

of misery and starvation under which the poor suffered at that time; some under the hard taskmasters of the illegal "truck system,"* and others from absolute want at home. Among many, of whose cases I have preserved notes, a shoemaker had to support his wife and five children on *eight shillings* a week; and, in order to feed his children better than himself, he subjected himself to privations which in time developed *scurvy*. His daily diet consisted of *one pennyworth of bread, with tea, but no milk, in the morning—no dinner—and one pennyworth of bread, with tea, and no milk, in the evening*. After existing *three months* on this diet he became scorbutic. But, apart from these extreme cases, the instances are innumerable in which deficiency of food acts as a predisposing cause of many diseases. It is now generally known that plethora and symptoms of an opposite state very nearly resemble each other, and a discrimination of these differences is of the greatest importance. While coma is often an attendant on plethora, it is not to be forgotten that it is one of the most severe and fatal signs of exhaustion from defective nutrition; and when it supervenes towards the termination of diseases of exhaustion, and the pulse becomes slower, it often acquires a degree of fulness, and gives an idea of strength, quite at variance with its previous character, and little to have been anticipated from the debilitated state of the system.

But in degrees far short of what is popularly known as starvation or famine, insufficiency of nourishment may bring very hurtful consequences to health. Local defects or local peculiarities of diet may exercise an important influence in determining or colouring particular localisations of disease; and generally it may be said, that in order justly to estimate the sanitary circumstances of a people, sufficient regard must be had to the quantity and quality of the people's meat and drink (SIMON, *Report on Public Health*, p. 11, 1864). The injurious effects produced by improper nutrition require to be studied both in relation to food and drink, and the diseases described, which form Section B of General Diseases, are greatly influenced by the nature of the food on which the people live.

CHAPTER XI.

GENERAL MANAGEMENT OF THE SYSTEM LIABLE TO CONSTITUTIONAL DISEASES.

IMPORTANT general indications for the treatment of the CONSTITUTIONAL diseases are suggested by their pathology as expounded in Chap. IX. This general treatment ought to be directed towards the manage-

* The "*truck system*" became developed chiefly during the formation of our great lines of railway throughout the country. The labourers (navvies) were poor, and came to work without money to buy provisions, and their field of labour was often far removed from any place where food could be bought in quantity. The contractors, their employers, then established provision stores, and in place of paying the men in money, they compelled them to take remuneration for their labour by value received in food. By this method, now declared illegal under all circumstances, the labourers often suffered from a deficient and bad supply of provisions.

It was found necessary in 1871, to inquire by Parliamentary Commission into the extent of this system, still carried on in some places.

ment of the system for the *prevention, control, or arrest* of the development of such affections, or to maintain them within such limits as are consistent, at least, with the well-being of the individual. The topics to be noticed under this head are of so comprehensive a scope that they can only be indicated as shortly as possible, with the object of directing the mind of the student to their more extended study. They embrace, in fact, the whole subject of general and individual hygienic management, and the personal care of health. The student is therefore referred at once to two standard authorities, with whose maxims and practice his mind ought to become most thoroughly imbued, in order to direct successfully the measures for the prevention and arrest of the CONSTITUTIONAL DISEASES. The works here referred to are—(1.) *The Management of Infancy*, by Dr. Andrew Combe, ninth edition, 1860, edited by the late Sir James Clark; (2.) *Practical Hygiene*, 1864, the work of my colleague, the late Dr. E. A. Parkes, which has passed through five editions. From the first of these books the student will learn how he must begin at the very beginning. He will learn to appreciate the influence of the constitution of parents on the health of their children, and will learn from it how to direct the management of infant life. From the second work he will learn how he may carry out the details of practical hygiene. This work of Dr. Parkes brings together an amount of material which shows the extent to which exactness and certainty have been imparted to the Science of Medicine. It is a work which marks a new era in Practical Medicine. Two posthumous works by the same author are also here recommended—namely, *Personal Care of Health*, one of the “*Manuals of Health*” published by the Society for Promoting Christian Knowledge; and *Public Health*—a short treatise published by Messrs. Churchill.

From the nature of the CONSTITUTIONAL DISEASES it is to be noticed that there are three periods in their progress and history necessary to be recognised in the pathology and treatment of them. The *first period* may be described as *the period of their constitutional development*. During this period the physician is rarely if ever consulted, unless a constitutional tendency to the disease is suspected, obvious, or perhaps hereditary. The *second period* may be described as the period during which the *constitutional disease is fully expressed* by the phenomena and symptoms already described as peculiar to each of them. The *third period* in the history of these diseases may be described as a *series of intervals*, during which the health seems to be improved between the febrile paroxysms, or the fully expressed conditions of ill-health.

It is for the cure of the fully expressed disease that the physician is generally consulted—a period when he knows he can do the least good. But when the public are aware of the extent to which health may be preserved, and constitutional diseases averted and mitigated by judicious management and treatment *during the first and last periods here noticed*, the physician will be more frequently consulted as to how the health is to be preserved and improved so as to ward off and prevent CONSTITUTIONAL DISEASES.

Whenever the physician has to treat any of these constitutional affections in their fully expressed condition, if he is successful in subduing

the symptoms for the time, by means of the treatment already mentioned under each of them, *the interval of comparative freedom from the paroxysmal expressions of disease is a time most precious, which ought not to be wasted, but which ought to be taken advantage of in preserving and improving the general health, especially by—*(1.) *Proper Diet*; (2.) *the use of Pure Water*; (3.) *the moderate and judicious use of Wines, and of other alcoholic beverages.*

I. DIET.—One of the highest problems in physiology is so to regulate the supply of the *nitrogenous substances*—the *albuminates*—that the *digestive power* of the stomach and intestines may be increased, together with the *formative power* in the nitrogenous tissues, and the *eliminating powers* in the after-stages of assimilation (PARKES). All these three parts of the process must be duly balanced, otherwise health is destroyed. “A great number of diseases are also produced by conditions of indigestibility of food, either dependent on physical or chemical conditions of the food itself, or of the digestive fluids. Indigestibility of food leads to its retention, the results of which are chemical changes and putrefaction going on in the stomach and bowels under the influence of warmth, moisture, and air. Irritation is produced, and dyspepsia, diarrhoea, or dysentery, are caused. There is also some reason for thinking that the albuminates sometimes pass into the circulation, less properly prepared than usual to undergo the action of the liver, and that they therefore produce irritation of that organ, and passing into the blood in some unassimilable state, produce irritation of the skin or kidney. Sometimes, indeed, albumen appears in the urine as if it had circulated like a foreign body in the blood. Such conditions are usually allied to some evident error in primary digestion, but occasionally are not obviously accompanied by any gastric disorder. Whether there is any similar imperfection in the digestion of starch or fat is not at present known” (PARKES, l. c., p. 205). Some forms of diabetes would lead one to believe so.

In the management of the system, therefore, the albuminous tissues can be, to a certain extent, brought under the control of the physician by a judicious adaptation of diet and exercise alone, without the employment of drugs. Drugs, however, when judiciously employed, are important aids at two ends of the intestinal canal—namely, to aid primary digestion and to assist elimination. *Fatty aliments* are essential to the formation of muscular, and especially also of nervous tissues. With the nitrogenous substances or *albuminates*, they are essential to the production of mechanical force. Animal fats appear easier of absorption than the vegetable fats; and the relative proportion of *fat* to *albuminates* ought to be as 1 or $1\frac{1}{4}$ to 2. *Starchy* and *sugary food* saves the *albuminates* and *fats* from too rapid disintegration; so that, by a judicious employment of them in a dietary, the elimination, and perhaps the formation of the albuminates and fatty tissues can be so modified that the administration or withholding of starches and sugars as articles of food must enter largely into the management of the system, according to the circumstances of the case and the nature of the constitutional disease. The production of *lactic acid* in the system seems probably connected with the metamorphosis of starch. To the constant introduction of an excess of alkali in the food, and the no less constant production of acid during the digestion of foods (especially

starches, sugars, and fats), associated with the effects of respiration, are to be ascribed the singular alternation of acid and alkaline fluids in the body. The relative amount of *starch foods* to the *nitrogenous* or *albuminate* substances ought to be as $2\frac{1}{2}$ or $3\frac{1}{2}$ to 1. Excess of starches, sugars, and fat produce excess of fat, sometimes also acidity and flatulence; and the urine may become saccharine under excess of starch as an article of diet.

These are the main points to be inquired into and provided for in adjusting the diet best adapted for the management of constitutional diseases. But every intelligent patient may aid his physician thus far—namely, that if he will consider the subject *bonâ fide* for himself, he will become the best judge of the exact diet which suits him. Dr. Parkes observes that probably 30 per cent. of the persons who consult physicians owe their diseases in some way to food, and in many cases they are perfectly aware themselves of their error or bad habit; yet, with the singular inconsistency of human nature, either conceal it from the man to whom they are professing perfect openness, or manage to blind themselves to its existence. Individual hygienic management—the *personal care of his own health*—must be inculcated upon the patient; while, by regulating diet on the principles laid down by Dr. Parkes in Chapter V. of his work *On Practical Hygiene*, the physician will find he can exercise a great power in the control and limitation of constitutional disease. Most valuable information respecting the adjustment of the dietary will also be found in Dr. Edward Smith's *Practical Dietary*; and in *A Manual of Diet and Regimen for Physician and Patient*, by Dr. Horace Dobell.

II. THE USE OF PURE WATER.—Its influence as an agent in the management of certain conditions of constitutional ill-health is apt to be too much neglected, or not sufficiently appreciated. Ever since the powerful agency of water, under the popular name of the "*water cure*," has been so much bequacked, the medical profession have almost allowed themselves to be seized with a kind of hydrophobia. Nevertheless, there can be no doubt that water has a most powerful influence for good or evil in the treatment of constitutional diseases. Under its influence as a fluid of daily consumption considerable chemical changes are promoted in the body. In the intervals between the extreme expressions of constitutional diseases, such as *gout*, *rheumatism*, *asthma*, and the like, it is a useful and most efficient remedial agent, not only as a diuretic, but also as a local application, especially in obviating the congestions of the viscera associated with these constitutional affections (SIMON). "To accelerate the defective textural metamorphosis or waste is very often the practical result to be aimed at, and such means exist in the simplest and most manageable form in many of the appliances of the so-called '*water-cure*.' With increased water-drinking there is increased discharge of solids by the urine. External appliances, such as the cold *sitz bath*, lasting a quarter of an hour, increase for a time the elimination of urea and uric acids. The protracted sudorific packings produce a still more considerable waste of tissue. Generally, therefore, in cases where the use of water is suitable, and conducted under competent medical advice and direction, the healthy nutrition of the body is promoted, in proportion as refuse materials are actively disengaged" (SIMON, in HOLME'S *Surgery*, Vol. I., p. 118).

III. THE MODERATE AND JUDICIOUS USE OF ALCOHOL.—The properties

of wines, and of alcoholic beverages generally, offer a wide and important field for the careful study of the physician. Although so extensively used as beverages with our daily food, and so generally recognised as valuable agents in the treatment of disease, there is not only great diversity of opinion as to their precise effects, but as yet very little is certainly known of the action of alcohol when administered in the form of *wine*, *beer*, or *spirits*. None of the general statements so frequently met with as to the composition or effects of any particular class of beverages can be relied on as a guide to the physician in prescribing them: and much error has prevailed on the subject, not only in the popular mind, but amongst medical men. *Alcohol* is the most potent agent for good or evil in all these beverages; and, therefore, the amount of its consumption and its effects challenge attention in the first instance. A pint of beer (twenty ounces) may contain *one or two or more ounces of absolute alcohol*, or *less than a quarter of an ounce*. This alcohol may be associated in the beer with an amount of *free acid* varying from *fifteen to fifty grains*, and with an amount of *sugar* varying from *half an ounce to three or four times that quantity*. A glass of sherry (two ounces) may contain from a *quarter to half an ounce or more of absolute alcohol*, with *sugar* varying in quantity from a *mere trace to twenty or thirty grains*, associated with a *very variable amount of free acid*, and with other ingredients. Even in brandy or whisky the amount of alcohol is widely different in different samples. It is impossible, therefore, for a physician to know what his patient is drinking, unless he is acquainted with the chief constituents and their amounts contained in the identical liquor which he may prescribe; and of course, before sound conclusions can be arrived at, the conditions under which these beverages are administered or taken, must also be very precisely observed.

For the purposes of the physician, the prominence of certain substances in wines renders some more desirable than others, or more useful as "*medicines*." These substances are—(1.) *alcohol*, (2.) *acids*, (3.) *sugar*, (4.) *solids* or *extracts*. Of these in their order:—

ALCOHOL, to a variable amount, ought to exist in wines *as a natural product of the fermentation of the grape*; and the conditions of its existence in the wine are then very different from those which obtain when alcohol (distilled even from wine) has been added to wine. It is highly desirable, therefore, to avoid adventitious alcohol; but it is impossible to prove that spirit has been added to a wine unless the amount is absurdly excessive. One vintage sometimes produces a wine with a considerably greater amount of *alcohol* than another of the same vineyard. For example, the samples tested in London (for duty) of *Chateau Lafite*, of vintage 1858, showed 16·5 per cent. of proof spirit; the same vineyard in 1859 showed 17·7; and in 1860 it gave 14·8—the three years varying about 3 per cent. The samples of common *St. Emilion* in 1858 showed 16·5 per cent. of proof spirit; in 1859 the same vineyard showed 15·4 per cent.; and in 1860 it gave 16 per cent.—thus not differing more in the three years than about 1 per cent. But so variable is the amount of natural alcohol in wines from the same district, that a very general notion only can be obtained from tabular statements, and a sample of the wine actually used must generally be analysed. The fermentation of the grape when properly done and complete, seldom yields more than 17 per cent. of alcohol.

Hence anything stronger than this is most likely due to added alcohol. Some of the finest wines do not contain more than 6 to 10 per cent. (PARKES).

The amount of the alcohol by measure may be most readily estimated by the vaporimeter of M. Geisler, of Bonn, which indicates the amount of the alcohol by the tension of vapour at a certain temperature, from the fluid containing the alcohol forcing up a column of mercury. Dr. Parkes gives also a very ready process by evaporation and the use of a urinometer (*Practical Hygiene*, 5th Edition, p. 280).

Having thus ascertained the percentage of alcohol in *wine*, *beer*, or *spirits*, it is easy to calculate by simple proportion the dose of alcohol administered. For example, a pint bottle of claret (*thirteen* ounces), of the strength of *eleven* per cent. of alcohol, will be found to contain 1·43 ounces of absolute alcohol, thus:—

$$100 : 11 : : 13 = 1·43.$$

It is the basis of the rule laid down by Dr. Parkes (l. c., p. 286), which says,—“*To tell how much pure alcohol is taken in any definite quantity of wine, measure the wine in ounces, multiply it by the percentage of alcohol, and divide by 100.*”

During the past eight or ten years a large amount of interest, both lay and professional, has been taken in the question as to the use of alcohol—alike in health and in disease—not only in this country, but also abroad; and not only by those who profess to be total abstainers, but by scientific men and others not abstainers, who are in no way disposed to admit extreme views. An important subject for consideration is also to be found in the facts that—(1.) During each year about 1,000,000,000 of gallons of alcoholic beverages are drunk in this country; and, (2.) among the cases of death recorded by the Registrar-General, over 800 are set down annually under the head of alcoholism, as *delirium tremens* or *intemperance*, a number which represents only a very small part of the mortality and disease produced by this cause,—otherwise estimated at 120,000. The most important movement recently made in connection with this question has been the “*Medical Declaration respecting Alcohol*,” published in December, 1871, and which obtained in a very short time the signatures of 269 London and provincial physicians and surgeons. It has generally been considered a step in the right direction, although it was not approved of at the time by many members of the profession. The *Edinburgh Medical Journal*, the *Pall Mall Gazette*, and other papers and periodicals were in favour of it, and it was speedily published in almost every newspaper in the kingdom.* Some of the medical societies were of opinion that the first

* The following is the text of the MEDICAL DECLARATION RESPECTING ALCOHOL:—“As it is believed that the inconsiderate prescription of large quantities of alcoholic liquids by medical men for their patients has given rise, in many instances, to the formation of intemperate habits, the undersigned, while unable to abandon the use of alcohol in the treatment of certain cases of disease, are yet of opinion that no medical practitioner should prescribe it without a sense of grave responsibility. They believe that alcohol, in whatever form, should be prescribed with as much care as any powerful drug, and that the directions for its use should be so framed as not to be interpreted as a sanction for excess, or necessarily for the continuance of its use when the occasion is past. They are also of opinion that many people immensely exaggerate the value of alcohol as an article of diet; and since no class of men see so much of its

paragraph of the declaration was an exaggeration, and cast an undeserved slur upon the whole profession, and they did not approve of medical declarations being issued to the public; others "did not agree with its censure upon the profession;" and had it not been for such implied censure, there is reason to believe that larger numbers of our profession would have subscribed to it. There can be no real doubt that "the sanction of a certain school of medical practice to the free use of alcohol in disease, had been interpreted by the public into a presumed sanction for its free use in ordinary states of health, as a tonic or strengthener. The importance therefore of bringing the weight of medical influence into the scale, *against* instead of *for*, the habitual use of alcohol as a tonic or as a food cannot be overestimated." The medicinal uses of alcohol were also unfortunately greatly exaggerated by some physicians, especially by the late Dr. Todd, of King's College, London, who did much to make the use of large quantities of alcohol fashionable in the treatment of disease. And although his own plan may not have been to use alcohol indiscriminately, yet he is generally considered as having done so; and it was unfortunate he did not live long enough to counteract the inevitable tendency of his doctrines and practice, as he assuredly would have done. For of late there has been a considerable reaction against this plan of treatment, so that more moderate doses are now given. Nevertheless there are great differences of opinion amongst the profession as to the necessity for the quantities used, especially at our public institutions.* Those who have

ill effects, and possess such power to restrain its abuse, as members of their own profession, they hold that every medical practitioner is bound to exert his utmost influence to inculcate habits of great moderation in the use of alcoholic liquids. Being also firmly convinced that the great amount of drinking of alcoholic liquors among the working classes of this country is one of the greatest evils of the day, destroying—more than anything else—the health, happiness, and welfare of those classes, and neutralising, to a large extent, the great industrial prosperity which Providence has placed within the reach of this nation, the undersigned would gladly support any wise legislation which would tend to restrict, within proper limits, the use of alcoholic beverages, and gradually introduce habits of temperance." We are informed that this document was drawn up by Professor Parkes and Mr. Ernest Hart, at the suggestion of Mr. Rae. But two earlier declarations are also worthy of reference:—namely, one in 1839, drawn up by Mr. Julius Jeffreys, and signed by over 100 leaders of the profession in medicine and surgery. A second declaration was originated in 1847 by Mr. John Dunlop, which was signed by more than 2,000 of the most eminent physicians and surgeons of England and Scotland. This declaration set forth that perfect health is compatible with total abstinence from all intoxicating beverages; that all such drinks can, with perfect safety, be discontinued either suddenly or gradually; and, that total and universal abstinence from alcoholic liquors and intoxicating beverages of all sorts would greatly contribute to the health, the prosperity, the morality, and the happiness of the human race (*Brit. Med. Journal*, Oct. 11, 1879). The fact is, that English medical men, for at least a century and a half, have taught and advised moderation in the use of alcoholics, and sometimes total abstinence. As a medicine, alcohol is not well fitted for self-prescription by the laity; and the medical profession cannot be held accountable for such administration, or for the enormous evils resulting therefrom.

* Dr. J. W. Eastwood, in an admirable paper on "*The Use of Alcohol in Health and Disease*" (*Brit. Med. Journ.* for Sept. 7, 1872), gives the following statistics for 1871 at the following hospitals, as regards the cost of patients for alcoholics weekly:—"At St. Thomas's Hospital, the patients cost 1s. 7½d. per head weekly; at the Sheffield Infirmary, 1s. 4½d.; at Leeds, Newcastle-on-Tyne, and Manchester, 1s. 1d.; at Glasgow, 8½d.; at Edinburgh, 8d.; and at Birmingham, 5½d. At the Newcastle Infirmary the amount has been diminished to the extent of £90 during the last year. In the county asylums for the insane poor there is a great diversity in the amount of wine, spirits, and porter consumed, beer being included amongst provisions. At the

defended such excessive use of alcohol in disease, freely admit that its use is nothing more than a superfluous luxury in health. They agree, too, that it must be considered as a very powerful drug; and that it should be prescribed with as much care and as strict limitation as *ammonia*, *ether*, or any other stimulant. It is a matter of daily observation, however, that the prescription of the physician in the matter of alcohol during illness, is apt to be the rule of life of the patient as regards alcoholic fluids for ever afterwards; and that in the present day the value of alcohol as an article of diet is still immensely exaggerated. Its prescription in disease is therefore not to be interpreted as a sanction for the continuance of its use when the occasion is past; and being firmly convinced that the great consumption of spirituous liquors by the working classes is one of the greatest evils of the day—destroying more than anything else the health, happiness, and welfare of those classes, and neutralising largely the great industrial prosperity within the reach of the nation—the members of our profession will no doubt gladly support any wise legislation tending to favour temperance. There can be no doubt also that this outspoken declaration of medical opinion has greatly stimulated the scientific and rational elucidation of an important subject; that both in the general literature of the period and in the action that is being taken on the part of the non-medical public, the use of alcohol both in health and in disease has become one of the most prominent and “burning questions” of the day; and therefore some members of our profession have thought it necessary from time to time to take some steps to vindicate themselves from the charge of indifference as to the consequences of medical practice in these respects. But many experienced and deep thinking men have also come to the conclusion that teetotalism has failed to produce any serious impression

asylums for Lancaster, Dorset, Notts, and Warwick, the weekly cost is a ½d. per head, at Worcester 4½d., at Durham 6d.; whilst at the borough asylums at Leicester and Norwich it is 7d. and 9½d. respectively. The total cost for the maintenance of each patient varies only from 6s. 10d. in Dorset, to 10s. 6d. in Surrey, per week, but it is more in the borough asylums. From the official reports I cannot find that the recoveries or the deaths bear any reference to these expenses. At the Cumberland Asylum no beer is given as an article of diet, and the patients appear as well off as those in other asylums. To invalids, however, stimulants are given in the usual manner, as the cost per head appears to be 2½d. weekly. At the various workhouses the same irregularity prevails. The 825 inmates of the Whitechapel Workhouse cost 2½d. per head weekly; Shoreditch and Bethnal Green pay 3½d. each, the City of London, 6d., Camberwell 7½d., whilst Paddington pays 9d. The comparison of the number of deaths with the supply of alcoholics affords no correct information as to their value, but it certainly does not prove anything in their favour. On what principle is there this difference in practice amongst the hospitals, asylums, and workhouses of this country? Surely none; and yet *hundreds of pounds* are spent every year, and no one can say *to what good*, if we look at the results. Almost every man will tell you that he is *guided by experience*? but when so many different experiences exist, who shall decide which is best?” From all I can learn, however, there does not seem to be any great diminution in the actual annual total consumption of alcoholics at the hospitals of London since the time of Dr. Todd, and although it may be the fact that all the physicians and surgeons may agree in the opinion that stimulants are ordered too freely, they yet go on ordering them without stint. There can be no doubt, however, that much less alcohol is used in the treatment of acute diseases than there used to be; but the tendency (and a very natural one) is to order it more largely in hospitals as a tonic, an aliment, and a comforter to the poor patient when not suffering from acute disease. On the other hand, the parish medical officers in charge of the hospitals got up of late years by the Local Government Board are extremely stinted in and cautious about the administration of stimulants.

upon the drinking habits of the country. The universality of alcoholic drinking is certainly an argument in favour of its moderate and dietetic use, founded on gratifying a natural desire; but the precise manner in which the alcohol acts has been and still is an unsolved problem. How far and in what manner it acts as a powerful and useful nerve-stimulant, nutrient, sedative, and tonic are still the questions to be solved. The dietetic and moderate use of alcohol is now therefore our first consideration; and what is considered to be "moderation" will be stated presently. But I think it is still the opinion of the profession "that persons in health require no *alcoholics*, and were better without them." Then comes the question, "What is to be the standard of Health?" (see page 5, *ante*.) Health is a very variable condition. Many persons take moderate quantities of alcoholics and enjoy good health; many other persons take none at all, and likewise enjoy good health. It has, however, been observed that total abstainers of the second generation are not good examples of their practice; but then, again, the children of moderate drinkers are also sometimes unhealthy. We know that the children of drunkards are often imbecile or insane, or in some way degenerate in health; but so are the children of others not intemperate.

What becomes of the alcohol taken in *dietetic quantities*? Is it given off as alcohol, or is it consumed in the system, or is it partly given off and partly oxidised? And what influence has it upon nutrition when taken in moderate doses? The physiological effects of alcohol have been investigated and inquired into with considerable care by Rudolf Masing, Böcker, and Mulder; by MM. Lallemand, Perrin, Duroy; by Percy, Ogston, Bence Jones, Carpenter, Spencer Thomson, Hammond, T. K. Chambers, Edward Smith, Anstie, Fraser, Parkes, and others. In answer to the questions propounded there is considerable difference of opinion and dubiety. One set of observers contend that it passes entirely unchanged from the body; another set maintains that a great portion of it, at any rate, is decomposed in the body, with the inference that it acts as a food. From the most recent and elaborate investigations, especially those of Parkes and Hammond, the weight of evidence and opinion seems to rest with those who believe that it acts as a food; and, that if alcohol be introduced into the body in small or moderate doses it is oxidised or destroyed in the system; that the body, in short, is able to oxidise and destroy small quantities of alcohol, to the extent, in a person of normal health, of *one and a half ounces* of absolute alcohol in twelve or twenty-four hours (FRASER). If an excess be taken that excess does not appear to be destroyed. The system seems to be incapable of using up more than the above stated amount in a normal state of health; and the elimination of the excess of alcohol occurs by the various excreting agencies of the body, unaltered in chemical constitution.

The following statements regarding the administration of alcohol may be taken as embracing the practical experience of some of the most eminent men in our profession who have recently given attention to this subject, and have expressed themselves in the "contemporary controversies" of the period on the "alcohol question":—Alcohol may be given with advantage when the nervous system is exhausting itself, and when the tissues of the body generally are being exhausted

by an activity in excess of the other bodily functions. The moderate consumption of alcohol is useful to many persons. It lessens the destructive metamorphosis which goes on; and chemical changes in the blood are partially arrested by it (HARLEY, quoted by PARKES). It may be given, increased in amount, or left off, under the guidance of the appetite for food. As long as a person in ill-health takes and digests food better with alcohol than without, so long will alcohol be of service to him. Beyond this general statement there is no evidence for or against its dietetic use. In *very small quantities* it appears to aid digestion in the stomach; in larger amounts it checks it. A moderate use of beer or of the weaker wines (*i.e.*, of pure unbranded well-fermented wines) may increase appetite and improve nutrition. On the other hand, the use of malt liquors (even when pure and good) is injurious to persons of sedentary habits, or unless much exercise be taken in the open air; but sound, well-fermented beer* is the best of all dinner drinks *for persons of good digestion, who work hard in the open air* (DRUITT). As a beverage alcohol should be taken in *very moderate* quantities, freely diluted, and usually at or after meals. The general evidence tends to show (but does not absolutely prove) that pure alcohol has its pernicious effects greatly lessened, and its good effects more powerfully developed, when highly diluted, and still more so by admixture with other substances, as the carbo-hydrates and salts contained in *beer and wines*. The use of strong wines (15 to 23 per cent.) undiluted should therefore be discouraged as much as possible; and if such an amount of alcohol is found necessary for the due preservation of the wine, and naturally results from the fermentation of the grape, the wine ought to be **HIGHLY** diluted with water when used as a beverage. On the other hand, light wines cannot be long exposed to the atmosphere without acetous fermentation commencing in them. Light wines with a small amount of alcohol, for the use of invalids, must therefore be preserved in bottles of such a size that the whole may be consumed in a day. Much mischief may arise if wines are used by invalids after acetous fermentation has commenced. Besides alcohol and ethers, wine contains several substances of great value as articles of diet,—namely, some albuminous substances, such as *sugar*, as well as other carbo-hydrates, and abundant salts. The vegetable salts are most valuable, and in this respect are highly antiscorbutic (PARKES, l. c.).

But it is the *moderate* quantity of alcoholic fluids which is not only difficult to define, but also to adhere to on the part of the drinker. While the discipline of moderation is better than the discipline of abstinence, it is also more difficult. The presumption in favour of moderation being generally better than abstinence, is strengthened by a comparison very broadly made between those of our race who do not, and those who do, habitually use alcoholic drinks. On this point Sir James Paget states that—"My study makes me as sure as I would ever venture to

* Good beer, well fermented, is easy of digestion, and is light on the stomach. If it is not well fermented the stomach has to do the work which the fermentation vat ought to have done; hence the evil effects of imperfectly or badly fermented beer. Well fermented beer ought to be light, bright and clear, not dull or turbid. On the top, when drawn, a light white thin cream should form, not a froth, thick and brown or coloured.

be on any such question, that there is not yet any evidence nearly sufficient to make it probable that a moderate habitual use of alcoholic drinks is generally or even to many persons injurious; and there are sufficient reasons for believing that such an habitual use is on the whole and generally beneficial; and every one may assume that he may safely use them *in such moderation as he does not find to be injurious*" (*Contemporary Review*, 1878).

In all the discussions which have taken place respecting the advantages or disadvantages of alcoholic drinks, one matter has generally been lost sight of—namely, that all persons are not constituted alike (MURCHISON). The constitutions of no two persons are identical; and hence, as regards both alcohol and other things, each constitution demands a treatment suitable to itself. Speaking generally, therefore, as regards alcoholic capabilities, the late Dr. Murchison arranged healthy persons into three classes:—(1.) Those who during all their lives drink daily a moderate or even a considerable quantity of alcohol, and are to all appearance none the worse. They die, perhaps at a good old age, of ailments with which alcohol can in no way be concerned. But for one person whose constitution enables him thus to live to old age, hundreds succumb early to diseases which are avowedly the result of alcoholic poisoning. Medical experience amply endorses the wisdom of the directors of insurance offices, who accept the lives of publicans only at a greatly increased premium, or decline them altogether. The ability to consume alcohol in any quantity depends much on the circumstances in which a man is placed. He who leads a country life, and takes active exercise in the open air, can consume without suffering an amount which would be positively injurious to him were he a sedentary student, or a professional man in town. It is the altered habits of the present generation that account in great measure for their being less tolerant of alcohol than their forefathers. (2.) There are those who consume a moderate quantity of alcohol, and perhaps at the time feel all the better for it. At length, however, disease overtakes them, and then it is forgotten that the brittle artery, the softened heart, the diseased liver, or the gouty kidney, or the other evidences of premature decay, which for years have been slowly and insidiously advancing, and which at length render life a burden, or terminate it altogether, might have been postponed, or perhaps might never have occurred, had it not been for the daily dose of alcohol, which induced an abnormal chemistry of the tissues, and the circulation of an impure blood. Dr. Murchison's experience led him to the conclusion that alcohol taken in what is usually regarded as moderation, is more or less directly the cause of a large number of the ailments which in this country render life miserable, and bring it to an early close. (3.) There are those, and by no means a small class of persons, to whom alcohol, even in small quantities, is an unmistakable poison; producing lassitude, achings in the limbs, frontal headache, inaptitude for bodily or mental work, want of sleep, and other distressing symptoms. Nevertheless such a patient cannot be prevailed upon to do what alone is necessary,—namely, to give up his daily dose of poison, the daily stimulus which alone is undermining his health, and is the real cause of his weakness. This intolerance of alcohol very often runs in families; like gout, with which it is often associated, it may be inherited; and not unfrequently it appears to be due to a state of the constitution induced

by various diseases, such as severe fevers. For this class of persons there can be no doubt that alcohol is an unmitigated evil; and that for them total abstinence is the best rule (*Contemporary Review*, l. c.).

The real difficulty is in deciding as to the advantage of alcohol to individuals belonging to the first two classes just named. So far as Dr. Murchison's experience enabled him to judge (and his experience was very extensive), it was this, that in a man who enjoys average health, who eats well and sleeps well, the judgment is clearer and the mental capacity greater when he takes no alcohol, than when he takes even a small quantity; and with regard to bodily work, although alcohol may enable him for a time to exert himself beyond his proper strength, the subsequent reaction requires a repetition of the stimulus, and ere long the frequent repetition of the stimulus causes the health to break down. The cases in which small quantities of alcohol are constantly taken with the object of enabling a man to get through his daily toil are among the most distressing examples of alcoholism with which the medical man is brought in contact. The physician who carefully watches the early beginnings of disease in individuals—the dyspepsia, for instance, which is often the first link—cannot fail to admit that these are due, in very many instances, to alcohol in some form or other, which though taken in what most persons would regard as moderation, yet has deranged the primary or secondary digestion, or has in some way disordered the chemistry of nutrition or of elimination. “What, then, are the conditions,” Dr. Murchison inquires, “of the animal economy in which alcohol may be of positive use?” That there are such conditions he believes cannot be denied; but then they are not the conditions of perfect health. It is especially when the circulation is weak or sluggish that a daily allowance of alcohol may do real good. And the following are the conditions he describes:—“(1.) Alcohol is useful in the course of most acute diseases, when the organs of circulation begin to fail, as they are apt to do. A moderate quantity usually suffices. The large quantities—*e.g.*, one or two bottles of brandy in 24 hours—still sometimes administered, may do harm by inducing congestion of various internal organs. (2.) In convalescence from acute diseases, or from other weakening ailments, when the circulation remains feeble and the temperature is often sub-normal, alcohol is useful in promoting the circulation and assisting digestion. (3.) In persons of advanced life the circulation is also often feeble, and a moderate allowance of alcohol often appears to be beneficial. (4.) All other conditions of the system marked by weakness of the muscular wall of the heart, whether permanent or transient, are usually benefited by alcohol. In its administration under any of these circumstances, one rule ought never to be forgotten, namely,—*that for whatever purpose alcohol be given, it ought never to be taken on an empty stomach; and that the prevalent practice of ‘nips,’ or of taking stimulants in the intervals of meals, is most injurious to health.*” A man who is in good health does not require alcohol; and is probably better without it. Its occasional use will do him no harm; its habitual use, even in moderation, may and often does induce disease gradually. On the other hand, again, Dr. Wilks writes, that “to describe the effects produced by alcohol as stimulating is a misnomer. Its stimulating effects may be regarded as *nil* compared

with those which may be styled its sedative or paralysing ones; and it is this property of alcohol which renders it of so great value in certain temperaments, and under many trying conditions of life. Hence the benefit of alcohol as a diet is felt at the close of the day, not to spur a man on to more work, but to quiet and refresh him when the business of the day is over, and especially when there is evidence of an excess of waste over repair. It is thus that it soothes a worried, nervous system; and by preventing wear and tear actually supports the frame. To the question before raised—"What is a moderate use of alcohol?"—no categorical answer can be given. To lay down the same rule for all persons would be like saying that every one must take the same amount of water, sleep, food, or exercise (RISDON BENNETT). Let those who thus ask try to define, to the satisfaction of any ten persons, what under all circumstances and to all people is moderation in bread, or the wearing of jewels; in hunting or the language of controversy" (Sir JAMES PAGET). Alcohol when properly used for dietetic purposes is found to be a natural and very potent means of comfort; but what moderation is must be found out by each individual for himself, each man in this matter must be a rule unto himself. Moderation has been exceeded if what is taken stimulates or excites, on the one hand, or stupifies or stultifies, on the other. The effect ought not to go beyond that of balancing, calming, and comforting the body and mind. It may be regarded as a prudent rule never to take so much alcohol as will cause flushing of the face, heat of surface, marked quickness of pulse, or subsequent thirst. If a man is taking full exercise in the open air, more alcohol can be taken without the production of such physiological symptoms than when he is remaining quiet at home. No alcohol should ever be taken before the mid-day meal. It is, however, a matter of much importance to endeavour to arrive at something like what may be regarded as an average amount of alcohol which may be daily taken without the probability of its causing any present or remote injury to the system. Dr. Garrod's opinion from a great number of observations is, that the quantity of alcohol taken in 24 hours should seldom exceed that contained in half a bottle of claret of good quality; and it will be found that in the case of French bottles this is very little more than half an imperial pint, or perhaps eleven fluid ounces. The amount of absolute alcohol in claret may be taken at 8 per cent., so that the amount of alcohol thus taken would fall short of a fluid ounce by more than one-tenth. In brandy, whisky, and rum, an ounce of alcohol is contained in two ounces and a quarter of these spirits. In port wine, sherry, Madeira, and Marsala, one in about five or six ounces. In champagne and Burgundy one in ten; and in Bordeaux (claret) one in twelve. In strong ales (Burton) one in twelve; in pale ales one in sixteen ounces; in porter one in twenty-five, and in stout one in sixteen. To the extent, then, of *an ounce to an ounce and a half (by volume)* of alcohol, in 24 hours, may be assimilated by the system, and to the improvement of digestion in various ways,—by exciting the glands of the gastric organs; by increasing the blood vascularity; and, perhaps also, by increasing the muscular contractions of the stomach. With regard to its action as a food, the experiments of Hammond show that *one and a half ounce* of alcohol may be regarded, generally speaking, as equivalent to a fourth part of food in

an ordinary mixed diet. His experiments show that, under certain conditions, alcohol increases the weight of the body; that such increase of weight is due to a retarding of the metamorphosis of formed tissues, to a formation also of new tissues, and to a diminished metamorphosis or destruction of fatty tissues. "We may assure ourselves, by common observation," writes Mr. R. Brudenell Carter, "that the moderate consumption of alcohol is useful to many persons; and that it does not produce, at least necessarily, or in any but exceptional cases, the dire effects which have been ascribed to it. These two positions as the teachings of experience, appear to be as unassailable as the familiar knowledge which we have of alcoholic excess as one of the chief causes of misery and disease among men; but the argument for discarding the use of anything for fear of the consequences of its abuse, is one to which the majority of mankind will turn a deaf ear so long as human nature retains any semblance of its present characteristics." The same able writer gives numerous examples of the good effects of alcohol in disease, and especially a remarkable instance in Russian ophthalmic practice. Dr. Gustav Braun, of Moscow, having been accustomed to lose no less than 45 per cent. of the eyes operated upon by him for cataract among badly nourished Russian peasants; and finding that other ophthalmologists were equally unfortunate with himself; he, at length (after trying many expedients, including the use of quinine and other tonic remedies), administered a dose of brandy or of sherry to every patient immediately after operation, and repeated it twice a day for two or three days. The result was that after a year's trial the number of cases in which the eye was totally lost was reduced from 45 per cent. to 6 per cent., with an additional 3 per cent. of imperfect recoveries.

Many can become total abstainers for a time from their accustomed alcoholics, not only without any unpleasant results, but sometimes with marked advantage; and so also from meat with equally beneficial results. There is a temperance in all things. On the other hand, I have known those, especially those past middle life, who, having become total abstainers for a time, found it necessary to return to the paths of moderation again, because they experienced symptoms which indicated that the nutrition of the system was not fully kept up without the aid of a small amount of alcohol in the pleasantest form to them that such a medicine can be taken; and it is more often in circumstances of mental rather than physical labour in which its help is so required.

"Thus, then," in the words of Sir James Paget, "from all the witness to the evils of intemperance we fail to get any clear evidence that there is mischief in moderation. Looking further, we find certain indications that it (moderation in the use of alcoholics) is, on the whole, generally beneficial." Its long abiding custom makes this very probable. Its *habitually moderate* use has been, for many centuries, the custom of a large majority of civilised nations. We are bound further to consider the great length of time during which the nations compared have followed their different habits in regard to drinking, say a thousand years—a time much below the mark. Knowing as we do the mischiefs transmitted by inheritance from the intemperate, it is hardly conceivable that if moderation were in any degree mischievous, its evils should

not by this time have become very evident. The accumulated evils of thirty generations of men given to moderate drinking should now be notable: they should have risen to the level of the manifest evils of one or two generations of excess; or, if they were not positively distinct, they should have appeared in a comparison of the heirs of these drinking generations with the heirs of thirty generations of abstainers. But the result is the reverse of this. West against East, North against South, the heirs of the moderate drinkers are better men in force of body and mind than are the heirs of the abstainers."

As regards disease, there is one general rule which cannot be set aside in the use of alcoholics—namely, that when the marked features of disease consist in the retention of effete matters which ought to be discharged, the use of alcohol must be totally abstained from, as, for example, in Bright's disease. Under all circumstances its effects must be watched; for while it may sometimes be desirable to diminish the metamorphoses of tissue by its use, it must not be forgotten that large quantities of alcohol tend to cause an accumulation in the system of imperfectly oxidised bodies, such as *uric* and *oxalic acid*.

ACIDITY.—Free acid in wine is a necessary result of its fermentation. Its presence is likewise necessary for the evolution of the *bouquet*, for the agreeableness of the wine, and for its wholesomeness. It is, therefore, a popular error to denounce absolutely the existence of acidity in wine. The *relative amount of free acid* present in any particular wine, is a very important point to determine. Much error prevails respecting the relative amount of acidity in different wines, and an excessive amount of free acid is very easily disguised by the relative sweetness of the wine. The estimation of the free acid may be measured by a solution of carbonate of soda, containing 530 grains in the 10,000 grain measure = 53 grammes in the litre; and the amount of acidity is represented by determining how many grains of crystallised oxalic or acetic acid a certain quantity of the soda solution will neutralise. The details of the process are as follow:—

(1.) Take 50 or 100 c.c. of recently opened wine; (2.) add from a burette a standard solution of soda, in small portions at a time (say 5 c.c., or drop by drop); (3.) after every addition, test the fluid by moistening a thin glass rod or feather with the mixture, and streak it across some well-prepared violet litmus paper—when the streaks cease to become red, the analysis is complete; (4.) estimate how much of the standard solution has been used, and express the acidity as equal to so many grains of crystallised oxalic or acetic acid. Good wine contains a quantity of acid that is equivalent to from 300 to 450 grains of crystallised tartaric acid in a gallon. Wines with less than 300 grains of acid in a gallon (6 bottles to a gallon, *i. e.*, 50 grains of acid to a bottle) are too flat to be drinkable with pleasure. Wines with more than 500 grains in a gallon (*i. e.*, 83·2) are too acid to be pleasantly drinkable; and wines with more than 700 grains in a gallon are undrinkably sour (DRUITT'S *Report on Cheap Wines*, p. 178). It is also very important to get at the *quality* of the acid—to the extent, at least, of dividing the *volatile* from the *non-volatile* (and less digestible acids). A large amount of acid and acid salts may not be readily digested; and in many constitu-

tional diseases the supply of acid to the system is a thing to be desired or prevented (see also PARKES, l. c., p. 219).

The necessary standard solutions and burette apparatus are supplied by Mr. Griffin, 119 Bunhill Row, who has prepared a set of apparatus which may show all that chemistry can teach of the quality of wine; by Messrs. Bulloch & Reynolds, Hanover Street, London; also, Harper & Sutton, Operative Chemists, Norwich.

SUGAR is characteristic of all the *sweet* wines, and of many wines in their immature condition. Its amount tends to diminish with age, so that old wines of the *sweet* sort may be less pernicious, as regards sugar, than the new. *Sweet* wines, or wines which contain a large percentage of sugar, are to be avoided by those who are disposed to corpulence or gout; and they are extremely injurious to those who are disposed to the formation of *oxalic acid*, or to the discharge of sugar in the urine. Its amount in any particular sample of wine is best determined by Soleil's *saccharimeter*. The principle on which the process depends is that the varieties of sugar possess the power of twisting the plane of polarisation of a ray of polarised light which is transmitted through solutions containing these varieties of sugar. *Cane sugar* and *glucose* twist it to the right hand; *fruit sugar* twists it to the left hand. All gradation lists given in books as to the amount of sugar in wines are purely empirical, and apply only to the particular sample of wine examined. In stating the sweetness of champagne, for example, nobody seems to notice that it is a manufactured article; and that the quantity of *syrup added to it* actually varies from *four to twenty per cent.* The champagne known as "*still*" has no sugar. Some sorts of Madeira are also nearly free from sugar; and some sorts of sherry are much sweeter than well fermented ports. The correlation of sweetness with acidity and with alcohol are most important points to be determined with reference to the easy digestion of wines; and hence they are all important for the physician to know.

THE AMOUNT OF SOLIDS may be learned (approximately) by determining the specific gravity after the alcohol is driven off. A low specific gravity shows that alcohol has been added, or that the solids are in small amount (PARKES, l. c., p. 226). The following table gives a standard for the determination of the solids (PARKES, l. c., p. 278):—

Specific Gravity after loss of Alcohol.	Per Cent. of Extract.	Specific Gravity after loss of Alcohol.	Per Cent. of Extract.
1004	1	1024	6
1008	2	1028·1	7
1012	3	1032·2	8
1016	4	1036·3	9
1020	5	1040·4	10

It is much to be regretted, especially for the sake of invalids, that so few wine merchants are acquainted, or care to be acquainted, with the chemical constituents of their different wines. Medical men should, therefore, deal only with those wine merchants who will tell them the amount at least of the several constituents in their wines that have been referred to—namely, of alcohol, acids, sugar, solids—and who will

guarantee the wine they supply as being in accordance with the sample of which they give the analysis. The sale-room of every wine merchant ought to be provided with the means and appliances here indicated for ascertaining the exact amount of the substances contained in the samples of wine they offer for sale. The *amount of alcohol, of free acid, of sugar, and of solids* should be recorded also of every wine on importation, so that the wine merchant may be able to watch the metamorphoses, in respect of these constituents, effected on them by lapse of time. Wine merchants could thus materially assist the efforts of medical men in the cure of disease, and physicians could then prescribe with some knowledge of what they were prescribing; and to that extent at least the welfare of invalids and the sick would be better cared for.

But although by ascertaining these facts a physician may prescribe with better knowledge of what he is prescribing, and to that extent at least be better able to care for his patients, it must not be concluded that chemistry can detect every form of adulteration in alcoholic beverages. As Dr. Druitt justly observes,—“The only real test for wine is the empirical one. It is impossible to say that such a wine must be good in such and such cases, because it contains certain ingredients. . . . The stomach is the real test-tube for wine; and if that quarrels with it, no certificate of Liebig and no analysis are worth a rush” (*Report on Cheap Wines*, p. 6). With all possible aids, the one safeguard against fraud is to deal only with wine merchants and brewers of established character and reputation, who will be above attempting to dispose of unsound or adulterated liquors.

The principal analyst of the Inland Revenue Department reports that he found illicit ingredients in no less than twenty out of twenty-six samples of beer tested in 1865; and he believes that, owing to the difficulty of detection, the practice of adulterating beer with poisonous ingredients is much more prevalent than might be inferred from the small number of discoveries made. These samples were all from the stores of licensed brewers; and although, doubtless, further adulterations are practised by the retailers, they are probably for the most part with less noxious materials, and chiefly with the object of increasing bulk—known as “*stretching*.” As to wines, the following advertisement, frequently appearing in the *Times*, shows that there is a trade openly carried on in this country in the treatment or cure of bad or spurious wines:—

“Wine merchants having wines turned acid may have them returned to their original wholesome perfection in two or three days, by applying to —,” &c., &c.

It is well known that the same practices prevail abroad, so that “pure as imported” is no guarantee for purity of production. Hambro’ wine has been shipped from London to Cadiz and back—an operation which raises the price of the stuff 100 per cent. The custom-house officers in 1865 stopped a large quantity of such stuff imported as “sherry,” which had not a drop of grape-juice in its composition; and it cannot be doubted that the increased consumption and increased competition for low prices have greatly added to the temptations to adulterate. Such liquors, for any medicinal purposes, must be worse than useless; and whenever wine, brandy, or beer is prescribed medicinally, they ought to be obtained from

wine merchants or brewers of the highest respectability. Wine merchants are generally quite willing to sell in small quantities either wine or spirits—a recent change in licensing laws enabling them to do so, and so making it no longer necessary to send to the public-house when only a single bottle is wanted. Wine, like razors, is often *made* for sale, irrespective of use.

The blindly empirical and routine mode in which alcoholic beverages are generally prescribed, in absolute ignorance of their constitution and genuineness, and the importance of them in the treatment of disease or of general ill-health, renders it advisable in a text-book to insist fully on these topics, believing that the physician cannot cope successfully with diseases, and especially with CONSTITUTIONAL DISEASES and the ill-health with which they are associated, unless he learns judiciously to use the immense power at his disposal in the influence of *diet, water, and alcoholic beverages*, as agents in the management of the system during the intervals between the paroxysms of these diseases. For much valuable information on wines, given in a very pleasing form, the reader is referred to *Report on the Cheap Wines from France, Italy, Austria, Greece, and Hungary; their Quality, Wholesomeness, Price, and their Use in Diet and Medicine*, by ROBERT DRUITT, Esq., F.R.C.P., 1865.

END OF VOL. I.

